R.C.P. EDINBURGH LIBRARY
R28317A0236

B
of
mo

Vy. 25

THE

PRACTICE OF MEDICINE.

BY

HORATIO C. WOOD, A.M., M.D., LL.D. (YALE),

PROFESSOR OF THERAPEUTICS AND CLINICAL PROFESSOR OF NERVOUS DISEASES IN THE UNIVERSITY
OF PENNSYLVANIA; MEMBER OF THE NATIONAL ACADEMY OF SCIENCE,

AND

REGINALD H. FITZ, A.M., M.D.,

HERSEY PROFESSOR OF THE THEORY AND PRACTICE OF PHYSIC IN HARVARD UNIVERSITY; VISITING
PHYSICIAN TO THE MASSACHUSETTS GENERAL HOSPITAL; FORMERLY SHATTUCK
PROFESSOR OF PATHOLOGICAL ANATOMY IN HARVARD UNIVERSITY.

ROYAL COLLEGE OF PHYSICIANS. EDINR.
LIBRARY.

LONDON AND PHILADELPHIA:
J. B. LIPPINCOTT COMPANY.
1897.

COPYRIGHT, 1896,
BY
J. B. LIPPINCOTT COMPANY.

ELECTROTYPED AND PRINTED BY J. B. LIPPINCOTT COMPANY. PHILADELPHIA, U.S.A.

PREFACE.

In offering this work to the medical public, the authors desire to state that the volume is the outcome of an attempt to view the practice of medicine simultaneously from the pathologic and therapeutic points of view. Whilst each author has written certain determinate portions of the book, there have been such careful joint consideration and discussion of the various subjects that there is a common responsibility, except in the very few places where final difference of opinion is indicated in the text either over initials or by the use of the pronoun "I" instead of "we."

The section on nervous diseases, the articles on diseases of the muscles and on the various infectious diseases except diphtheria, dysentery, tuberculosis, leprosy, and syphilis, the chapter on acute and chronic poisoning, and all the therapeutics of the book, have been written by H. C. Wood. The remainder of the volume, namely, the articles on the infectious diseases above specified, the chapter on diseases of the blood and of the ductless glands, the chapter on parasites, and the sections on diseases of the circulatory, respiratory, digestive, and urinary systems, except the therapeutics, are from the pen of R. H. Fitz, who desires to express his obligation to A. K. Stone for constant assistance and to R. C. Cabot for expert aid in the preparation of the articles on diseases of the blood. The thanks of the authors are also due to Mr. J. McCreery for very valuable service in the supervision of the proof-reading.

Digitized by the Internet Archive
in 2016

https://archive.org/details/b21964014

CONTENTS.

SECTION I.

GENERAL DISEASES.

CHAPTER I.

DISEASES OF THE BLOOD AND OF THE DUCTLESS GLANDS.

PAGE

General Considerations—Simple Anæmia—Chlorosis—Pernicious Anæmia—Leukocytosis — Leukæmia — Chloroma —Pseudo-Leukæmia — Myeloma—Hemorrhagic Diathesis — Hæmophilia — Scurvy — Purpura — Rheumatic Purpura — Henoch's Purpura—Purpura Hæmorrhagica—Hæmoglobinæmia—**Diseases of the Spleen**—Movable Spleen—Splenoptosis—Embolism and Abscess of the Spleen—**Diseases of the Thyroid Gland**—Goitre, Bronchocele, Struma—Exophthalmic Goitre—Myxœdema—Tumors of the Thyroid—**Diseases of the Thymus Gland**—Tumors of the Thymus Gland—**Diseases of the Adrenal Glands**—Addison's Disease 1

CHAPTER II.

LOCOMOTOR AND CONSTITUTIONAL DISEASES.

Myositis—Acute Polymyositis—Primary Myopathy—Pseudo-Hypertrophic Myopathy — Atrophic Myopathy — Thomsen's Disease — Rickets — Hemorrhagic Rickets—Osteomalacia—Obesity—Acute Articular Rheumatism—Gonorrhœal Rheumatism—Chronic Articular Rheumatism—Arthritis Deformans—Muscular Rheumatism—Gout—Lithæmia—Diabetes Mellitus—Diabetes Insipidus . . 44

CHAPTER III.

INFECTIOUS DISEASES.

Scarlet Fever—Measles—German Measles—Chicken-Pox—Small-Pox—Cow-Pox—Miliary Fever—Typhoid Fever—Typhus Fever—Relapsing Fever—Infectious Jaundice—Cerebro-Spinal Meningitis — Influenza — Dengue — Plague — Diphtheria—Whooping-Cough—Mumps—Erysipelas—Septicæmia—Pyæmia—Tetanus — Malarial Diseases — Dysentery — Cholera — Cholera Nostras — Yellow Fever—Actinomycosis—Mycetoma—Rabies—Anthrax—Foot-and-Mouth Disease—Glanders—Tuberculosis, General and Local—Lupus—Scrofula—Leprosy—Syphilis 95

CHAPTER IV.

DISEASES DUE TO ANIMAL PARASITES.

PAGE

Protozoa—Amœba coli—**Sporozoa**—Hæmatozoon malariæ—Coccidium oviforme—Psorosperms—**Infusoria**—Megastoma entericum—Cercomonas—Trichomonas—Balantidium coli—**Helminthiasis**—**Tæniæ**, Tape-worms—Tænia solium—Tænia saginata—Bothriocephalus latus—Cysticercus cellulosæ—Echinococcus—**Trematodes**—Flukes—Distoma hæmatobium—Distoma pulmonale—Distoma hepaticum—**Anellides**, Leeches—**Nematodes**, Round and Thread Worms—Ascaris lumbricoides—Oxyuris vermicularis—Eustrongylus gigas—Strongylus longivaginatus—Ankylostoma duodenale—Tricocephalus dispar—Filaria medinensis—Filaria sanguinis hominis—Hæmatochyluria, Lymph-Scrotum—Trichina spiralis—**Arthropodes**—Pentastoma denticulatum—Sarcoptes hominis—**Parasitic Insects**—Pediculus capitis, vestimentorum, pubis—Cimex lectularius—Pulex irritans—Sarcopsylla penetrans—**Myiasis**, Maggots 321

CHAPTER V.

POISONING.

Acute Poisoning.

Narcotics: Opium, Alcohol, Chloral, Chloroform and Ether, Illuminating Gas, Prussic Acid, Nitrobenzol, Carbolic Acid, Oil of Tansy, Santonin, Belladonna, Hyoscyamus, Datura, Jamestown ("Jimson") Weed, Cannabis Indica, Hyoscine, and Cocaine.—**Convulsants**: Strychnine, Cocaine.—**Paralyzants**: Chloral, Calabar Bean, the Nitrites, Gelsemium, Lobelia, Coniine, Woorari, Pelletierine.—**Cardiants**: Digitalis, Veratrum Viride, Aconite, the Nitrites, Antimony.—**Irritants**: Mineral Acids and Vegetable Acids, Oxalic Acid, Cantharides, Irritant Oils, Savine, Phosphorus 350

Chronic Poisoning.

Lead, Arsenic, Antimony, Alcohol, Delirium Tremens, Opium, Cocaine 362

SECTION II.

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

GENERAL SYMPTOMATOLOGY.

Disturbances of Motion—Disturbances of Coördination—Disturbances of Sensation—Vaso-Motor and Trophic Disturbances—Disturbances of Intellection . . 377

CHAPTER II.

FUNCTIONAL NERVOUS DISEASES.

Insanity—General Considerations—**Constitutional Insanities**—**Pure Insanities**—Melancholia—Mania—Confusional Insanity—Terminal Dementia—**Neuropathic Insanities**—Paranoia—**Periodical Insanity**—**Neurasthenia**—Hysteria—Astasia Abasia—Singultus—Vertigo—Epilepsy—Periodic Paralysis—Laryngismus Stridulus—Convulsions—Local Spasms—St. Vitus's Dance—Reflex Chorea

PAGE

—Chorea of Pregnancy—Convulsive Choreas—Automatic Chorea—Hereditary Chorea—Tetany—Paramyoclonus Multiplex—Paralysis Agitans—Traumatic Neurosis—Caisson Disease—Heat Exhaustion—Thermic Fever—Occupation Neuroses—Headaches—Migraine—Sleep, its Disorders and Accidents—Correlated Disorders of Memory and Consciousness—Neuralgia 389

CHAPTER III.

ORGANIC DISEASES OF THE BRAIN AND ITS MEMBRANES.

Cerebral Localization—Motion—Sensation—Athetosis—Aphasia—Cerebellar Localization—**Diseases of the Membranes of the Brain**—Pachymeningitis—Leptomeningitis—Acute Meningitis—Tubercular Meningitis—Chronic Meningitis—**Disorders of the Cerebral Circulation**—Cerebral Anæmia—Cerebral Hyperæmia—**Diseases of the Blood-Vessels of the Brain**—Cerebral Thrombosis and Embolism—Cerebral Aneurisms—Thrombosis of Cerebral Sinuses—Cerebral Hemorrhage—Cerebral Palsy of Children—**Diseases of the Brain**—Acute Hemorrhagic Encephalitis—Suppurative Encephalitis—Hydrocephalus—Acute Periencephalitis—Chronic Periencephalitis—Disseminated Sclerosis—Intracranial Tumors—Cerebral Syphilis 471

CHAPTER IV.

DISEASES OF THE MEDULLA OBLONGATA.

General Considerations—Glosso-Labial Paralysis 536

CHAPTER V.

ORGANIC DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

Spinal Localization—Spinal Hemorrhage—Spinal Embolism and Thrombosis—Spinal Anæmia—Hyperæmia of the Spinal Cord—Acute Spinal Meningitis—Chronic Spinal Meningitis—Spinal Abscess—Spinal Tumor—Acute Ascending Paralysis—Acute Myelitis—Chronic Myelitis—Compression Myelitis—Acute Poliomyelitis—Chronic Poliomyelitis—Syringomyelia—Locomotor Ataxia—Antero-Lateral Sclerosis—Combined Sclerosis—Friedreich's Ataxia—Syphilis of the Spinal Cord and its Membranes 540

CHAPTER VI.

ORGANIC DISEASES OF THE NERVES.

Local Paralyses of Motion—Neuritis—Simple Neuritis—Parenchymatous Neuritis—Mesoneuritis—Multiple Neuritis—Sciatica—Inflammation of the Facial Nerve—Inflammation of the Trigeminal Nerve—Neuritic Muscular Atrophy—Neuroma—Syphilis of the Nerve 594

CHAPTER VII.

VASO-MOTOR AND TROPHIC DISEASES.

Raynaud's Disease—Perforating Ulcer—Angioneurotic Œdema—Scleroderma—Morphœa—Facial Hemiatrophy—Xeroderma pigmentosum—Sclerema neonatorum—Œdema neonatorum—Acromegaly 619

SECTION III.

DISEASES OF THE CIRCULATORY APPARATUS.

CHAPTER I.

DISEASES OF THE PERICARDIUM.

PAGE

Pneumopericardium — Hydropericardium — Hæmopericardium — Acute Pericarditis — Chronic Pericarditis 628

CHAPTER II.

DISEASES OF THE HEART AND MYOCARDIUM.

Malformation—Hypertrophy—Dilatation—Fatty Infiltration—Fatty Degeneration—Acute Myocarditis—Chronic Myocarditis—Thrombosis—Cardiac Aneurism—Rupture—Tumors 637

CHAPTER III.

DISEASES OF THE ENDOCARDIUM.

Acute Endocarditis—Chronic Endocarditis—**Chronic Valvular Diseases**—Mitral Insufficiency—Mitral Stenosis—Aortic Insufficiency—Aortic Stenosis—Tricuspid Insufficiency—Tricuspid Stenosis—Pulmonary Insufficiency—Pulmonary Stenosis—Treatment of Chronic Heart Disease—**Cardiac Neuroses**—Palpitation—Irregularity—Tachycardia—Bradycardia—Arhythmia—Angina Pectoris 651

CHAPTER IV.

DISEASES OF THE ARTERIES.

Arterio-Sclerosis—Aneurism 686

SECTION IV.

DISEASES OF THE RESPIRATORY APPARATUS.

CHAPTER I.

DISEASES OF THE NOSE, LARYNX, TRACHEA, AND BRONCHI.

Diseases of the Nose—Epistaxis—Rhinitis—Autumnal Catarrh—**Diseases of the Larynx**—Laryngitis—True and False Croup—Tumors—Spasm—**Diseases of the Trachea and Bronchi**—Trachitis—Acute Bronchitis—Chronic Bronchitis—Membranous Bronchitis—Bronchiectasis—Bronchial Obstruction—Asthma 700

CHAPTER II.

DISEASES OF THE LUNGS.

Congestion—Hæmoptysis—Thrombosis and Embolism—Œdema—Atelectasis—Emphysema—Acute Pneumonia—Chronic Fibrous Pneumonia—Broncho-Pneumonia—Gangrene—Abscess—Tumors—Cancer 730

CHAPTER III.

DISEASES OF THE PLEURA AND OF THE MEDIASTINUM.

PAGE

Diseases of the Pleura—Pneumothorax—Hydrothorax—Hæmothorax—Acute Pleurisy—Chronic Pleurisy—Empyema—Tumors—**Diseases of the Mediastinum**—Mediastinitis—Tumors 770

SECTION V.

DISEASES OF THE DIGESTIVE APPARATUS AND OF THE PERITONEUM.

CHAPTER I.

DISEASES OF THE MOUTH, PHARYNX, AND ŒSOPHAGUS.

Diseases of the Mouth, Tongue, and Salivary Glands—Stomatitis—Glossitis—Psoriasis of the Tongue—Ranula—Parotitis—**Diseases of the Pharynx and Tonsils**—Acute Pharyngitis—Chronic Pharyngitis—Angina Ludovici—Retropharyngeal Abscess—Acute Tonsillitis—Chronic Tonsillitis—**Diseases of the Œsophagus**—Malformation—Stenosis—Dilatation—Diverticula—Perforation—Rupture—Œsophagitis—Tumors—Cancer—Spasm—Paralysis 791

CHAPTER II.

DISEASES OF THE STOMACH.

Methods of Examination—Gastroptosis—Dilatation—Perforation—Rupture—Hemorrhage—Acute Gastritis—Pseudo-membranous Gastritis—Phlegmonous Gastritis—Chronic Gastritis—Ulcer—Tumors—Cancer—Neuroses—Nervous Dyspepsia 817

CHAPTER III.

DISEASES OF THE INTESTINE.

Enteroptosis—Hemorrhage—Acute Enteritis—Chronic Catarrhal Enteritis—Pseudo-membranous Enteritis—Ulcerative Enteritis—Diphtheritic Enteritis—Phlegmonous Enteritis—Appendicitis—Intestinal Obstruction—Intussusception—Cancer—Constipation—Typhlitis—Colitis—Proctitis—Ulcer—Tumors—Neuroses 855

CHAPTER IV.

DISEASES OF THE LIVER, GALL-BLADDER, AND BILE-DUCTS.

Diseases of the Liver—Malformation—Malposition—Fatty Infiltration—Congestion—Perihepatitis, Acute and Chronic—Subphrenic Abscess—Acute Yellow Atrophy—Acute Suppurative Hepatitis—Abscess of the Liver—Chronic Fibrous Hepatitis—Hypertrophic Cirrhosis—Amyloid Degeneration—Cancer-Tumors—**Diseases of the Gall-Bladder and Bile-Ducts**—Jaundice—Cholangitis—Cholecystitis—Cholelithiasis—Tumors—Cancer 909

CHAPTER V.

DISEASES OF THE PANCREAS AND OF THE PERITONEUM.

PAGE

Diseases of the Pancreas—Hemorrhage—Acute Pancreatitis—Chronic Pancreatitis—Calculi—Cysts—Cancer—Tumors—**Diseases of the Peritoneum**—Hemorrhage—Ascites—Acute Peritonitis—Chronic Peritonitis—Chronic Serous Peritonitis—Cancer—Tumors 950

SECTION VI.

DISEASES OF THE URINARY APPARATUS.

CHAPTER I.

DISEASES OF THE KIDNEYS.

Anomalies of Shape and Position—Fused Kidney—Movable Kidney—Disorders of Secretion — Hæmaturia — Hæmoglobinuria — Urobilinuria — Indicanuria — Melanuria—Alkaptonuria, Brenzkatechinuria, Hydrochinonuria — Chyluria, Lipuria—Albuminuria, Globulinuria, Nucleoalbuminuria (Mucinuria), Albumosuria, Peptonuria—Fibrinuria—Lithuria, Uraturia, Uricaciduria—Oxaluria—Phosphaturia—Cystinuria—Glycosuria, Melituria—Acetonuria, Diaceticaciduria — Lipaciduria — Hydrothionuria — Casts — Pyuria — Uræmia — Dropsy—Congestion—Thrombosis and Embolism—Acute Nephritis—Chronic Nephritis—Chronic Diffuse Nephritis—Chronic Fibrous Nephritis—Amyloid Degeneration—Suppurative Nephritis, Abscess of the Kidney 989

CHAPTER II.

CYSTS AND TUMORS OF THE KIDNEY AND DISEASES OF THE RENAL PELVIS AND THE BLADDER.

Renal Cysts—Multilocular Cystic Kidney—Tumors—Pyelitis, Pyelonephritis—Hydronephrosis, Pyonephrosis—Suppurative Paranephritis — Nephrolithiasis—Renal Colic—**Diseases of the Bladder**—Enuresis, Incontinence of Urine—Neuralgia of the Bladder, Irritable Bladder—Cystitis 1041

FORMULARY 1063

CHARTS OF TEMPERATURE 1067

THE PRACTICE OF MEDICINE.

SECTION I.

GENERAL DISEASES.

CHAPTER I.

DISEASES OF THE BLOOD AND OF THE DUCTLESS GLANDS.

GENERAL CONSIDERATIONS.

ALTHOUGH the composition of the blood is variously altered in many diseases, yet there are only certain conditions in which the constancy of the changes and their importance in the production of symptoms are such as to be sufficiently characteristic. Especial attention is, therefore, to be paid to variations in the quality and quantity of the blood-corpuscles and to the amount of hæmoglobin present. The total number of red and of white blood-corpuscles is to be estimated and their proportion determined. The morphological characteristics of the red and white corpuscles are to be ascertained, and the latter are to be classified in accordance with intrinsic differences which are to be observed by especial methods of preparation, and the ratio of such differentiated leukocytes to each other is to be calculated. Furthermore, the percentage of hæmoglobin is to be ascertained. Herz calls attention to the possibility of a further inquiry into the specific gravity of the red blood-corpuscles and of the plasma, by means of which additional modifications in the characteristics of the red blood-corpuscles may be determined.

For practical purposes the examination of the blood is limited to the recognition of the number of red and white blood-corpuscles present in the cubic millimetre and the percentage of hæmoglobin. The blood-corpuscles are to be counted by means of the Thoma-Zeiss or Gowers counting apparatus, or by the hæmatokrit, as advocated by Daland. Normal blood contains about five million red corpuscles and six thousand white corpuscles in the cubic millimetre. Variations in the result of the calculation of the total number of red corpuscles to the extent of two hundred thousand in a cubic millimetre and of the white corpuscles to the extent of one thousand are possible even to accurate observers, and are, therefore, of no practical significance. Daland asserts that less time, skill, and fatigue are necessary if the hæmatokrit is employed. Its use is objected to as requiring more blood, a cut in the thumb, and a bulky instrument. Fleischel's apparatus is most frequently used for the determination of the percentage of hæmoglobin, although the value of such estimates is merely relative.

In the examination of the red blood-corpuscles attention is to be paid to variations in size and shape, and a distinction is to be drawn between corpuscles of normal size and those which are large,—macrocytes,—or small,—microcytes,—or irregularly shaped,—poikilocytes. Nucleated red blood-corpuscles—normoblasts—may be found in small numbers in normal blood, but are increased in anæmia, while large nucleated red blood-corpuscles—macro-, megalo-, or giganto-blasts—are found to any considerable extent only in pernicious anæmia. Poikilocytosis occurs in a variety of diseases in which there are serious disturbances of nutrition. The white blood-corpuscles,—leukocytes,—according to peculiarities made evident by staining, especially by the Ehrlich-Biondi method, are divided into lymphocytes, mononuclear, transitional, and polynuclear leukocytes, and eosinophiles or eosinophilous leukocytes. They occur in normal blood in the following proportions:

Lymphocytes, twenty to thirty per cent.
Polynuclear leukocytes, sixty to eighty per cent.
Mononuclear and transitional leukocytes, six per cent.
Eosinophiles, two to four per cent.

A considerable deficiency of red blood-corpuscles produces a series of symptoms due essentially to a diminution in the quantity of hæmoglobin in the blood. Like symptoms occur when the quantity of hæmoglobin is diminished, although the total number of red blood-corpuscles may be normal. A distinction is thus drawn between anæmias, in which the total number of red blood-corpuscles is diminished, and chlorosis, in which the percentage of hæmoglobin is diminished, although the total number of red blood-corpuscles may be relatively normal. Such a distinction is not absolute, since in the severer varieties of chlorosis the number of red blood-corpuscles is usually diminished.

A diminution in the total number of red blood-corpuscles occurs under a variety of conditions, some of which are readily apparent, as a visible hemorrhage; others are obscure or unknown: hence a distinction is drawn between anæmia or simple anæmia, chlorosis, and pernicious anæmia.

ANÆMIA OR SIMPLE ANÆMIA.

Simple anæmia is usually secondary to some obvious cause, as hemorrhage, serious disturbance of nutrition (whether from disease, insufficient or improper food, unwholesome occupation, or faulty hygienic surroundings), or from the action of poisons, as lead, mercury, and arsenic. Residence in the tropics often produces a pallor of the skin called tropical anæmia, but Glogner and others find in such cases that the red blood-corpuscles and hæmoglobin are not diminished. The above-mentioned causes of anæmia are especially active, according to Weiss and Monti, when affecting young children.

Symptoms.—In all varieties of anæmia there are certain symptoms in common, although varying in severity and in rapidity of development. They are dependent partly upon a deficiency of hæmoglobin, partly upon unknown modifications in the composition of the blood. Such symptoms are mild or severe, may begin as the former and end as the latter, or may be severe from the outset. Shortness of breath and palpitation are indicative of disturbance in oxygenation. Headache, backache, neuralgia, dizziness, obscured vision, ringing in the ears, fainting, mental and bodily weakness, are the effects of anæmia upon the nervous system. Loss of appetite, epigastric discomfort after eating, belching, flatulence, and constipation are the evidences of an altered digestion. Disturbances of menstruation are frequent, the catamenia being irregular or suppressed, scanty or profuse, often painful and exhausting, and the discharge light-colored. The skin, lips, and tongue are pale. Emaciation may or may not be present. A systolic souffle is often to be heard both at the base and at the apex of the heart, there is accentuation of the pulmonic second sound, and a venous hum is usually present at the base of the neck. The pulse is rapid, compressible, and of diminished volume. The temperature is frequently elevated, usually to a slight degree, and the course is often irregular. The urine may be pale or high-colored, but is generally of lowered specific gravity. The blood-count shows a deficiency of red blood-corpuscles and a proportionate diminution in the percentage of hæmoglobin.

Diagnosis.—Simple or secondary anæmia is to be recognized by the appreciation of an obvious cause for the deficiency of red blood-corpuscles and hæmoglobin.

Prognosis.—The prognosis is favorable when the cause can be removed, provided its action has not been excessively prolonged.

Treatment.—The treatment of simple anæmia, after removal of the cause, consists in the administration of iron with strychnine and other bitter tonics to aid in digestion, with laxatives if constipation be produced.

It has been shown by the various chemists working under the control of Kobert that the administration by the mouth of the ordinary preparations of iron does not perceptibly affect the elimination of iron by the kidneys, although after the hypodermic injection of iron and sodium citrate (one milligramme for seven kilogrammes of weight) forty per cent. of the preparation can be obtained from the urine unaltered. It would seem, therefore, that the iron should be used hypodermically. It was found, however, that unless the dose is very small it produces great renal irritation: so that the quantity mentioned above should never be exceeded. Further, it has been proved by Moerner, Gottlieb, Carl Jacobi, and others that iron is eliminated through the intestinal tract very freely: so that the question whether it does or does not escape from the kidneys is of comparatively little importance. The theory of Bunge, that the value of iron in chlorosis is due to a peculiar chemical action in the alimentary tract, which causes the natural albuminous iron compounds in the food to be absorbed, is certainly unproved and improbable. The discovery by Busch that the administration of pure or impure hæmoglobin distinctly increases the elimination of iron from the urine, in conjunction with results already quoted, and with judicious advertisement by interested pharmacists, has led to the large use of hæmoglobin compounds or derivatives.

The chemical work which has been done upon the absorption and elimination of iron is, however, at this time of no practical value. The fact that a certain preparation of iron yields a large percentage to the urine is no proof of its superiority. It is the iron which remains, and not that which escapes from the body, that does good; and it may well be that a preparation which is absorbed easily and which escapes easily is of less value than one which is absorbed slowly and does not escape at all. Clinical experience is the only guide in this matter; it is possible that manganese may be of service in simple or chlorotic anæmia, and that the pepto-manganates of iron may have especial value: but our experience is that the older and simpler preparations will effect all that can be achieved by the more modern, complex, and expensive forms of iron. Among solid preparations fit for exhibition in pill may be mentioned, as being as free as may be from astringency, pilulæ ferri carbonatis, pills of ferrous carbonate, Blaud's pill, dose one to two pills; and ferrum reductum, reduced iron, dose two to five grains. For exhibition in solution we would recommend ferri et ammonii citras, iron and ammonium citrate, dose three to six grains; also ferri et ammonii tartras, iron and ammonium tartrate, dose three to six grains. All the sulphates of iron are highly astringent. The tincture of the chloride is astringent, and very deleterious to the teeth.

CHLOROSIS.

This variety of anæmia, sometimes called the green sickness, is so named from *χλωρός*, "pale green," and is usually regarded as a primary disease of the blood. It is conventionally characterized by a marked deficiency of hæmoglobin without a corresponding diminution in the number of red blood-corpuscles.

ETIOLOGY.—Chlorosis usually occurs in females, especially among blondes, generally at or soon after the age of puberty, although Rieder reports typical cases occurring in persons between thirty and forty-two years of age. It is probable that a congenital predisposition to this affection may exist dependent upon debility or disease of the parents. Faulty hygienic surroundings, as bad air, unwholesome food, overwork, mental, moral, and physical strain or excesses, are important as exciting causes. Meinert has found gastroptosis in forty cases of chlorosis, and regards this condition as a cause of the disease, attributing its occurrence to the use of corsets during the developmental changes at puberty. The etiological importance of the development of the sexual function is generally recognized from the frequent occurrence of chlorosis at or about the age of puberty, and especially in those in whom irregularities exist in the development of this function. Sir Andrew Clark has suggested that the absorption in the colon of toxic products from retained fæces is important in the etiology, while Pick attributes a like effect to toxic absorption from the dilated stomach which he frequently finds in chlorosis. Von Noorden, however, disputes the theory that chlorosis is due to the absorption of putrefactive products in the intestine. Enlargement of the spleen, found by Jacobi in severe cases, has been observed by Chvostek and Clement in many cases, and the latter regards chlorosis as of infectious origin.

MORBID ANATOMY.—An anatomical basis for chlorosis was first rendered probable by the researches of Virchow, who frequently found a hypoplasia of the arteries. This was especially characterized by a thin, narrow, and elastic aorta, with irregular origin of its primary branches, especially of the intercostal and lumbar arteries, and by a superficial, spotted, fatty degeneration of its intima. Hypoplasia of the heart, uterus, and ovaries is frequently associated, although in the adult secondary hypertrophy of the heart and hyperplasia of the uterus may be present.

SYMPTOMS.—The patient suffers from symptoms of a mild or severe anæmia, chiefly attributable to the deficiency of hæmoglobin. Headache, dizziness, fainting-fits, blurring of vision, and ringing in the ears often occur. Backache, neuralgia, hebetude, and muscular weakness may supervene, and hysterical paroxysms are frequent. Palpitation and dyspnœa on slight exertion or in consequence of emotional excitement are present. Slight elevations of temperature without apparent cause are not uncommon, and may continue for days or weeks. Digestive disturbances are conspicuous, and an abnormal appetite for pickles, slate-pencils, and chalk, an excessive fondness for sweets, a lack of appetite for nutritious foods, epigastric distress after meals, frequent eructations of gas, occasional vomiting, and persistent constipation are noticeable. Oswald and others find that an excess of free hydrochloric acid in the stomach is the rule, and this observer attributes the digestive disturbance to a lack of motor activity, a view which is favored by the noticeable occurrence in this disease of a displaced and dilated stomach. The secretion of urine is but little affected. It is usually pale, sometimes alkaline, its specific gravity about 1015, with a slight diminution in the elimination of urea. Menstrual disturbances are frequent, especially amenorrhœa, or a scanty flow, and dysmenorrhœa. Displacements of the uterus and ovaries are common, and chlorotic women are usually early subjected to gynæcological treatment. The patients often appear well nourished, although puffiness of the eyelids and ankles may occur, especially after the symptoms have existed for some time. Pallor of the skin, mouth, and tongue is conspicuous. The occasional occurrence of a greenish or greenish-yellow tint has already been indicated, and flushing of the cheeks from emotional excitement is often of ready occurrence. The impulse of the heart, especially when rapid, is visible and palpable over an increased area, and a systolic souffle is frequently to be heard either over both base and apex or over one or more valves, especially the pulmonary valve, the second sound of which is usually accentuated. A carotid pulse is often visible, and a murmur is, as a rule, easily heard on auscultation of this artery. Auscultation of the internal jugular vein, especially the right, at the base of the neck usually reveals the venous hum or *bruit de diable*, which, however, is to be heard in other than chlorotic individuals. The pulse varies in frequency in accordance with the degree of excitability of the patient, and is of moderate volume and diminished tension.

The examination of the blood shows that the red blood-corpuscles are not especially modified in number, although during the course of the disease and especially in the severer types there may be oligocythæmia to the extent of less than one-half the normal number of red corpuscles. They are often noticeably pale in color, vary considerably in size and shape, and the nucleated forms are relatively numerous. The percentage of hæmoglobin may be reduced to thirty-five per cent. even when there is no considerable diminution in the number of red blood-corpuscles. It may be as low as twenty-five per cent. when there is a considerable reduction in the number of red corpuscles. The alkalinity of the blood is stated to be diminished.

The symptoms of chlorosis are often prolonged over a period of months or years, in the latter case temporary improvement and repeated occurrences of the symptoms being frequent. Serious complications may arise, the most frequent of which is ulcer of the stomach, this affection being often found in young women who have previously suffered from chlorotic symptoms, especially from gastralgia. In fatal cases of chronic tuberculosis and chronic diffuse nephritis hypoplasia of the aorta is frequently found, and the suggestion is direct that such affections may pursue a more severe course when occurring in chlorotic patients. Dieulafoy and Hanot report cases of transitory nephritis in the course of chlorosis. Thrombosis of the peripheral veins and of the cerebral sinuses has occurred in the later stages of severe chlorosis, and may prove the immediate cause of death.

DIAGNOSIS.—Since chlorosis is a disease in which anæmia is a condition, the diagnosis largely depends upon the time when the symptoms occur and upon the absence of causes of a secondary anæmia. The diagnosis is to be confirmed by the examination of the blood, since most writers regard as essential an excessive diminution of the hæmoglobin in the blood without a proportionate loss of red blood-corpuscles. In the advanced stages of chlorosis the number of red blood-corpuscles as well as the percentage of hæmoglobin may be largely lowered, in which case a knowledge of the etiology and course of the symptoms is essential in order to make a correct diagnosis. Henry maintains that in one series of cases the number and size of the red blood-corpuscles may be normal while the hæmoglobin is deficient, in a second series with a normal number of red corpuscles there may be a diminution of their size while the percentage of hæmoglobin is normal, and in a third series with a diminution in the number of red blood-corpuscles there may be a diminished or an increased quantity of hæmoglobin.

In cases of doubt chlorosis is to be differentiated from secondary anæmias by the etiology of the latter, and from pernicious anæmia by the progressive course of this disease, of which retinal hemorrhages and the examination of the blood furnish additional evidence.

PROGNOSIS.—Chlorosis is rarely fatal, and then usually from such complications as venous thrombosis or ulcer of the stomach. Recurrent attacks are frequent, especially in the autumn and winter, and are sometimes induced by pregnancy, which condition may also relieve the symptoms.

TREATMENT.—The treatment of a case of chlorosis must vary in many ways in accordance with the individual needs of the case. In the mildest form of the affection it may be simply necessary to turn the patient out-doors in the country, to give nutritious, easily digested diet, and to administer iron and arsenic. On the other hand, in the severest case a strict rest-cure (see page 402) may be required. Between these extremes every grade of case exists. Whenever it is practicable, the city girl should be sent to the country, put into warm but light clothing, and required to spend many hours a day in the open air, and the most of the remainder in bed. The exercise must be in proportion to the strength of the patient: any exertion which produces exhaustion or sleeplessness at night, or increased apathy or indifference to exertion the next day, is in all probability deleterious. Complete change of climate, from the mountains to the sea-shore, or from the sea-shore to the mountains, is often very serviceable. Better results are also obtained when the vacation from the city is spent partly in the mountains and partly at the sea-shore. The diet must be varied according to the individual case: the fat chlorotic girl should use carbo-hydrates and fats in moderation; the lean girl, on the other hand, should be given butter, cream, sweet oil, cod-liver oil, and similar substances to the point of gastric tolerance.

Alcohol should never be given in excess, but the moderate use of red wine or of malt liquors at meals may be distinctly beneficial. If, however, they produce flushing of the face, or disturb the stomach, they should be altogether withdrawn.

Two medicines which are essential in the treatment of chlorosis are iron and arsenic, but other drugs are frequently beneficial. Thus, laxatives, preferably vegetable, should be used unless there is already a tendency to looseness of the bowels; bitter tonics and strychnine should be employed when, as is commonly the case, there is failure of appetite, with inactivity of the digestive organs; quinine in moderate doses is often useful, but in large doses may do harm. Active treatment of the amenorrhœa is not often effective or judicious, the suppression of the menstruation being probably a secondary and not a primary phenomenon, which is remedied by the return of health. We have never had satisfaction in the use of apiol, potassium permanganate, or other lauded emmenagogues; the old-fashioned Dewees's emmenagogue mixture (see formula 1) has in our hands given the greatest benefit of any remedy of the class. If at any time a menstrual flow appear, immediate absolute rest in bed should be enforced, with the use of the hot hip bath, an active aloetic or podophyllin purge, and the drinking of hot ginger or other tea. Except in rare cases, local treatment of the pelvic organs, other than the use of douches, is unjustifiable.

In addition to what has been said as to the use of iron (see page 4), it should be stated that in chlorosis the iron should be given in great excess, and that there is no sufficient reason for believing that dried hæmoglobin peptonate or other organic complicated preparation of iron is superior to the older and simpler forms. It will often be found that one preparation of iron disturbs the stomach less than does another. These individual peculiarities can be judged of only by trial, but should be carefully attended to in every case. When iron produces digestive disturbance the preparation should be varied and the doses reduced until toleration is obtained. Arsenic is a valuable remedy, to be given in moderate doses with the iron: Fowler's solution, two drops after meals.

In some cases of chlorosis hydrotherapeutic treatment is of value; when there is not exhaustion, short sea-baths may be useful, care being taken not to produce exhaustion by struggles with breakers, or by the removal of the bodily heat through long immersion in cold sea- or other water. In weak cases quiet baths in warm sea-water are more efficacious. Very commonly the cold douche in the morning, followed by hard rubbing with a towel, is of excellent service: usually the momentary hot douche followed by a momentary cold affusion is very effective. The cold pack, given once or even twice in the day, may be serviceable: the naked person should be enveloped in a sheet wrung out of ice-cold water, and then wrapped in blankets and allowed to stay perfectly quiet from fifteen minutes to one hour. In all cases in which cold water is used, if reaction be not complete harm rather than good will be done by continuing the remedy.

PERNICIOUS ANÆMIA.

This variety of anæmia, sometimes called *idiopathic* or *essential*, and, in virtue of its course, *progressive*, is characterized especially by an excessive diminution of red blood-corpuscles and a not more than corresponding deficiency of hæmoglobin.

ETIOLOGY.—The term progressive pernicious anæmia is based upon the clinical course of certain cases of anæmia of obscure origin. Easily excluded are fatal cases of secondary anæmia from obvious causes, as profuse or repeated hemorrhages, whether from injury or from disease, and cancer or sarcoma of various organs. Atrophy of the mucous membrane of the stomach has been regarded as a satisfactory explanation for the concurrent fatal anæmia. Recent investigations have rendered it possible to eliminate from the group of pernicious anæmias those due to parasites, especially to the *anchylostomum duodenale*, while the *filaria sanguinis hominis* and the *distomum hæmatobium* have long been known as causes of serious if not fatal anæmia.

Pernicious anæmia in the limited sense occurs in both sexes, rather more frequently in the female, and is rare in childhood, although Griffith records the occurrence of ten cases under the age of twelve. Pregnancy and parturition have seemed to be exciting causes in many instances, while sudden or extreme mental shocks have been important antecedents in other cases.

MORBID ANATOMY.—Conspicuous among the lesions found after death is the pale lemon-yellow color of the skin, with but little loss of subcutaneous fat-tissue. The blood is notably pale. The diffuse and speckled opaque yellow color of the heart is indicative of its fatty degeneration, and a yellow discoloration of the kidneys and liver is evidence of a like degeneration of these organs. Fatty degeneration of the glands of the stomach and intestine is also present. Hunter has called attention to an excess of iron, both as ferrated pigment and as colorless iron, in the peripheral region of the hepatic lobules. The kidneys also contain an excess of pigment. The spleen is sometimes enlarged. Lichtheim, Burr, and others have found secondary degeneration in the posterior columns of the spinal cord. Numerous minute hemorrhages, attributed to embolism of the smaller vessels, have been seen in the mucous and serous membranes and in the retina, sometimes in the skin. The marrow of the long bones is deprived of much of its fat, is red, gelatinous (lymphoid), and contains numerous nucleated red blood-corpuscles, especially those of large size,—megaloblasts.

SYMPTOMS.—Vertigo, faintness, palpitation, and rapid breathing on slight exertion, loss of appetite, nausea, vomiting, epigastric pain, and constipation, sometimes diarrhœa, are the chief symptoms. They differ from those of secondary anæmia in being more persistent and progressive, until increasing debility results in confinement to the room, eventually to the dorsal position, and finally to the bed. The temperature is often elevated from 100° to 101° F., sometimes for a period of weeks.

The extreme pallor of the visible mucous membranes and the skin, the latter being of a light lemon-yellow color, is conspicuous, while the absence of emaciation and the frequent presence of abundant subcutaneous fat-tissue are striking. The pulsation of the heart is visible over an increased area, and a systolic or diastolic murmur is to be heard at the apex. Pulsation in the arteries at the base of the neck is often conspicuous, and a venous hum is to be heard on auscultation of the jugular vein. Cutaneous hemorrhages are occasionally to be seen, and retinal hemorrhages are frequently found with the ophthalmoscope. The pulse is rapid and full, and its tension diminished. The urine is high-colored from an excess of urobilin, although numerous observations of a pale urine are recorded, and the urea and urates are increased. A trace of albumin may be present in the later stages of this disease, and Von Jaksch has noted the presence of peptonuria.

Especial importance is to be attached to the examination of the blood. The red blood-corpuscles are diminished in number, and, in extreme cases, may be as few as two hundred and fifty thousand to the cubic millimetre. They show no tendency to form rouleaux. They vary widely in size and shape, microcytes and poikilocytes being numerous, and Browicz and Kollmann have observed active irregular contractions of the latter. Nucleated red blood-corpuscles—normoblasts—are increased in number; but especial importance is to be attached to the presence of large nucleated red corpuscles,—megaloblasts,—which may exceed the former in number. Megaloblasts are also stated to have been found in large numbers in anæmia from malaria and in that due to the presence of the anchylostomum, as also in a variety of severe anæmias which have not proved fatal. Despite the extreme diminution in the number of red blood-corpuscles, the loss of hæmoglobin, which may fall below twenty per cent., is not in proportion, since the individual red blood-corpuscle is frequently more deeply colored than normal. The blood-plates may be increased or diminished in number, and, although the leukocytes are apparently increased from the loss of red corpuscles, and Litten states that a transitory leukocytosis may occur, they are not permanently increased in number.

Diagnosis.—The progressive character of the severe symptoms, and the absence of a satisfactory cause, are of especial importance in the differential diagnosis between the various forms of secondary anæmia and pernicious anæmia. The diagnosis is confirmed by the recognition of retinal hemorrhages, and established by the results of the examination of the blood, especially by the discovery of a greater number of megaloblasts than-of microblasts.

Prognosis.—Progressive pernicious anæmia is almost invariably fatal within the course of one or two years. The reported cases of recovery are to be doubted, since temporary improvement is possible, and recovery may take place from the graver forms of secondary anæmia, the diagnosis of which is capable of being confounded with that of pernicious anæmia until the terminal stages of the latter affection.

TREATMENT.—In pernicious anæmia systematic exercise, high feeding, out-door life, the rest-cure, all hygienic measures which seem indicated in the individual case, should be insisted upon. Iron and tonics are of no avail. The only remedy concerning whose power there is any favorable testimony is arsenic. It should be given after meals in the form of Fowler's solution, beginning with doses of two drops, steadily but slowly increased until a drachm or even a drachm and a half is taken each day, unless intolerance previously occur. The arsenic is generally well borne, but should be temporarily withdrawn when it disturbs the digestive tract. Slight puffiness of the face should not cause the withdrawal of the drug unless the urine contain albumin. Some practitioners prefer to give the arsenic hypodermically: five minims of Fowler's solution diluted with ten minims of water may be injected once a day, and increased as borne. Bone-marrow has been used with alleged excellent results. Red marrow from the small bones of calves or other young animals should be taken, since the marrow of long bones from old animals is chiefly fat. The raw marrow, in capsule, or a glyceride, may be used.

LEUKOCYTOSIS.

DEFINITION.—A temporary increase in the number of leukocytes, especially of the polynuclear variety, in the blood.

The blood normally contains about six thousand white blood-corpuscles to the cubic millimetre, and since the discovery by Ehrlich of the differences in their structure and in their behavior towards staining fluids, their relation to health and disease has been investigated by numerous observers.

A distinction is to be drawn between a physiological and a pathological leukocytosis. The former is found to take place during digestion, beginning a short time after eating. It is also present in infants and young children, and in pregnancy. Exercise, massage, and baths are productive of an apparent increase in the number of white blood-corpuscles, which, however, may be due, as suggested by Thayer, to a local active congestion of the cutaneous vessels, and a corresponding increase in the presence of leukocytes.

A pathological leukocytosis has been observed in a great variety of conditions. It is frequent during the death-agony, possibly from a passive congestion due to an enfeebled heart, and has been found after hemorrhage. It has been repeatedly observed in inflammatory affections of the skin, including those which are the local manifestations of an exanthem (with the exception of measles), and in numerous infectious diseases, in suppurative processes, and in malignant disease. Its absence is conspicuous in malaria, typhoid fever (with rare exception), tuberculosis, and intestinal obstruction.

The presence or absence of a leukocytosis has a definite clinical value both in diagnosis and in prognosis. The constancy of its presence in pneumonia may enable the diagnosis to be made in central pneumonia when typical physical signs are absent. Of still greater diagnostic importance, since it directly leads to the appropriate treatment, is its value in the recognition of deep-seated suppuration. R. C. Cabot mentions its constant presence in suppurative salpingitis, and states that this affection may thus be differentiated from hæmatoma and hæmatocele, and from uterine and ovarian tumors. The recognition of leukocytosis may be of value in permitting the diagnosis of an osteomyelitis. In about one-half of the cases of malignant disease examined by Rieder and Hayem leukocytosis was present; and according to Sadler it may be extreme, sixty to ninety thousand in the cubic millimetre, or may be entirely absent. The leukocytosis of malignant disease is more frequent in sarcoma, especially osteosarcoma, than in cancer. When the uterus is affected with cancer leukocytosis is more frequent than in cancer of the breast, stomach, or œsophagus. The usual absence of leukocytosis in typhoid fever may permit the exclusion of this disease in a case of questionable nature,—for example, acute appendicitis. The occurrence of leukocytosis in typhoid fever has led to the recognition of a complicating pleurisy or pneumonia. Its absence may also prove of diagnostic importance in the exclusion of malaria and uncomplicated acute tuberculosis, in which affections the leukocytes are not increased. A failing leukocytosis in case of doubt would permit acute intestinal obstruction to be differentiated from a general peritonitis, in which affection leukocytosis is present.

The degree of leukocytosis is no evidence of the severity of the disease concerned. In pneumonia it is present, as a rule, throughout the disease despite the pseudo-crises, persisting not infrequently for several days after the temperature has fallen, apparently being closely related to the process of resolution. An absence of leukocytosis in pneumonia is a grave prognostic sign, death usually occurring in such cases.

LEUKÆMIA.

Definition.—A permanent increase in the number of the white blood-corpuscles in the blood, associated with lesions of the spleen, lymphatic glands, or bone-marrow.

Etiology.—But little is known concerning the cause of leukæmia. It occurs more often in males than in females, especially in adults, and is rare in infants and in old age. It is more frequent among the poorer classes, and is epidemic and endemic in certain countries. Its repeated presence in connection with pregnancy, malaria, syphilis, pernicious anæmia, chronic diarrhœa, and injuries to the spleen and bones has suggested that these affections may be of etiological importance. The occasional rapid course and the associated lesions resemble those of an infectious disease, and bacteria have been suggested as a cause, but without any positive evidence of value. Auto-intoxication from the digestive tract has also been mentioned as of importance in the etiology.

MORBID ANATOMY.—The organs especially diseased are the spleen, lymphatic glands, and bone-marrow: hence splenic, lymphatic, and myelogenous varieties of leukæmia are discriminated. Combined lesions are not infrequent, particularly of the spleen and lymph-glands, whence the term spleno- or lieno-lymphatic leukæmia. A purely lymphatic leukæmia is rare, and a purely myelogenous leukæmia is of doubtful occurrence. The spleen may be enormously enlarged, weighing sixteen pounds, the enlargement being due to a hyperplasia, either diffused or nodular. Its color is red or reddish brown, sometimes modified by the presence of tumors, hemorrhage, and fatty degeneration, and its density is increased. The enlargement is either symmetrical or nodular, and on section opaque-gray tumors, leukæmic lymphomata, of various size and number, may be present. The lymph-glands in the various regions of the body may become so enlarged as to cause extreme degrees of deformity. The hyperplastic glands are soft or hard, of an opaque-gray color on section. They may vary considerably in size, and may be discrete or agglutinated. The tonsils, the lymphatic glands at the base of the tongue, the solitary follicles, and Peyer's patches may show like alterations. Similar nodules, leukæmic lymphomata, may be found in the heart, liver, kidneys, retina, brain, cord, lungs, pleuræ, and skin. The liver and kidneys may become enlarged from a diffuse infiltration with the lymphomatous tissue, and a thus affected liver is reported to have weighed forty-two pounds. The alterations of the bone-marrow consist in a disappearance of the fat and an increase in the number of nucleated red blood-corpuscles,—lymphoid marrow,—or in an increase of the large mononuclear marrow-cells,—myelocytes,—which in combination with the excess of leukocytes produce an opaque-yellow appearance,—the pyoid or puriform marrow. Evidences of hemorrhage in various parts of the body may be found, and dropsy is frequent. The blood has a pale pink color, and the clot found in the heart is soft, gelatinous, and sometimes so suggestive of pus as to have led Bennett to regard the disease as a suppuration of the blood,—pyæmia. This alteration in the color of the blood is due partly to the increase in the number of leukocytes, and partly to a deficiency of red corpuscles. The former may even exceed in number the latter, which in normal blood are at least five hundred times as abundant as the leukocytes. Charcot-crystals may form, on exposure to the air, in the blood, bone-marrow, spleen, and liver.

SYMPTOMS.—The incipient symptoms of leukæmia are due to deficient hæmoglobin, and are essentially those of anæmia,—namely, headache, debility, shortness of breath, palpitation, obscure abdominal pain, pallor, and slight œdema. These symptoms become more severe when enlargement of the spleen or lymph-glands is apparent. The pallor becomes more extreme, the appetite fails, diarrhœa may occur, and loss of flesh, as well as of strength, is conspicuous. Hemorrhages are frequent in various parts of the body, either from the nose, stomach, intestine, or urinary or genital tract, or into the lungs, brain, or skin. Hemorrhage into the retina may obscure vision, while hemorrhage into the auditory nerve may be a cause of deafness. Obscured sight and hearing may also result from a lymphomatous growth in the retina or the labyrinth, and Gannoie has recently recorded symptoms resembling those of Ménière's disease from leukæmic growths in the ear. Priapism is an occasional symptom, sometimes lasting several weeks, and has been attributed to a passive venous congestion from the pressure of the enlarged spleen. The pulse is increased in frequency, and is of diminished tension. Respiration is accelerated, in part from the deficiency of red blood-corpuscles, and in part from the obstruction to the descent of the diaphragm when the spleen is enlarged. The temperature may be normal or at times elevated one or two degrees. Anæmic murmurs may be heard on auscultation of the heart. The urine is abundant, high-colored, 1020 to 1027, and the urates are increased.

The enlargement of the lymphatic glands is usually first seen in the neck, the glands in the axillæ, groins, and abdomen being subsequently diseased. The swollen glands are neither painful nor tender, but may press upon neighboring parts, as the veins, causing œdema, or upon the trachea or bronchi, producing dyspnœa. The enlargement of the spleen often produces a conspicuous distention of the abdomen. According to the degree of enlargement its outline is more or less easily determined. The lower edge may lie in the pelvis, the anterior border may extend beyond the navel, and a notched edge is often to be appreciated. Localized tenderness of the surface of the spleen, the result of peritonitic adhesions, may exist. In acute leukæmia these symptoms develop in the course of a few weeks, whereas in chronic leukæmia the process extends over months or years.

The blood is characterized by the excess of white blood-corpuscles, the increase in the number of which is due chiefly to the presence of the large mononuclear variety. The polynuclear leukocytes and eosinophiles are also increased in number, while the percentage of the small mononuclear lymphocytes is diminished. The leukocyte especially characteristic of leukæmia, according to Ehrlich, is the large granular mononuclear neutrophile, almost never found in normal blood, but only in the bone-marrow, whence the term myelocyte. These cells have no amœboid movement, and, although significant of leukæmia when many are present, they may be absent in this disease. The red blood-corpuscles are diminished in number, abnormally pale, vary in size and shape, and nucleated red blood-corpuscles, both small and large, may be absent or numerous. Charcot-crystals may be found, especially in blood rich in eosinophiles.

In the examination of the blood it is important to discriminate between a leukocytosis and a leukæmia. For this purpose the relative proportion of red to white corpuscles is of no value except in extreme cases, since a proportion of one leukocyte to twenty red corpuscles has been found in leukocytosis. In leukocytosis the total number of red blood-corpuscles is usually relatively normal, in leukæmia it is markedly diminished. In leukocytosis, according to Ehrlich, there is a disproportionate excess of polynuclear leukocytes, which normally constitute three-fourths or two-thirds of the total number of leukocytes. It may be impossible from the examination of the blood alone to make a diagnosis of leukæmia in its early stage or when myelocytes are absent. The characteristics of the blood may vary from time to time. A predominance of the large mononuclear forms is suggestive of a conspicuous affection of the spleen, while an excess of the small mononuclear forms indicates an affection of the lymphatic glands, and abundant myelocytes, —myelæmia,—if occurring, would indicate a conspicuous medullary or myelogenous leukæmia. According to Fraenkel, the blood of acute leukæmia is distinguished from that of chronic leukæmia by a preponderance of large and small mononuclear leukocytes and an excessive diminution of the polynuclear variety. In chronic leukæmia there is an increase of all varieties in addition to the presence of the myelocytes. The existence of changes in the bone-marrow is favored by conspicuous tenderness of the bones, although Litten denies the diagnostic importance of this symptom.

Diagnosis.—The results of the physical examination of the blood determine the diagnosis of leukæmia, although enlargement of the spleen or lymph-glands with symptoms of anæmia may suggest its presence. Leukæmic blood has been found in cases in which there were no alterations of the spleen and lymphatic glands, and in pseudo-leukæmia enlargement of the spleen or lymphatic glands is present without increase of the leukocytes. The diagnosis of the especial variety of leukæmia depends upon the results of the physical examination of the spleen, glandular regions, and bones, as well as of the blood. The distinction between leukocytosis and leukæmia based on the examination of the blood has been mentioned above.

Prognosis.—Leukæmia is generally considered to be a fatal disease, although cases of recovery are occasionally reported. Some of these are perhaps cases of leukocytosis, while others may represent a not infrequent temporary improvement which may extend over a period of months. Acute leukæmia is usually of the lymphatic type with abundant small and large lymphocytes, and may terminate fatally in the course of a few weeks. Osler, however, mentions a case of leukæmia of ten years' duration in which, at the end of this time, ninety per cent. of the leukocytes were lymphocytes. Chronic leukæmia is the variety usually seen: it generally extends over a period of two years or more, death eventually resulting from progressive weakness ending in pulmonary œdema or a complicating pneumonia.

Treatment.—The treatment of leukæmia is the same as that of pseudo-leukæmia (see page 19).

Excision of the spleen has been performed twenty-five times, with twenty-four deaths, and is hardly a justifiable operation.

CHLOROMA.

This term is applied in virtue of its green color to a tumor which presents a lymphadenoid structure. It occurs in multiple form in various parts of the body, not especially in the lymphatic glands or the spleen, and is associated with a profound anæmia and with a condition of the blood analogous to that found in leukæmia, the leukocytes being increased as one to five. Dock has recently published an article showing the intimate relation between chloroma and leukæmia. Tumors are found especially at various parts of the head, but also in the internal organs. In the former situation, although presenting the characteristics of sarcoma of the bone, they apparently do not arise from the periosteum. With their extension throughout the body the clinical characteristics are those of a leukæmia, especially of the lymphatic type, which it further resembles in the large proportion of lymphocytes in the blood and in the rapidity of its course, death usually occurring in the course of a few months. It differs from leukæmia in the slight alterations of the lymph-glands and spleen, although the thymus is not infrequently diseased.

In children enlargement of the spleen and lymphatic glands associated with anæmia has been found by Von Jaksch to be accompanied with a persistent leukocytosis. He applies the term pseudo-leukæmic anæmia to these cases, since the lesions characteristic of leukæmia were not found at the autopsy.

PSEUDO-LEUKÆMIA.

Definition.—A disease characterized by persistent and progressive anæmia, enlargement of the spleen or lymphatic glands or of both, but without an excess of leukocytes, whence the designation pseudo-leukæmia.

Synonymes.—Hodgkin's disease, malignant lymphoma, malignant lympho-sarcoma, lymphadenoma, adénie, splenic anæmia, lymphatic anæmia.

Etiology.—Nothing definite is known concerning the origin of this disease. In certain cases local irritation has seemed important, since the glands first enlarged were those receiving lymph from an irritated region. An infectious origin has recently been suggested by Ebstein from the occurrence of cases in which a chronic relapsing fever, splenic enlargement, and multiple lymphomata in the viscera were associated. Malaria, syphilis, and tuberculosis have been assigned an etiological importance. An intimate relation between certain cases of malignant lymphoma and pseudo-leukæmia is suggested by a generalization of the former, while the rare termination of pseudo-leukæmia in leukæmia is indicative of an intimate relation, at times, between the latter affections. H. C. Wood, in 1871, observed that every grade exists between a pronounced leukæmia and a strict pseudo-leukæmia, and that the same patient at one time may show the characteristics of the one disease, while at a later period the phenomena of the other are presented. The disease occurs more often in male infants, children, and adults, less frequently in the aged and in females.

MORBID ANATOMY.—The variations in the shape, size, and consistency of the glands found in leukæmia are to be met with in pseudo-leukæmia, and there is but little tendency to suppuration or degeneration. The longer the enlargement of the glands persists, the more likely are they to be found dense and agglutinated. The glandular affection usually occurs first in the neck, then in the axillary, inguinal, retroperitoneal, mediastinal, and mesenteric glands. These groups of glands may become diseased in continuous sequence, or conspicuous alterations of remote regions may exist, the intervening glandular collections being relatively normal. In some cases the superficial glands may show but little change, and the deep-seated glands, especially the mediastinal and the retroperitoneal, may become extensively diseased. To this variety the term *lymphatic anæmia* is applied.

When the spleen was conspicuously and exclusively altered the term *splenic anæmia* has been applied, but there is no essential difference between the alterations of the spleen when alone affected and when the lymphatic glands are also diseased. The spleen, as in leukæmia, is enlarged, either symmetrically or from the presence of tumors, lymphomata, varying in size and number. The degree of enlargement varies considerably, being less when the lymph-glands are simultaneously diseased than when they are free from alterations.

Abnormalities of the bone-marrow are infrequent, although it may become red, lymphoid, and may contain lymphomatous nodules. As in leukæmia, so in pseudo-leukæmia a pure myelogenous form is of doubtful occurrence. In advanced cases of this disease multiple lymphomata may be found not only in the spleen and bone-marrow, but also in the skin, tonsils, thymus gland, stomach, intestine, liver, kidneys, lungs, central nervous system, and retina.

SYMPTOMS.—Enlargement of the cervical lymph-glands is usually first noticed. With their increase in size and number similar enlargements of the axillary and inguinal glands are observed. The patient then becomes pale, and suffers from the familiar symptoms of anæmia, as palpitation, shortness of breath, disturbed vision, ringing in the ears, failing appetite, and loss of flesh and strength. Eventually hemorrhages and œdema may occur, and obstinate itching of the skin has been observed. With increasing anæmia the patient may become delirious or comatose. An elevated temperature is frequent, even at the outset, and there may be exacerbations and remissions at regular intervals.

Pressure-symptoms are important, especially when the internal lymphatic glands are diseased. Pressure upon the superficial veins gives rise to local œdema, dilatation of the veins, and ulceration of the skin. Dyspnœa results from pressure upon the larynx or trachea, and may be of an asthmatic character when the bifurcation of the trachea is involved. Dysphagia occurs from pressure on the œsophagus. Deafness may result from lymphomata in the pharynx. Hoarseness may follow pressure on the recurrent laryngeal, and palpitation may result from involvement of the pneumogastric nerve. Pain may arise from pressure upon the sensitive nerves, and irregular pupils from pressure upon the cervical sympathetic nerve. When the abdominal glands are enlarged, ascites and jaundice may result from pressure on the portal vein and bile-ducts. Bronzing of the skin sometimes occurs, and pressure upon the inferior vena cava has produced extensive œdema of the legs. The enlarged glands may diminish somewhat during intercurrent febrile attacks, and especially towards the end of life.

The enlarged spleen is readily palpated, but rarely extends below the level of the navel.

The blood shows a deficiency of red blood-corpuscles and hæmoglobin corresponding to the degree of anæmia. Poikilocytosis is inconsiderable, and normoblasts are not especially numerous. The main feature in connection with the lesions is the absence of leukocytosis, although in rare instances the blood may assume a leukæmic character from an excessive formation of lymphocytes, a condition which has suggested that pseudo-leukæmia may represent an aleukæmic stage of leukæmia.

Diagnosis.—Pseudo-leukæmic hyperplasia of the lymphatic glands may be mistaken for enlargement due to tuberculosis, leukæmia, or benignant lymphoma; and A. K. Stone has repeatedly seen an irritative lymphadenitis from vermin mistaken for pseudo-leukæmia. Inflamed and tubercular glands are usually limited to a single region, are less freely movable, and the latter are prone to caseation, softening, and evacuation with the formation of sinuses. Leukæmic lymphomata are to be differentiated by the examination of the blood. Benignant lymphoma is to be differentiated by persistence of the tumor without extension, and by the absence of anæmia or pressure-symptoms. Time alone suffices to make the differential diagnosis clear.

The splenic variety of pseudo-leukæmia is to be recognized from the association of the palpable enlargement of the spleen with the symptoms of anæmia. Leukæmic enlargement of the spleen is excluded by the examination of the blood. The lacking history of malaria eliminates hyperplasia, and amyloid enlargement of the spleen may be excluded by failing evidence of this disease elsewhere, and by the absence of symptoms or signs of tuberculosis and syphilis, the usual antecedents of amyloid degeneration.

Prognosis.—Permanent recovery from pseudo-leukæmia is rare, although temporary improvement with diminution in the size of the glands may take place. The removal of a localized collection of enlarged glands which may have produced a deformity for a number of years is often followed by the rapid development of pseudo-leukæmia. The duration of the disease varies: some cases run a rapid course, terminating fatally within a few weeks, while others, the rule, extend over a period of several years. Cases in which a conspicuous involvement of the internal lymphatic glands exists are more rapidly fatal than those in which the superficial glands are especially diseased.

TREATMENT.—The treatment is the same as that of pernicious anæmia. (See page 11.)

When there are enlarged glands the local use of arsenic is believed by some authorities to bring about disintegration of diseased tissue. Each day there should be injected into a gland not before treated a mixture of equal parts of fresh Fowler's solution and of a two per cent. solution of carbolic acid in water. The first dose should be four drops, and an additional drop should be added daily until twenty drops are reached or toxic symptoms are produced. There may be no immediate disturbance, or there may be local pain for some hours afterwards. Cutaneous inflammation or abscess may follow, or temporary enlargement of the gland, and œdema. As an immediate result of the treatment, the patient may suffer from a bad taste in the mouth, a burning in the throat, thirst, loss of appetite, nausea, vomiting, diarrhœa, abdominal pains, and jaundice. The temperature and pulse may rise. These symptoms demand temporary cessation of the treatment. If the glandular swellings return, a renewal of the treatment is indicated.

MYELOMA.

Von Recklinghausen has recently applied this term to designate a tumor of a lymphadenoid type arising in the bone-marrow. Its structure resembles that of the pyoid marrow found in leukæmia, but its blood-vessels have no defined walls, and its cells and their nuclei are larger. It differs in the manner of its growth, since it produces enlargement, absorption, and perforation of the bone, with extension to the neighboring parts, which do not occur in the leukæmic affections of the bone-marrow. Its clinical course is like that of pseudo-leukæmia, and is manifested by progressive anæmia with the formation of multiple nodules in various parts of the body. Leukocytosis is usually absent, but in a single case presenting the other characteristics of myelæmia a sudden invasion of the blood with leukocytes took place.

HEMORRHAGIC DIATHESIS.

This expression includes a variety of conditions in which hemorrhages, usually multiple, in the skin, from mucous membranes, and in various organs and tissues of the body, are a common characteristic. This series includes factors which are evidently of congenital and inherited origin, as well as others which are acquired. On the one hand, conspicuous importance is to be attached to errors in diet and faulty hygienic surroundings, while, on the other, the probability of the action of various infectious agents is strongly suggested.

It is important to eliminate from the series of diseases under consideration those conditions in which multiple hemorrhages represent a result of well-recognized causes. These are usually included under the term *symptomatic purpura*, since the purple spots resemble those occurring in the disease purpura, the cause of which is not well defined. Such symptomatic hemorrhages occur in consequence of infections, poisons, chronic diseases with conspicuous nutritive disturbance, vaso-motor affections, and mechanical passive congestions.

In the infectious group of symptomatic hemorrhages are included those occurring in the so-called black, malignant, or hemorrhagic scarlet fever, measles, and small-pox. The hemorrhagic condition is also seen in typhus and typhoid fevers, relapsing fever, Oriental pest, cerebro-spinal meningitis, acute articular rheumatism, acute ulcerative endocarditis, influenza, septicæmia, puerperal infections, yellow fever, and cholera. In this series also probably belong the hemorrhages which occur in acute yellow atrophy.

The toxic causes of symptomatic, circumscribed hemorrhage are snake poison, and various drugs, as potassium iodide, phosphorus, mercury, copaiba, ergot, quinine, chloral, belladonna, and alcohol.

The cachectic group includes syphilis, tuberculosis, cancer, nephritis, fibrous hepatitis, pernicious anæmia, leukæmia, and pseudo-leukæmia. Vaso-motor or neurotic hemorrhages may occur in acute and chronic myelitis, multiple neuritis, sometimes in neuralgia, and is most conspicuously seen in the rare cases of stigmata. Cutaneous hemorrhages from passive congestion are especially seen in spasmodic affections like whooping-cough and epilepsy.

The diseases in which hemorrhage is a conspicuous characteristic are hæmophilia, scurvy, purpura, and hæmoglobinæmia.

HÆMOPHILIA.

DEFINITION.—A congenital tendency towards multiple hemorrhages occurring either spontaneously or from trifling injury, often in persons otherwise in good health.

ETIOLOGY.—Heredity is the only known cause of hæmophilia. Hoessli has found evidence of its occurrence in the same family for two hundred and fifty years. The male members are more often affected than the females, although the tendency to bleed is usually transmitted through the latter, even where they are not bleeders.

MORBID ANATOMY.—No satisfactory anatomical basis for the occurrence of the hemorrhage has been found. Virchow has suggested that the pressure of an excess of blood upon the delicate wall of blood-vessels of insufficient capacity may produce the hemorrhage. Abnormally thin vascular walls have been found, and an increased number of red blood-corpuscles has also been observed. Clotted blood may be detected attached to mucous surfaces when copious hemorrhage has occurred.

SYMPTOMS.—The hemorrhages are mild or severe, usually a slow capillary oozing, and may occur as petechiæ or as bleeding from mucous surfaces, especially in the nose and from the gums. Bleeding may also take place from the stomach, intestine, or urinary tract. Senator has reported a case of unilateral renal hæmophilia in a female, diagnosticated by means of the cystoscope, and cured by removal of the kidney. Hemorrhage from the genital tract may be excessive during menstruation or after delivery. Hemorrhage into the joints, especially the large joints, is not infrequent, and may be associated with swelling, pain, and fever suggesting rheumatism. The local symptoms may disappear, or an inflammation of the affected joint follow with permanent deformity which may be mistaken for articular tuberculosis.

The existence of hæmophilia is usually made known in early childhood, and may be manifested immediately after birth by severe umbilical hemorrhage. Slight causes, as bruises, scratches, needle-pricks, cuts, and especially the pulling of teeth, may serve to produce severe, even fatal, hemorrhage.

DIAGNOSIS.—Hæmophilia is to be diagnosticated when frequent and obstinate multiple hemorrhages from trivial causes occur, especially in one whose ancestors have had a similar history. If deformity of the joints results, it is to be distinguished from a tuberculosis of the joints by the absence of a tendency to suppuration and the formation of sinuses.

PROGNOSIS.—Mild and severe cases of hæmophilia occur. The latter may prove fatal in the course of twenty-four hours. The tendency to hemorrhage may disappear in adult life, while frequent and profuse hemorrhages produce a debility which is the frequent cause of the early death of hæmophilic children.

TREATMENT.—The treatment of hæmophilia is exceedingly unsatisfactory, as there are no known drugs which possess the power of altering the inherited tendency, and whilst extract of ergot, oil of erigeron, plumbic acetate, gallic acid, aromatic sulphuric acid, and the whole list of anti-hemorrhagic drugs may be used when hemorrhage occurs, their controlling power is slight. Whenever it is possible the flow of blood should be arrested by mechanical measures. The exhaustion and anæmia which follow excessive bleeding are to be treated upon general principles. The children of such families as suffer through successive generations should by physical culture, open-air life, careful feeding, and other well-known methods be rendered as robust as possible ; although it is doubtful whether the most careful hygienic treatment from early infancy will overcome the constitutional inheritance.

In hæmophilic patients the greatest care should be taken to prevent abrasions or wounds, and the slightest surgical procedure, even vaccination or the pulling of a tooth, ranks in its danger as a major operation.

SCURVY.

DEFINITION.—A disease characterized by mental and physical weakness, anæmia, and frequent hemorrhages, generally occurring among a number of people in a limited locality.

ETIOLOGY.—Scurvy is a disease usually found among sailors or soldiers, prisoners or paupers, or among travellers, especially in the Arctic regions. A number of persons exposed to like conditions are usually affected. Although epidemics and endemics of scurvy are the rule, isolated cases may occur. The best recognized cause is unsuitable food, especially too exclusive a diet. Especial prominence has been assigned to a lack of potassium salts, since many sufferers have been obliged to live for a long time on salted and corned foods in which these salts are lacking. The relief afforded in such cases by fresh meats and a vegetable diet seems an argument in favor of this view, but scurvy may arise among vegetarians, and may be absent among people in the polar regions whose diet contains no vegetables. Although especial importance is to be attached to an exclusive diet, favoring causes are to be found in faulty hygienic surroundings, excessive mental and physical exertion, and, particularly, mental depression. The possibility of an infectious origin for certain epidemics and endemics in Russia is strongly maintained. The term infantile scurvy has been applied of late years to a dietetic hemorrhagic disease of infants which in this work is designated hemorrhagic rickets. (See page 55.)

MORBID ANATOMY.—The anatomical changes are primarily those due to hemorrhages, cutaneous, subcutaneous, intramuscular, and within and in the neighborhood of the joints. They are also to be found in the mucous membranes of the digestive, bronchial, and urinary tracts, in the serous membranes, in the serous cavities, and within the kidney. Swollen and sloughing gums are especial characteristics of the disease. A hyperplastic spleen and parenchymatous degeneration of the heart, liver, and kidneys are also present.

SYMPTOMS.—The hemorrhages occurring in scurvy are usually preceded by a gradual loss of flesh and strength, associated with failing appetite and symptoms of anæmia. After a week or more of malaise, multiple cutaneous hemorrhages make their appearance, at first, usually, in the legs. The hemorrhages are petechial, or occur as patches or flattened nodules. As a rule, the gums, especially near the teeth, soon become affected, and are swollen, soft, and spongy, bleeding freely, and tending to become gangrenous. The teeth are likely to become loose, and may drop out. Hemorrhages also take place from the several mucous membranes, the respiratory mucous membrane being the least often affected. Bleeding from the nose and mouth is the most frequent. Bleeding may also take place into the muscles, joints, and serous cavities. Pains are frequent both in the trunk and in the extremities, and are sometimes referred to the region of the joints, but are not dependent upon local hemorrhages. As the disease progresses the prostration becomes extreme. There are palpitation and dyspnœa on slight exertion. The skin becomes œdematous. Ulcers arise due to hemorrhages, or to inflammation of scars of previous injuries. Necrosis of bone may take place, whilst acute inflammation of the serous membranes, lungs, or kidneys is not infrequent, and may prove the immediate cause of death. In such cases the temperature is elevated, although in the absence of febrile complications it is either normal or subnormal. In the later stages of the disease the patient may become sleepless and delirious, and may suffer from night-blindness or day-blindness, convulsions, or paralysis.

The skin is dry and scaly, and in certain epidemics may present an erythematous, vesicular or papular eruption. The tongue is red and swollen, the breath fetid. The pulse is soft, not increased in frequency. A systolic murmur is heard over the heart. The examination of the blood shows a diminution in number and variation in size of the red blood-corpuscles, and the diminution in hæmoglobin characteristic of anæmia. According to Henry, the blood-count varies between two and five millions, according to the severity of the disease. The urine is dark-colored, has a high specific gravity, and, in severe cases, contains albumin.

DIAGNOSIS.—The diagnosis of scurvy depends upon the coexistence of malaise and weakness with a tendency to hemorrhages from the mucous membranes and under the skin: a peculiar lividity of the spongy gums should always arouse suspicion, unless there be other disease to account for it. When a number of persons exposed to like conditions are simultaneously affected the diagnosis is plain; but even in an isolated case the true nature of the disease should be suspected and a correct decision be arrived at by noting the beneficial effect produced by a suitable change of diet.

PROGNOSIS.—Recovery usually takes place, provided appropriate treatment can be applied before the later stages of the disease are reached, although convalescence is likely to be prolonged. In fatal cases death may occur either suddenly from intracranial hemorrhage or from syncope during undue muscular exertion, or more gradually from intercurrent disease, as inflammation of the lungs, serous membranes, or intestine.

TREATMENT.—In the treatment of scurvy drugs are of very secondary importance, even for the relief of symptoms. Tonics, astringents, especially sulphuric acid, and alcoholic and other stimulants, may be used to overcome failure of appetite, tendency to bloody fluxes, debility, and cardiac weakness, but will rarely act effectually; whilst these symptoms will of themselves rapidly yield to the proper treatment of the underlying condition. Mercurials, alkalies, and all depressing remedies are absolutely contra-indicated.

Lemon juice or its equivalent lime juice may be considered a specific for the disease; from one to two ounces should be given every two to four hours, diluted with an equal amount of water. When obtainable, the juice of fresh lemons is preferable to the preserved juice, though the latter acts favorably. No artificial imitation is of any value. There is no possible contra-indication for its use: the more severe the gastro-intestinal disturbance the more rapidly it should be administered. Next to lemon juice in effectiveness come fresh vegetables; especially active are various cruciferous plants, some of which on account of their growing in remote Arctic or Antarctic regions have been so much used as to have especial reputation. Lettuce, spinach, sorrel, celery, and any vegetable which is taken raw should be eaten freely. Cooked vegetables are distinctly less active. Thus, it is doubtful whether potatoes used in the ordinary methods have any influence over the disease; but scraped raw potatoes have in various emergencies saved life. Apples and other fruits are active antiscorbutics. Any young plants which can be digested should be used when the more suitable foods are not attainable. Fresh air and absolute quiet are useful, but not essential, in the treatment of the disease.

PURPURA.

Definition.—A disease characterized by multiple hemorrhages without obvious cause, frequently associated with rheumatic pains and lesions of various degrees of severity.

It is probable that a number of diseases of differing etiology are included under this designation, although there are only three varieties usually classified as presenting common as well as individual characteristics. These are simple purpura, rheumatic purpura, and hemorrhagic purpura.

SIMPLE PURPURA.

Simple purpura occurs oftenest in children, sometimes in old people, and may be found among both the weak and the strong. It is characterized by the presence of multiple, circumscribed, cutaneous hemorrhages, either petechiæ or ecchymoses, especially the former, which may be limited to the extremities, particularly the lower, or distributed over the entire body. Internal hemorrhages are usually absent, yet hæmaturia is sometimes observed.

Symptoms.—There is but little constitutional disturbance, although loss of appetite, diarrhœa, debility, muscular pains, and slight elevation of temperature may be present. When muscular pains and sensitive joints occur, this variety resembles the milder forms of rheumatic purpura.

Diagnosis.—Purpura is to be distinguished from scurvy by the mildness of its symptoms and by the absence of the peculiar changes in the gums, as well as of the tendency to ulceration of the soft tissues, to inflammation of mucous membranes and internal organs, and to necrosis of bone.

PROGNOSIS.—Recovery usually takes place in the course of a fortnight, although recurrences at intervals of a week or two frequently occur before the health is fully restored.

TREATMENT.—In the treatment of simple purpura the indications are to maintain the bodily health by rest and careful feeding, with nutritive, easily digested food at short intervals, to meet any vital depression which may occur by stimulants, and to use certain remedies because they have been used before, and, unless given in overdose, are incapable of harm. Among the more important of these standard remedies are ergotin, tincture of ferric chloride, and dilute sulphuric acid. As stimulants may be employed quinine, strychnine, alcohol, and digitalis, according to the needs of the individual case. Although the symptoms so closely resemble those of scurvy, lemon juice has no curative power. Arsenic in ascending doses has been strongly recommended by some recent writers.

RHEUMATIC PURPURA.

DEFINITION.—A disease characterized by ecchymoses, various cutaneous eruptions, inflamed joints, and rheumatic pains. It is also called *peliosis rheumatica* and *Schoenlein's disease.*

SYMPTOMS.—It occurs oftenest among young male adults. Its onset is frequently sudden, indicated by moderate fever, loss of appetite, debility, occasional sore throat, and painful swelling of the large joints, especially of the lower extremities. In the course of a few days multiple cutaneous hemorrhages appear upon the lower extremities, then upon the upper, and finally on the abdomen or the chest. The hemorrhages are often associated with urticaria, erythematous nodules, or bullæ. The articular symptoms may disappear with the appearance of the eruption. Albuminuria may occur. Relapses, perhaps several, are frequent, usually occurring in the course of a week or ten days, and recurrences may take place after a considerable interval of time.

DIAGNOSIS.—The diagnosis is based upon the presence of acute polyarthritis, followed by cutaneous hemorrhages, with or without urticaria and œdema. It may be necessary to eliminate the symptomatic hemorrhages occurring in acute infectious diseases, especially those complicated with joint-affections like acute articular rheumatism and scarlet fever. In rheumatic fever the articular affections precede by a considerable interval the hemorrhages which are due to acute endocarditis. In scarlet fever the hemorrhages are more general, and precede the affections of the joints. Scurvy may be simulated, since muscular and articular pains may be present in this affection, and the gums may be affected in rheumatic purpura. But rheumatic purpura is an acute febrile disease from the outset, and lacks the preliminary cachexia and the dietetic etiology of scurvy.

PROGNOSIS.—The prognosis as to the individual attack is favorable, recovery usually occurring in the course of two or three weeks. The liability to relapses and recurrences has already been mentioned.

TREATMENT.—In rheumatic purpura the general management of the case is that of simple purpura, but we have seen very remarkable and positive effects from the exhibition of salicylates, preferably of ammonium salicylate, which should at first be given until it produces distinct tinnitus aurium, and afterwards be administered in small doses. When there is a marked tendency to relapses the continuous use of the salicylates has been in our experience effective. A mixture of the ammonium and strontium salts is preferable. From time to time the administration of the drug should be interrupted, for fear of producing too much depression.

HENOCH'S PURPURA.

Of late attention has been called, especially by Henoch, to a severe type of rheumatic purpura in which the onset is often violent and the arthritis, cutaneous hemorrhages, eruptions, and œdema are associated with hemorrhages from the mucous membranes, abdominal pain, vomiting, and diarrhœa. In some cases the œdema is conspicuous, in others the hemorrhagic spots or erythematous nodules, and in still others the gastro-intestinal symptoms. In addition to an elevated temperature a splenic tumor is often found, and hemorrhagic nephritis has been observed in a number of reported cases. Recurrences are frequent, and the disease may then be prolonged over a period of months. Quincke has called attention to a circumscribed œdema called angioneurotic, which may be recurrent and associated with effusions into the joints, hemorrhage from the mucous membranes, vomiting and colic, lasting for hours or days. It rarely presents the characteristics of an infectious disease. Osler suggests that rheumatic purpura and angioneurotic œdema may be closely related and due to a toxæmia.

PURPURA HÆMORRHAGICA.

DEFINITION.—This affection, also called *Werlhof's disease*, or *Morbus maculosus Werlhofii*, is characterized by extensive hemorrhages into the skin and from the mucous membranes, associated with painful and swollen joints.

ETIOLOGY.—Girls or young women are more likely to be affected, especially those living under bad hygienic conditions. Its infectious origin is suggested by the simultaneous disease of mother and fœtus, and by the experiments of Petrone, who produced multiple hemorrhages in rabbits by introducing blood from a diseased patient. Letzerich has found in three cases a bacillus, the inoculation of pure cultures of which produced symptoms resembling those of hemorrhagic nephritis. Kolb also has inoculated animals with cultures of a bacillus found in fulminating purpura, with the production of purpuric spots and internal hemorrhages.

MORBID ANATOMY.—Hemorrhages may be found within the skin, the mucous and serous membranes, and more rarely within the serous cavities and in the joints. Enlargement of the spleen is frequent.

SYMPTOMS.—The progress of this affection resembles that of an acute infectious disease. After several days of loss of appetite, perhaps vomiting, muscular pains, painful and swollen joints, and elevated temperature, numerous multiple cutaneous hemorrhages occur, especially on the lower extremities, and tend to become confluent. Extensive hemorrhages also take place from the mucous membranes, particularly of the digestive and urinary tracts. Bronchial and pulmonary hemorrhages are rare. In the milder cases the patient may recover in the course of a fortnight, although recurrences are not infrequent, the disease then extending perhaps over a period of months. In the severer cases—*purpura fulminans*—death may occur in the course of twenty-four hours, or at a later period, from collapse or intracranial hemorrhage. In the latter series of cases there may be colic and diarrhœa with high fever and a typhoid state, and endocarditis and hemorrhagic nephritis may be found as complications. Apparently mild cases may become severe.

DIAGNOSIS.—Scurvy is to be differentiated by the usual restricted occurrence, the absence of a preliminary cachexia and faulty diet, and the freedom of the gums from disease. Simple purpura is to be excluded by the severer symptoms and the occurrence of internal hemorrhages, while purpura rheumatica is to be differentiated by the absence of conspicuous primary affections of the joints. The fulminating cases may be confounded with an apoplectiform hemorrhagic exanthem, especially hemorrhagic small-pox, and with cerebro-spinal meningitis. These affections are to be excluded by the slight fever and the absence of epidemics.

TREATMENT.—The treatment of purpura hæmorrhagica is that of simple purpura, combined with the free administration of gallic acid, oil of erigeron, ergot, turpentine, aromatic sulphuric acid, and other of the internal hæmostatics, whose control over the bleeding is, however, untrustworthy. The subsequent anæmia should be treated in the usual way with iron and tonics.

HÆMOGLOBINÆMIA.

A term applied to the presence of free hæmoglobin in the blood. This condition is due to a variety of causes which result in the escape of hæmoglobin from the red blood-corpuscles and its solution in the blood-serum. According to Ponfick's researches in particular, such dissolved hæmoglobin when in small quantity is disposed of by the spleen and liver. If the quantity set free is excessive the kidneys eliminate the excess and hæmoglobinuria results. For the further consideration of this subject, see Hæmoglobinuria.

DISEASES OF THE SPLEEN.

Although the spleen plays an important part in disease, its alterations are usually secondary to disease elsewhere, and the associated symptoms are rather due to the primary affections than to accompanying modifications in the function of this organ, the physiology of which is so little known.

The physical exploration of the spleen is, however, of decided importance to the physician, enlargement of this organ being of frequent occurrence in a number of diseases, especially those due to an infection of the blood. The establishment of the diagnosis of typhoid fever, septicæmia of obscure origin, malaria, fibrous hepatitis, and amyloid degeneration may be largely aided by the physical examination of the spleen. For this purpose percussion may be of but little importance, although an oval-shaped area of dulness due to the spleen is normally found between the ninth and eleventh left ribs. Gaseous distention of the stomach or colon, or fluid in the pleura, or solidification of the lung, however, may so modify the results of percussion as to prevent satisfactory recognition of the position of the spleen. Fortunately, the alterations of the spleen of diagnostic importance are those due to enlargement of this organ, any considerable degree of which is to be appreciated by palpation. It is to be remembered that the lower edge of a normal spleen may be felt when displaced in consequence of deformity of the chest or spine causing a permanent depression of the diaphragm. For examination, the patient should lie either on the back or on the right side with a slight backward inclination, preferably with the thighs flexed. In the former case the physician applies light but firm pressure with the finger-tips of both hands closely approximated upon the abdominal wall in the left hypochondrium from below upward. As the patient takes a long breath the edge of the enlarged spleen may be felt as it descends below the costal cartilages. If the patient is lying on the side, the physician should face the back of the patient and should press with the finger-tips of both hands against the abdominal wall in the left hypochondrium from below upward, or should apply pressure with the finger-tips of the right hand upon the abdominal wall from below upward, the left hand being firmly pressed against the lower ribs. As the patient draws slowly a long breath the edge of the enlarged spleen may be felt to strike against the fingers.

Enlargement of the spleen may be either acute or chronic, the former rarely resulting in any great increase in the size of this organ, the latter giving rise to some of the largest abdominal tumors. Acute enlargement is usually occasioned by infection, although it may be due to congestion from injury or embolism, and palpation of the lower border of the spleen may offer important evidence concerning the existence of an infectious process. An acute splenic enlargement is always to be sought for in malaria; it is present towards the end of the first week in typhoid fever. The viscus is usually somewhat sensitive to palpation. The associated symptoms serve for the recognition of typhoidal enlargement of the spleen, while the symptoms or examination of the blood establish the diagnosis of malarial enlargement. In chronic enlargement the spleen is superficial, smooth, resistant, with a sharply defined lower edge and sometimes a lobulated border near the navel. It is to be distinguished from the kidney by its superficial position and the absence of an overlying colon, while the mobility of an enlarged spleen on inspiration is greater than that of an enlarged kidney. Chronic enlargement of the spleen of moderate degree may result from malaria and from chronic passive congestion in fibrous hepatitis. The more extreme degrees of chronic enlargement are due to leukæmia and pseudo-leukæmia, while amyloid degeneration produces both the lesser and more considerable degrees of enlargement. The leukæmic enlargement of the spleen is to be differentiated from the pseudo-leukæmic hyperplasia by the examination of the blood. A diagnosis of amyloid infiltration of the spleen is to be made when the enlargement of this organ is associated with an enlargement of the liver or with albuminuria, dropsy, and chronic diarrhœa in the sequence of chronic suppuration, especially of bones and joints, and in that of tuberculosis and syphilis. A congenital enlargement of the spleen without amyloid degeneration is important evidence of inherited syphilis.

MOVABLE SPLEEN. WANDERING SPLEEN. SPLENOPTOSIS.

ETIOLOGY.—Excessive mobility of the spleen occurs as the result of congenital or acquired conditions, the former being represented by an abnormally long ligament, the latter by sudden muscular violence, protracted muscular strain, or increased size of the organ. Prolapse of the spleen, *splenoptosis*, at times is a part of the general *splanchnoptosis*, in which a prolapsed stomach, colon, liver, kidney, uterus, and ovaries may be conjoined.

SYMPTOMS.—There may be no disturbance resulting from a wandering spleen, or the patient may complain of a sensation of discomfort in the left side, perhaps associated with the feeling of a movable object in the abdomen. Pain in the left shoulder is at times complained of, and is explained by the communication between the splanchnic and pneumogastric nerves by means of the semilunar ganglion. The movable spleen may be found in the left iliac fossa or to the right of the navel, and has been confounded with a tumor of the kidney, uterus, or ovary. It is usually freely movable, its hilus directed upward, and it may be returned to the left hypochondrium, in which the normal area of splenic dulness is absent. The mobility of the spleen may be so great as to cause a twisting of its ligaments and vessels. *Perisplenitis* is then likely to arise, and is manifested by the symptoms of a localized peritonitis. Adhesions may thus be formed between the spleen and the stomach or intestine, and dilatation of the former or obstruction of the latter result.

The twisting may be so considerable that enlargement or atrophy of the organ follows. In the former event the discomfort is aggravated, whereas the atrophy is likely to afford relief to the symptoms. In rare instances the twisting may be so extreme as to cause a complete detachment of the spleen.

TREATMENT.—For treatment of malarial enlargement of the spleen, see page 212 ; other splenic enlargements are not amenable to any known medicinal or hygienic treatment. In great enlargement of the spleen, and in wandering spleen, an abdominal bandage is often of service.

EMBOLISM AND ABSCESS OF THE SPLEEN.

Embolism of the spleen is of frequent occurrence, and abscess of the spleen is usually a result of infectious embolism, although sometimes due to localized inflammation in such infectious diseases as typhoid and relapsing fever, septicæmia, pyæmia, and cholera, or from the extension of an inflammatory process from the peritoneum, stomach, kidney, or lung. Bland embolism of the spleen follows the transfer of an embolus from the aorta or the left side of the heart, except in the rare cases when an embolus from a venous thrombus passes through an open foramen ovale from the right auricle into the arterial circulation. The diseases in which such embolism is likely to occur are chronic endaortitis, valvular endocarditis, and interstitial myocarditis. Hemorrhagic infarction and an eventual scar result. The quantity of spleen destroyed is chiefly determined by the size and number of the emboli. The embolus may be sufficiently large to obstruct the main splenic artery, in which case a consequent thrombosis of the splenic vein may extend into the portal vein, producing thrombosis of the latter.

The occurrence of splenic embolism is to be suspected when a sudden attack of pain in the splenic region is associated with a chill and followed by enlargement of the organ in a person presenting the conditions favorable to arterial thrombosis, especially chronic valvular endocarditis. Although recovery from bland embolism of the spleen is the rule, the exception above stated may take place, and the patient die from hemorrhagic infarction of the intestine and acute peritonitis dependent upon secondary thrombosis of the intestinal branches of the portal vein.

If the embolus is infectious, as in malignant endocarditis, one or more abscesses of the spleen, tending to become confluent, follow the mechanical results of embolism. A similar result is seen in abscesses of the spleen from the other previously mentioned causes. The entire spleen may be transformed into a bag of pus, surrounded by an acute perisplenitis. Perforation may result, with the production of a general peritonitis, or the abscess may be evacuated into the stomach, the intestine, or the pelvis of the kidney ; the pus may also escape into the pericardium, or through the diaphragm into the lungs, or through the abdominal wall.

Abscess of the spleen can usually be recognized by the existence of constitutional disturbance indicative of pus-formation, such as recurring chills or fever, profuse sweating, diarrhœa, loss of flesh and strength, associated with localized pain, rapid increase in the size of the spleen, with marked tenderness, and in some cases audible or even palpable friction.

The prognosis of abscess of the spleen is very serious, death frequently resulting from septicæmia even after the successful spontaneous or surgical evacuation of the pus. The aspirator should always be used, at least for diagnostic purposes, but, pus being found, we believe it better to open the abscess by free incision. It is necessary, however, in such cases to defer the operation until adhesions have been formed between the spleen and the abdominal walls.

DISEASES OF THE THYROID GLAND.

Until within the past few years any radical treatment of the diseases of the thyroid gland was almost exclusively the province of the surgeon. Inflammation was extremely rare, except as a secondary process during the progress of a goitre, and, although resolution might take place, the more frequent result was an abscess requiring the use of the knife. In like manner tumors of the thyroid, whether benignant or malignant, the latter including the rare malignant adenoma as well as the sarcoma and cancer, were regarded as surgical affections. The discovery by Murray of the marvellous effects of thyroid extract in myxœdema has led to its use in the treatment of other affections of the thyroid with conspicuous success in a number of cases.

GOITRE. BRONCHOCELE. STRUMA.

DEFINITION.—A persistent enlargement of the thyroid gland.

ETIOLOGY.—This disease is endemic in various parts of the world, particularly in mountainous regions. Osler and, more recently, Dock have called attention to its occurrence in the regions bordering upon the great lakes of North America, principally in Michigan and Canada. Munson finds that goitre exists among the North American Indians, among whom it is most prevalent in the southern part of Montana. According to him, its distribution follows the course of the Rocky Mountains, and is independent of high altitude, climate, or excess of calcium salts. Both cretinism and exophthalmic goitre are rare among these people. The immediate cause of goitre is unknown, although Virchow concluded that it was likely to exist in the drinking-water. Heredity is sometimes conspicuous. Females are more often affected than males, and the disease usually first appears in young persons near the age of puberty. In goitrous regions domesticated animals may be affected, especially horses and dogs.

MORBID ANATOMY.—The disease begins as a local or general hyperplasia, due to an excessive formation of the follicles: hence the term adenoma of the thyroid or hyperplastic or follicular goitre. The enlargement of the gland may be chiefly composed of the new-formed follicles, although in some cases an excess of fibrous tissue, in others, of vascular tissue, is formed: hence fibrous and vascular forms of goitre. The hyperplastic cells are prone to undergo a hyaline, gelatinous, or colloid degeneration: thus cavities of various size arise within the tumor, filled with colloid material of greater or less density,—colloid goitre. By the absorption of the walls intervening between such cavities cysts are formed, which are often numerous and large, containing chiefly liquid contents, and give rise to the term cystic goitre. Lime salts are often deposited in the connective tissue of the goitre, producing a calcification or ossification of the wall. The enlarged thyroid forms a tumor not infrequently of the size of the fist, and it has attained the size of a man's head.

SYMPTOMS.—The disturbances produced by the goitre result from pressure upon neighboring parts, and do not arise until the gland has attained a considerable size. A rapid growth of the tumor not infrequently takes place, with a corresponding increase in the severity of the symptoms. Most important among these is dyspnœa from pressure upon the trachea. The larynx may also be compressed, with modification in the character of the voice and respiration. Pressure on the œsophagus may produce difficulty in swallowing, and pressure upon the veins, especially when the growth of the thyroid extends beneath the sternum, has produced fatal thrombosis. Pressure upon the sympathetic nerve may cause a narrowed pupil. Patients with goitre sometimes suddenly die and no obvious cause of death is discovered; such an event is usually attributed to asphyxia from sudden compression of the trachea or paralysis of the vocal cords.

PROGNOSIS.—Goitres may rapidly disappear, especially in young persons, but only in the absence of calcification and cystic degeneration. They usually persist throughout the life of the patient.

TREATMENT.—Fibrous, cystic, amyloid, colloid, and calcareous degenerations of the thyroid gland are not amenable to any medical treatment, and therefore belong to the province of surgery. When degeneration has not supervened, the tumor frequently disappears spontaneously upon removal to a non-goitrous district, or it may yield to the internal administration of iodine, Lugol's solution, ten to twenty drops, three times a day (well diluted), aided by the free use of iodine ointment. Ergot has been recommended by numerous authorities, given internally in full doses. Simple and ferruginous tonics are sometimes of service. Bruns states that out of twelve cases he succeeded in curing nine with the thyroid extract. We have seen the remedy tried in one case with most happy result. Upon old cases with continuing residence in the infected district medicines have no curative influence.

EXOPHTHALMIC GOITRE. GRAVES'S DISEASE. BASEDOW'S DISEASE.

DEFINITION.—A disease especially manifested by disturbed circulation, protruding eyeballs, goitre, and muscular tremors.

ETIOLOGY.—It is found more often in women than in men, usually in adult life, although it has been observed in the young and in those of advanced years. It usually occurs in persons of inherited sensitive nervous organization. The disease itself may be inherited, and it may be present in several members of a family.

A practical distinction is to be drawn between the primary or essential variety of exophthalmic goitre and the secondary variety, in which similar symptoms may occur in the course of simple goitre, pregnancy, or affections of the nose. Abortive or doubtful forms of exophthalmic goitre, "*formes frustes*," are usually regarded as representing mild varieties of this disease.

Immediate causes in persons predisposed may be emotional excitement, prolonged mental or physical strain, or severe acute disease.

MORBID ANATOMY.—A variety of anatomical changes have been found in exophthalmic goitre, most of which are rather a result than a cause of the disease. Among these are hypertrophy and dilatation of the heart, inflammation of the endocardium and pericardium, and degeneration of the myocardium. Lesions of the sympathetic nervous system, as hypertrophy or atrophy, sclerosis, and pigmentation, have been observed, but are not essential. The alterations present in the thyroid are those occurring in simple goitre, vascular dilatation being the most frequent.

The conspicuous nature of the nervous symptoms has led numerous observers to regard this disease as a nervous affection, localized more particularly in the sympathetic nervous system, either at its origin or in its course. Although local lesions are sometimes found, they are neither constant nor characteristic. The frequent mental and motor disturbances were attributed to lesions in various parts of the brain. If none were found, disturbances of circulation in the regions concerned were assumed as an explanation. At the present time the theory advanced in 1891 by Möbius prevails. According to this, the resulting disturbances are due to a toxæmia dependent upon a pathological activity of the thyroid gland. This view is based upon a comparison of the symptoms of exophthalmic goitre and those of myxœdema, in which affection there is atrophy of the thyroid and of cachexia strumipriva following extirpation of the thyroid in man and animals. Certain of these symptoms are antagonistic: *e.g.*, in exophthalmic goitre there are acceleration of the pulse, profuse perspiration, and increased mental excitability, while in myxœdema and cachexia strumipriva a slow pulse, dry skin, and sluggish mind occur.

Exophthalmic goitre and myxœdema may be present in the same family; the latter may follow the former, or the two may concur in the same individual. Goitre may be followed in the course of years by symptoms of exophthalmic goitre, and symptoms of exophthalmic goitre may rapidly follow extirpation of the diseased thyroid. Thyroid extracts in myxœdema and in health produce some of the symptoms of exophthalmic goitre, and are not well borne in certain cases of the latter disease, although sometimes the goitre is diminished and the patient is improved. The theory that disease of the thyroid causes exophthalmic goitre is further favored by the relief to this disease which sometimes follows removal of the thyroid.

SYMPTOMS.—The symptoms of exophthalmic goitre are usually of slow and gradual development, but they may suddenly arise and rapidly progress. They are generally regarded as neuroses,—that is, as functional disturbances without obvious cause. A rapid pulse is one of the most constant symptoms. The average beat of the pulse may be upward of a hundred, and upon exertion or excitement its frequency may be nearly doubled. The patient complains of palpitation, and a tumultuous throbbing of the heart, carotids, and abdominal aorta is visible. Murmurs may be heard over the heart and the large arteries, and there is accentuation of the valvular sounds.

Protrusion of the eyeballs, exophthalmos, when present, usually follows the circulatory disturbance. It is apparently due to the presence of an increased quantity of blood or lymph in the orbit, since it may take place suddenly, may vary at different times in the same individual, and disappears after death. It is sometimes so extreme that the lids cannot be closed. With the persistence of the disease increase of the orbital fat-tissue may take place, causing permanent protrusion of the eyeballs. Graefe discovered that in certain instances the upper lid remains immovable instead of following the eye when it is turned downward, and Stellwag called attention to an increased separation of the eyelids due to retraction of the upper lid even when there is no protrusion of the eyeball, while Möbius observed an inability of the eyes to converge upon an object in the immediate vicinity. The pupil is usually unaltered, and vision is undisturbed. Ulceration and opacity of the cornea sometimes take place from the unprotected condition of the eye or from a disturbance of its trophic nerves.

The enlargement of the thyroid is essentially due to a dilatation of its blood-vessels, especially the arteries. It gradually increases in size, but may undergo sudden and rapid changes of volume when the patient suffers from attacks of palpitation. Follicular enlargement may also take place, and colloid degeneration and calcification may occur. The enlargement may be partial or total. There are often visible pulsation, a palpable thrill, and a double systolic murmur. Guttmann regards the presence of the last as characteristic of Graves's disease, not finding it in simple goitre. The experience of Dock agrees with that of Guttmann, although Mannheim states that it was absent in fourteen out of thirty-seven cases. He admits the possibility that it may be present at some time in every case of exophthalmic goitre.

Persons suffering from Graves's disease are usually neurasthenic or easily excited or depressed, and may become maniacal or melancholic. General muscular tremors, superficial, occurring every few seconds, are so constant as always to be sought for. Spasmodic movements of the muscles resembling those of chorea may take place, or epileptiform attacks occur, while muscular weakness, both general and local, is frequent, the inspiratory expansion of the chest even being diminished. Joffroy observed that the forehead failed to contract when the patient with head bent forward was told to look up without raising the head.

The respiratory tract may be affected, as shown by a spasmodic cough and rapid breathing. The voice may be feeble. Vomiting and diarrhœa, frequent micturition, increased quantity of urine, albuminuria, and glycosuria may be present, and disturbances of menstruation are frequent. There may be temporary febrile attacks. The skin flushes readily, and *taches cérébrales*, the red streaks produced by drawing the finger-nail over the skin, are easily induced. Sweating is frequent, and pigmentation, scleroderma, herpes, urticaria, and circumscribed or general œdema are occasional symptoms. Charcot has observed a diminution in the resistance of the skin to the galvanic current. There may be premature loss of hair and teeth, and the hair may early turn gray. Epistaxis, hæmoptysis, and gastro-intestinal hemorrhages may occur. With the persistence of the symptoms the patient loses flesh and strength and becomes pale.

DIAGNOSIS.—In the presence of the three characteristic symptoms, namely, tachycardia, goitre, and exophthalmos, the diagnosis is easy. The goitre may be small or absent, and exophthalmos is usually of late development, although it may exist for years before the other symptoms arise. Tachycardia is the most constant symptom, and if persistent and accompanied by muscular tremor and a number of the symptoms above mentioned, the diagnosis may be made even in the absence of goitre and exophthalmos. Time may, therefore, be essential to the diagnosis, since a tachycardia may prove to be an early symptom of exophthalmic goitre, and a characteristic grouping of the symptoms may not appear until fifteen years after palpitation has existed.

PROGNOSIS.—Complete recovery from genuine exophthalmic goitre rarely takes place, although temporary improvement is not infrequent. The disease extends over a period of years, and death usually is due to some intercurrent disease, although it may result from rapidly developed acute mania or progressive emaciation and debility. The prognosis is more favorable in the secondary variety, in which enlargement of the thyroid has preceded other symptoms by a period of years, or in which the symptoms rapidly follow fright or occur in nasal affections and pregnancy.

TREATMENT.—There is no known specific treatment directly curative of exophthalmic goitre. Preparations of the thyroid gland have been much administered, but in our experience, which finds corroboration in the printed records, they have distinctly aggravated the symptoms. We have seen spontaneous and permanent recovery occur during an acute splenitis ending in abscess, and have in three cases used glycerin extract of spleen (ten to twenty minims given hypodermically daily) with apparently marked benefit. Pronounced amelioration may often be obtained by change of climate; in our experience high elevations have greatly aggravated the cardiac distress, and much benefit has been derived from a sea-shore residence. On the other hand, Oppenheim affirms that living in the Alps at an elevation of from three to five thousand feet in the earlier stages of the disease often produces a notable good effect. Prolonged rest in bed, with massage, and a more or less strictly carried out rest-cure, are frequently of temporary service, and should from time to time be resorted to when the symptoms become severe. In the acute cases absolute rest in bed should be enforced. The application of Leiter's tubes or of an ice-bag over the heart or over the thyroid gland itself for half an hour to an hour at a time sometimes distinctly quiets the heart's action, but is not always even temporarily beneficial. The cardiac drugs have very little influence over the rapidity of the pulse. In robust cases with evident excess of cardiac power, aconite may be carefully tried. and occasionally brings relief. In failing heart digitalis and strophanthus are indicated, but are rarely effective; some authorities prefer strophanthus to digitalis. Belladonna given in ascending doses until it produces marked dryness of the mouth or even slight dilatation of the pupil sometimes gives relief. Extract of ergot in large doses is commended by some writers. Neither arsenic nor iodine has any distinct control over the disease. When anæmia exists, a non-astringent preparation of iron should be administered.

Very good results are alleged to have frequently followed the use of electricity in Graves's disease. Vigouroux especially recommends the faradic current applied by means of a broad anode to the back of the neck and a smaller cathode placed over the sympathetic ganglia in the front of the neck and afterwards shifted to the motor points of various muscles of the face and neck, and still later replaced by a large cathode placed over the heart region and the sternum, a very strong current being used to the sympathetics, a weaker one to the heart region. Most authorities, however, prefer the galvanic treatment, the usual plan being to put the cathode just behind the angle of the jaw with the anode over the heart or upper sternum. It is probable that the direction of the current makes little difference: thus, Osler directs that the cathode should be placed at the back of the neck and the anode over the heart, whilst

Erb states that the anode should be placed upon the cervical spine and the cathode upon the peripheral nerves. If direction makes any difference, it is probably better to send the current down the nerve. The current should be of such strength as to produce slight pain. There can be no doubt that the heart is often immediately slowed, probably by irritation of the pneumogastric nerve, but it is very doubtful whether the electrical treatment of Graves's disease is ever effective. The séances must be carried over months, and any changes which occur in the course of the disease are probably due to psychical impression, or to other measures instituted, or represent the apparently spontaneous remissions of the disease which occur from time to time.

Graves's disease has been treated surgically by electrolysis with asserted advantage, and also by ligation of the thyroid arteries and by excision of the thyroid gland, which must be partial, otherwise myxœdema will result. According to Osler, "out of sixty-eight operations on record, eighteen completely recovered; in twenty-six there was more or less improvement; nine showed no change; in five death was almost immediate, and in four death occurred within twenty-four hours."

Under the name of *exothyroipexia* Jaboulay has introduced a new operation: an incision having been made in the median line, the gland separated from the trachea is left covered simply by an antiseptic dressing. It is said to decrease rapidly in size, and when the norm has been reached the wound is closed.

MYXŒDEMA.

Definition.—A disease especially characterized by an infiltration of the connective tissue, especially beneath the skin, with a gelatinous substance, by mental sluggishness, and by atrophy of the thyroid gland.

It has long been known that there is an intimate relation between alterations of the thyroid gland and cretinism. The latter affection occurs in regions in which goitre prevails, and if the children of families removing into such regions were goitrous, those born after such removal became cretins. It has, therefore, been considered probable that a common etiological factor exists in goitre and cretinism, both of which conditions may be combined. Endemic cretinism results in a stunting of the body and a blunting of the intellect, each varying considerably in degree. In congenital or sporadic cretinism these alterations are still more conspicuous. The body is dwarfed. The subcutaneous tissue is exuberant, and many of the mental and physical characteristics resemble those to be found in myxœdema. The thyroid gland is atrophied, or absent in some cases. This condition is now generally considered as a congenital variety of myxœdema.

In 1873 Sir William Gull reported a series of cases of myxœdema under the title "On a Cretinoid State supervening in Adult Life in Women." A condition was described with resemblances to and differences from cretinism. A few years later Ord published the appearances found after death, and introduced the explanatory term myxœdema. It soon became evident that atrophy of the thyroid gland was present, and importance was attached to this fact. It was then found, especially by Kocher and Reverdin, that total extirpation of the thyroid gland in the treatment of goitre, especially in young persons, resulted in the production of mental and physical debility, progressive anæmia, and swelling of the subcutaneous tissue. To this condition the term *cachexia strumipriva* was applied. Horsley observed that a similar condition resulted from the removal of the thyroid in the lower animals.

Myxœdema is thus to be distinguished from sporadic cretinism and cachexia strumipriva in virtue of its occurrence independently of the above-mentioned causes.

ETIOLOGY.—This affection exists much more often in women than in men, rarely in children, except as sporadic cretinism. It may occur in several members of a family. It may follow or concur with goitre, and exophthalmic goitre and myxœdema may be present in members of the same family, even in the same individual.

MORBID ANATOMY.—The subcutaneous fat is diminished or increased, and is often replaced by a gelatinous material which becomes opaque on the addition of acetic acid. The fat-tissue elsewhere in the body may be increased. The thyroid gland is atrophied or absent, and the parenchymatous portion may be atrophied, although the gland is enlarged. The pituitary body has been found increased in size in a number of cases of myxœdema, as well as in cretinism.

SYMPTOMS.—There is bloating especially of the face and neck, perhaps of other parts of the body, and supraclavicular swellings are frequent. The skin does not pit on pressure, and the bloating is evidently due to an increase in the volume and density of the subcutaneous tissue, to which the skin is not intimately adherent as in scleroderma. In consequence of this condition the physiognomy is altered. The face is broadened; the lineaments and wrinkles are obliterated; the lips are thickened and everted; the tongue may become enormously enlarged, interfering even with the swallowing of saliva, which may flow from the mouth, and tumors may form beneath the lower jaw from infiltration of the submaxillary glands. The face presents a pale, mask-like appearance suggesting that of a chalked clown, although a reddish patch may appear on the cheeks. The skin, especially of the hands, is coarsely wrinkled, dry, rough, and scaly, and of a brownish tint. The nails are frequently atrophied and brittle. The hair becomes coarse and dry, and premature loss of hair and teeth often occurs.

Mental symptoms are also striking. The patient becomes phlegmatic, dull or stupid, loses interest in her surroundings, and is slow of speech. Memory fails. Hallucinations, especially of sight, are frequent, and the tendency is towards dementia. Numbness and neuralgic pains are often early symptoms, and muscular weakness is complained of. The patellar reflex may be diminished. The pulse is slow, the temperature is subnormal, and the hands and feet are cold. Leukocytosis has been found, and hemorrhages from the mucous membranes sometimes take place. Albuminuria is frequent, and casts may be present. Glycosuria is sometimes observed, and in a case recently under observation we found an almost pure albumosuria, the precipitation with nitric acid resembling that caused by one-half per cent. of serum albumin. The disease is usually slowly progressive, extending over a period of years, but Osler reports a case regarded as myxœdema in which, although there was enlargement of the thyroid, the bloating of the face gradually disappeared after persisting three or four months.

DIAGNOSIS.—The appearance of the patient is suggestive of a dropsy, but the skin does not pit on pressure. The expressionless face, the peculiar hands, and the mental sluggishness are sufficiently characteristic. Albuminuria and casts are temporary, and therefore do not indicate a chronic nephritis.

PROGNOSIS.—The prognosis is favorable, the symptoms rarely disappearing spontaneously, although amenable to treatment. In advanced cases death usually results from some complicating disease, especially tuberculosis. Starr suggests that among the cases in insane asylums regarded as chronic dementia there may be found some of curable myxœdema.

TREATMENT.—The hygienic treatment of myxœdema consists chiefly in protecting the patient from cold. The drug treatment is practically ineffective, though arsenic, iron, and strychnine have been largely used, On the other hand, the most beneficial results follow the use of the thyroid gland, which probably acts by yielding to the system some principle necessary for the general nutrition. It is evident that the remedy cannot produce a permanent cure: hence the treatment by it is naturally divided into a first period, in which the gland or its preparation is given in as large doses as can be borne until apparent cure is obtained, and a second period, in which the administration of small doses is continued through months or years in order to maintain the normal metabolism. The gland was formerly used finely minced, raw or very slightly broiled,—from a quarter to half of a gland taken from the sheep being daily administered. Experience, however, has shown that a glycerin extract, or the dried and powdered gland administered in tablets or capsules, is thoroughly efficient. It is better to begin with a very small dose (one grain of the dried gland three times a day) and steadily increase it until fifteen to twenty grains a day are taken or symptoms of the so-called thyroidism are produced: these are nervous disturbance, shortness of breath, great restlessness, delirium, rapid pulse, and excessive irritation of the skin. The evidences of amelioration are rapid loss of weight and recovery of the natural perspiration, increase of urine, elevation of the bodily temperature and of the pulse-rate, and lessening of the mental torpor. In a few cases symptoms like those of Graves's disease have followed the over-administration of the extract. The symptoms of thyroidism almost invariably subside with the cessation of the administration of the gland.

Cretinism, which may be looked upon as a form of congenital myxœdema, in its early stages is also amenable to the thyroid therapy.

TUMORS OF THE THYROID.

Other tumors of the thyroid than those occurring as goitre are rare, although sarcoma is sometimes to be found. The possibility that the adenoma of the thyroid is sometimes malignant, essentially a cancer, is suggested by the occurrence in various parts of the body of tumors with a structure resembling that of the thyroid gland. Such a generalization may be regarded as an exaggeration of what is more frequently observed, —namely, misplaced accessory thyroids, which may be found in the neck, in the anterior mediastinum, or at the base of the tongue, as reported by J. Collins Warren. Such accessory thyroids may form tumors of considerable size presenting the structural and degenerative peculiarities of goitre.

The thyroid, whether goitrous or not, may be the seat of an abscess, and may contain tubercles or gummata.

DISEASES OF THE THYMUS GLAND.

Considerable pathological importance in former times was attached to diseases of the thymus gland, especially in infants, in whom it varies considerably in size and reaches its maximum development at the age of two years. It remains relatively unaltered from that time until the age of puberty, when it begins to shrink, and in the adult traces of it are found with difficulty. Spasm of the glottis in infants has been supposed to be the result of an enlargement of the thymus, and was formerly regarded as a *thymic asthma*, a view which is now generally rejected, since this symptom is usually absent when the thymus is enlarged. Jacobi, however, although maintaining that most cases of spasm of the glottis are due to central nervous changes, feels justified in assuming that fatal cases of laryngismus stridulus or spasm of the glottis may be owing to enlargement of the thymus gland.

Minute hemorrhages are not infrequent in infants dying of suffocative diseases and in those suffering from a hemorrhagic diathesis.

Abscesses are rare, if the cases of probable softened tubercles and gummata reported as abscesses are excluded.

TUMORS OF THE THYMUS GLAND.

Most important of the alterations affecting the thymus gland are tumors, which form the large majority of tumors of the anterior mediastinum. To such tumors the term sarcoma or lympho-sarcoma is often applied, although the term to be preferred is lymphoma. The thymus alone may be the seat of the lymphoma, or similar tumors may be found in remote parts of the body as well as within or near the thymus gland. In such cases of multiple lymphomata the condition is one of leukæmia or pseudo-leukæmia, the thymus being affected as part of the process. Lymphoma of the thymus in contrast to lymphoma of the mediastinal lymph-glands forms a homogeneous instead of a conglomerate tumor. The symptoms resulting from tumors of the thymus are those common to other mediastinal tumors, and will be mentioned in the section on Tumors of the Mediastinum.

DISEASES OF THE ADRENAL GLANDS.

Although anatomical changes in the adrenal glands are not infrequently found at post-mortem examinations, they are usually insufficient to be associated with any characteristic symptoms. Amyloid degeneration and variations in the quantity of fat and pigment are encountered. Extensive hemorrhage may be found without its presence being suspected, and bland or malignant tumors occur. The significance of the transplantation of accessory adrenals in the capsule of the kidney will be mentioned in the consideration of tumors of that organ. Of greatest clinical importance are the cheesy alterations forming the most frequent lesions found in Addison's disease.

ADDISON'S DISEASE. SUPRARENAL MELASMA.

Definition.—An affection characterized by disturbances of digestion, extreme debility, and pigmentation of the skin, and usually associated with chronic tuberculosis of the adrenal glands.

Etiology.—Males are three times as often affected as females. The disease generally occurs in adult life, and has been found rarely in children. It prevails among the poorer classes, and may be secondary to tuberculosis elsewhere.

Morbid Anatomy.—Although some alteration of the adrenal glands is usually found, they may be free from any appreciable changes. The changes generally observed affect each gland, and consist in enlargement and induration due to a fibrous and cheesy transformation, the result of tuberculosis. The centre of the diseased portion consists of a cheesy material, either dry and hard or moist and soft, perhaps infiltrated with lime salts, while the periphery forms a fibrous capsule in which and in the vicinity of which miliary tubercles may be seen. Tubercle-bacilli have repeatedly been found in the diseased capsule. The symptoms of Addison's disease have also been present when signs of tuberculosis were absent. In such cases malignant disease or hemorrhage, amyloid degeneration, sclerosis, or gummata of the suprarenal capsules have existed. In rare instances no disease of the adrenal gland is present.

From the frequency with which degenerative and inflammatory changes of the solar plexus have been associated with disease of the capsules, it has been thought that disease of the sympathetic nervous system is the cause of the symptoms and may explain the occurrence of cases in which the adrenal glands were unaffected. In opposition to this view, however, is the fact that in Addison's disease the adrenal glands are more often diseased than is the solar plexus, and the latter structure is only occasionally abnormal in Addison's disease, and may show alterations like those which have been found in Addison's disease without there having been any symptoms of that affection. The alterations to be found elsewhere, with the exception of pigmentation of the skin, are largely attributable to the anæmia or intestinal disturbance symptomatic or characteristic of the disease, the former including the splenic swelling and red marrow, the latter the hyperplastic follicles of the intestine and the enlarged mesenteric glands. Although the anatomical appearances indicate an intimate relation between Addison's disease and alterations of the adrenals, experimental investigation has thrown no light upon this view. The same obscurity has followed experiments upon the abdominal sympathetic with the thought of determining the etiological importance of lesions of the semilunar plexus. At present it is recognized that in Addison's disease the suprarenal capsules are far more often diseased than the semilunar ganglion, but in rare cases the symptoms of this disease may occur without anatomical changes in either of these structures.

Symptoms.—Although pigmentation of the skin is the essential symptom in diagnosis, its presence is usually preceded by gradually increasing digestive disturbances and debility. The former are characterized by loss of appetite, nausea, vomiting, a sense of weight in the epigastrium, and frequent and obstinate diarrhœa. Pain, at times severe, may be complained of, and is usually referred either to the epigastrium or to the lumbar region, and the latter may be sensitive to deep palpation. The debility is out of all proportion to the digestive disturbances. The patient may be unable to walk, and becomes confined to the bed. Headache, vertigo, and attacks of fainting may occur, while still later convulsions, delirium, and coma shortly precede death.

The pulse is quickened and feeble. Respiration is not especially affected, and fever is absent. Examination of the blood has shown no material changes of any constancy. The urine also presents no characteristic changes, although indican has been found increased in a certain number of cases, and albuminuria may be present towards the end of life.

Pigmentation of the skin may be an early or a late symptom, although it is usually the first to call attention to the nature of the disease. The color varies between a pale lemon-yellow and a dark brown suggesting bronze. It is most abundant in those parts which are exposed to the light, as the face and the back of the hands, and is later conspicuous in the axillæ, the bend of the elbow, and the groins. Circumscribed patches of pigment of various size may also be found both in the skin and in the mucous membrane of the mouth, especially of the cheeks. The conjunctivæ and sclerotics are usually free from pigmentation, and are hence strongly contrasted with the pigmented skin. The pigment is deposited in the deeper layers of the rete Malpighii, and apparently represents an excess of that normally found in this region.

DIAGNOSIS.—The diagnosis essentially depends upon the association of pigmentation of the skin with extreme debility. It is therefore important to exclude other causes of pigmentation of the skin, as jaundice, exposure to the sun, racial characteristics, and cutaneous disease. Especial importance in differential diagnosis is to be attached to the peculiar distribution of the pigment, particularly to its presence in the mucous membrane of the mouth. It is difficult, perhaps impossible, to diagnosticate cases of suprarenal disease in which the debility is present, but the discoloration of the skin is either slight or doubtful.

PROGNOSIS.—Addison's disease usually terminates fatally in the course of two years, although temporary periods of improvement may occur. It may prove fatal in the course of a few weeks, and, more rarely, may persist throughout a period of ten years. In the past the reported cases of recovery have been so exceptional as to suggest an error of diagnosis; how far the new treatment will avail the future will show.

TREATMENT.—There is no specific drug treatment for Addison's disease. Rest in bed, iron, strychnine, and various tonics, the meeting of symptoms as they arise, careful feeding, with an easily digested nutritious diet, and the occasional use of the milk-cure, constitute the ordinary means for protracting a hopeless battle. We have seen in apparently typical, very advanced Addison's disease recovery follow the hypodermic use (ten to fifteen minims a day) of the glycerin extract of the suprarenal capsules of beef, fifteen minims representing ten grains of the capsules.

Other cases have been reported of similar benefit from the administration by the mouth of the raw or dried gland. Failures have also been recorded, but at present writing the evidence is sufficient to demand thorough trial of the method in every case. Failure is to be expected in some cases, on account of the malignant nature of the local disease; thus, adrenal tuberculosis might become the starting-point of a wider infection. The dose and the best method of exhibiting are not yet known: two uncooked adrenals of the sheep may be eaten in a day, or probably about half a gland from a steer, or five grains of the dried gland may be administered three times a day in capsules. In any case the amount taken should be increased until a marked amelioration is obtained, some disagreeable symptoms are produced, or established failure has been reached. The glands should always be procured by a veterinarian, to prevent mistake.

CHAPTER II.

LOCOMOTOR AND CONSTITUTIONAL DISEASES.

MYOSITIS.

Definition.—Inflammation of the muscles.

Rheumatic myositis is discussed under the head of muscular rheumatism (page 74).

A *suppurative myositis* is not rare in pyæmia, and occasionally follows influenza, typhoid fever, and other infectious diseases. It is to be treated by local blood-letting, continuous application of cold, and other antiphlogistic methods.

Under the name of *myositis ossificans progressiva* has been described a rare disease, in which either in localized spots or in wide-spread areas the muscle-tissues undergo ossification. This disease, which occurs chiefly in men, commences with swelling, tenderness, and other indications of inflammation. After these have subsided the muscle remains hard and resistant, and gradually undergoes conversion into a bone-like tissue. The calcification seems to be especially abundant in the neighborhood of the attachment of the muscles; but the whole muscle may be converted into a bony plate. The heart muscle may be affected. Concerning the etiology of myositis ossificans we have no knowledge; the course of the disease is extremely slow, and is not modified by treatment.

ACUTE POLYMYOSITIS. PRIMARY MYOSITIS.

Definition.—An acute inflammation which attacks most or even all of the muscles of the body.

Etiology.—The cause of polymyositis is unknown; it may be some specific germ or poison.

Morbid Anatomy.—In polymyositis there seems to be a true inflammation of the muscles, involving both the muscular fibres and the interstitial connective tissue, with congestion, exudation of leukocytes about the vessels, softening, and finally destruction. Hepp reports in his case that the intramuscular fibrous tissue was scarcely involved, and that the muscle-fibres were in a condition of hyaline degeneration. The peripheral nerves have been found to be perfectly normal. All the voluntary muscles of the body are attacked.

Symptomatology.—Without apparent cause the subject of polymyositis, who is usually a young or middle-aged person, begins to suffer with pains in the arms, legs, and trunk, associated with more or less tenderness on pressure, and soon followed by loss of motor power. Fever, if it come not on at once, soon appears, and a marked œdematous swelling occurs, beginning in the extensor side of the extremities and spreading until it involves the whole body, even to the face. The œdematous swelling may be very great and the parts stiff. Enlargement of the spleen appears to be a universal symptom. In many cases there is a peculiar erythematous rash irregularly scattered over the trunk and extremities, leaving behind it distinct pigmentation.

As the disease progresses, the muscles of deglutition and of respiration become affected, producing great difficulty in swallowing and severe dyspnœa. Bronchitis and lobular pneumonia are soon developed, with a peculiar distressing inability to expectorate. In the recorded cases death has usually occurred within a few weeks, from paralysis of respiration. In some instances, however, life has been prolonged for two or more years, during which time there has been atrophy of some of the affected muscles. Whether or not there are mild cases of polymyositis ending in recovery seems at present somewhat doubtful.

Diagnosis.—Polymyositis is distinguished from multiple neuritis by the pronounced œdema. It may be indistinguishable from trichinosis save by an examination of a piece of the affected muscle or by a knowledge of the etiology of the disorder.

Treatment.—Experience with polymyositis has been so limited that no settled opinion can be arrived at concerning the best method of treatment. It is probable that the direct action of medication will be found very slight, and that the only treatment will be one of stimulation and support, such as is adopted in cases of low fevers.

PRIMARY MYOPATHY.

Definition.—A chronic degenerative disease of the muscles with atrophic loss of fibre, not dependent upon any affection of the nervous system.

Pseudo-muscular hypertrophy and the various atrophic muscular diseases were considered as representing several distinct affections until the researches of Erb showed that anatomically as well as clinically the gradations between the various types are so complete that these should be considered as one disease. It is hardly necessary here to describe all the varieties of the disorder, but some of the more remarkable and consistent forms will be separately discussed in the section on symptomatology under the grouping of the pseudo-hypertrophic form, in which enlargement of the affected muscles predominates, and the atrophic forms, in which atrophy is more apparent than is enlargement.

Etiology.—The causes of primary myopathy are unknown. The disease usually begins during childhood, but may come on in early youth, and in extremely exceptional cases as late as forty years of age. It is probably due to some original vice of constitution, especially as it is not rarely a family affection recurring through several generations.

Morbid Anatomy.—The essential lesions are exclusively in the muscles. They consist of atrophy and hypertrophy of the primitive muscle-fibres, increase of the muscle-nuclei, growth of the sheaths of the muscle-fibres and bundles with deposition of fat in them, and final destruction of the muscle-fibres, with vacuolation and fatty degeneration. Erb believes that the primary change is the hypertrophy of the fibre.

Sacara-Tulbure noted in one case enlargement of the lymphatics, with dilatation of the lymphatic vessels and sclerosis of the arteries all over the body, and a peculiar hyaline degeneration of the walls in many parts. There have also been found various alterations in the nerves and nerve-centres; but these differ so much as to have no significance, and have been altogether wanting in various cases. If there were any doubt as to the correctness of the teaching of Charcot that the disease is an essential myopathy, it has been set aside by Babinski and Onanoff, who have shown that the disease in its development follows the embryological muscular territories.

Pseudo-Hypertrophic Myopathy.

SYMPTOMATOLOGY.—The onset of pseudo-hypertrophic paralysis may be so insidious that the disease may have existed for years before attention is called to the disorders of position and gait. In the fully-formed disease these are characteristic. In standing the feet are wide apart, whilst the belly is thrust far forward and the shoulders far backward, producing an excessive lordosis, which is chiefly due to the weakness of the muscles of the back, and persists in the sitting posture unless support to the back be afforded. In walking the child preserves its balance with difficulty, tripping and falling over the slightest obstacle; the gait is waddling, the pelvis rising and falling much more than is normal. Owing to the weakness of the muscles of the back and of those going from the pelvis to the thigh and from the thigh to the lower leg, the child in rising out of a chair helps himself with the hands upon the thigh or the knee. Getting up from the floor is a laborious task, which is accomplished by first rolling over on the face, then elevating the rump, then getting on the knees, then raising the knees from the ground with the help of the hands and feet, then placing one hand upon the knee commencing the elevation of the back, and then using both hands climbing up the legs, so to speak. (See Fig. 1.) In bad cases it may be impossible for the subject to rise from the horizontal position without assistance from some external support.

Owing to the weakness of the shoulder muscles, the shoulder-blades fail to yield a fixed support to the arm in its movements, and even when quiet have their inner margins thrown out in projections almost like wings. An examination of the stripped child will show in most cases great enlargement of the affected muscles. With this enlargement there are frequently prominence and hardness, but the muscles lack the well-known outlines present in persons with highly developed normal muscles, the limbs being rounded and the outlines flowing and regular, somewhat as is seen in stuffed animals. In many cases muscular atrophy and hypertrophy coexist. The affected limbs are apt to be very cold.

The muscles most prone to suffer are the cucullaris, the greater serratus anticus, the sterno-costal portion of the pectoralis major, the latissimus dorsi, the rhomboid, infra-spinatus, and deltoid, the biceps, brachialis internus, and long supinator, the erector muscles of the back, the glutæi, the quadriceps femoris, the adductors, the calf-muscles, and some of the peroneus group.

FIG. 1.

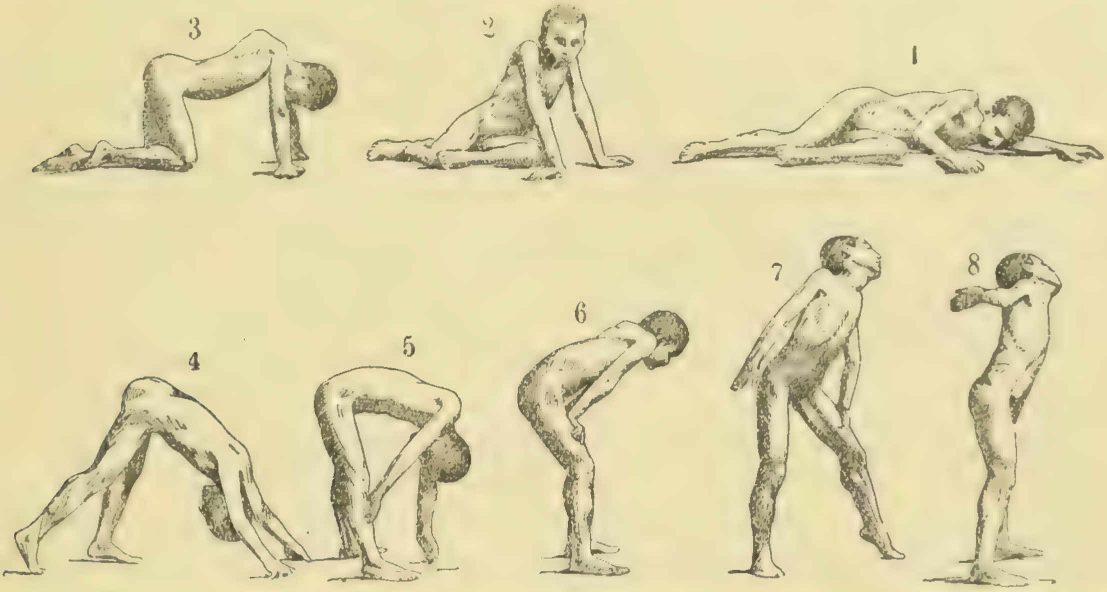


Diagram showing the method of getting up off the floor in pseudo-hypertrophic paralysis, beginning with No. 1.

Fibrillary contractions in the affected muscles may be pronounced, and various French authors state that there is a period of excessive muscular irritability. Nevertheless, as the cases ordinarily come under observation, the patellar and other reflexes are diminished or lost, and the mechanical excitability of the muscles is lowered. The electrical reaction gradually weakens as the muscles disappear in atrophy, but never undergo qualitative changes, the reaction of degeneration never appearing. According to Legros and Onimus, a pronounced susceptibility to loss of electrical excitability under the continuous use of the faradic current is characteristic of the disease.

Late in the disease contractures appear and produce various deformities. Arthropathies and other trophic changes have been noted so infrequently that they must be looked upon as accidents and not as features of the disease. Disturbances of sensibility are rarely pronounced, but more or less complete anæsthesias of sensibility or of special senses may exist.

The thyroid gland is often abnormally developed, and various anomalies of the genital organs have been recorded. Sacara-Tulbure asserts that the skull of the child is usually peculiar, and that the teeth are nearly always striated, denticulated along their free borders, irregular, often imbricated, and frequently excessive in number.

Atrophic Myopathy.

From time to time there have been described various forms of progressive muscular atrophies, all of which essentially belong to primary myopathies, differing from the pseudo-hypertrophic variety only in the failure to deposit inert matter in the muscle whose true structure is undergoing atrophy. As a rule, it is in this class of cases that the evidences of distinct heredity are most pronounced. It is not necessary here to describe more than two types of the atrophic forms of primary myopathy, although in rare cases the localization of the attack varies almost indefinitely.

Type 1.—Scapulo-humeral; juvenile form of Erb.—In this type of myopathy the affection usually commences at about the twentieth year, and involves the muscles of the upper arm and shoulder and of the buttocks and thighs. The deltoid muscle is often spared. The calf-muscles are frequently attacked, but undergo pseudo-hypertrophy, so that they may be firm and hard whilst all the other muscles are wasted. The face-muscles are not affected.

Type 2.—Facio-scapulo-humeral type of Landouzy and Déjerine; the infantile form of Duchenne.—This affection usually commences in infancy, but may be delayed until puberty; it appears first in the face, especially in the orbicular muscle of the mouth, from which it spreads until all the muscles of the face are symmetrically involved. In this way is produced a very peculiar physiognomy, *the myopathic face.* In repose the face is serious, immovable, usually with a somewhat apathetic expression of chagrin. The lips are protuberant, thick, and everted; the eyes cannot be closed, and in some cases the mouth cannot be shut. Under emotional excitement the face is, as it were, in a mask; movements are impossible or exceedingly slow. Whistling or any other act that requires closing of the lips is impossible. After a time the muscles of the shoulder and upper arm are invaded, and in some cases the muscles of the hands finally undergo atrophy. Indeed, the disease may affect the muscles of the lower extremities and the back; and cases have been reported in which the diaphragm, the intercostal muscles, and the abdominal muscles have participated. As there is little tendency in this type to enlargement of the muscles, the case may finally put on the aspect of a wide-spread progressive muscular atrophy.

DIAGNOSIS.—The diagnosis of pseudo-muscular hypertrophy is usually very easy. In certain cases, however, of the atrophic form it may be difficult to distinguish between the affection and progressive muscular atrophy. Typical progressive muscular atrophy comes on late in life, and begins in the muscles of the hands, whilst primary myopathy develops early, and begins in the muscles of the face, the shoulder-girdle, the calves, or the thighs.

TREATMENT.—There is no known treatment that has any effect upon the degeneration of the muscles. Gymnastic training and all forms of muscular exercise do harm rather than good. Massage and electricity have no distinct effect on the muscles.

THOMSEN'S DISEASE. CONGENITAL MYOTONIA.

DEFINITION.—An hereditary affection, characterized by the occurrence of tonic cramps in the muscles on the attempted performance of voluntary acts.

ETIOLOGY.—Nothing is known as to the causes of this affection, except that it is a family disease, appearing in groups of cases in successive generations.

MORBID ANATOMY.—The only knowledge that we have as to the pathology of this disease has been obtained by examination of exsected pieces of muscle. These show great hypertrophy of the primitive bundles, with an increase of the nuclei of the sarcolemma of the interstitial tissue.

SYMPTOMATOLOGY.—This disease may develop in early childhood, but more frequently comes on about the time of puberty. The muscles are usually over-developed in size, but with less than the normal contractile power, often seemingly stiff and awkward in movement. The characteristic symptom of the disease is that when a group of muscles which have been long quiet are brought into action the voluntary movement is arrested by a tonic muscular contraction which lasts from five to thirty seconds and then relaxing permits the motion until the spasm recurs. When the voluntary action is persisted in, the tonic contractions become less and less, and soon disappear, so that the subject can continue movements unembarrassed. The tonic contractions are especially severe when the originating voluntary movement is sudden and energetic ; they are usually manifested in nearly all the muscles of the body, but in some cases are confined to certain groups. The muscles of the eye and of respiration are very rarely affected ; the tongue is often implicated. The contractions are increased by exposure to cold, by excitement, and by the nervousness caused by being watched ; they are diminished by warmth, by mental quiet, and by free alcoholic potations.

The mechanical excitability of the muscles, but not of the nerves, is greatly increased, so that percussion of a nerve may have no effect, but percussion of a muscle will produce a prolonged contraction. The electrical excitability of the nerves is altered. According to the researches of Erb and Huet, the faradic reaction of the muscles may be normal, or there may simply be a tendency for the muscles to be thrown by very slowly interrupted currents into myotonic spasm lasting from ten to twenty seconds ; but even a weak faradic current applied to the nerve will produce tonic contractions, lasting for a long time after the withdrawal of the current. On the other hand, the galvanic excitability of the nerve is somewhat lessened, whilst the galvanic muscular excitability is increased. There is often also a qualitative change in the muscle, the closure and opening contractions becoming equal or even inverted. When a strong galvanic current is passed uninterruptedly along the muscles, rhythmic undulations occur, which commence near the cathode and pass forward towards the anode. For the development of this a strong current must be used, one electrode being placed upon the back of the neck, the other upon the arm or the hand. Epilepsy, chorea, and various nervous symptoms have been present in cases of Thomsen's disease, but they are only accidental complications.

DIAGNOSIS.—The symptoms of Thomsen's disease may be so latent as to appear only after exposure to cold, but are always characteristic when they occur. Eulenberg has described under the name of *paramyotonia congenita* a case in which, after exposure to cold, there was a tonic contraction lasting from a quarter of an hour to several hours and followed by paralytic weakness. Mechanical excitability of the muscles was not increased, and the eye-muscles were affected.

PROGNOSIS AND TREATMENT.—Complete recovery probably never occurs. Sometimes there are remissions, and there seems to be no tendency to a fatal ending. No known treatment is of avail.

RICKETS. RACHITIS.

DEFINITION.—A disease of early childhood, characterized by abnormalities in the growth of the bones.

ETIOLOGY.—A distinction is to be drawn between true rickets and disturbances in the growth of bone which have been regarded as varieties of rickets.*

The designation late or tardy rickets is applied to the occurrence of changes resembling those in rickets in childhood or in adult life. Rickets is found with equal frequency in males and in females, the manifestations beginning during the first two years of life, although usually not earlier than the fourth month. It is doubtful whether rickets is inherited, although the disease may exist in successive generations and in children of the same family. It is possible that a congenital predisposition may exist based upon debility of the parents, whether from disease, as tuberculosis or syphilis, or from faulty hygienic surroundings. The view advanced by Parrot, that rickets is a manifestation of congenital syphilis, is not generally accepted, since this disease occurs without other manifestations of syphilis, and the changes in the epiphyseal cartilage differ from those of syphilis. The disease prevails in the larger cities of Middle Europe. According to Cohn, upwards of sixty-five per cent. of the cases appearing at the Berlin Polyclinic show manifestations of rickets. It is rarely found strongly pronounced in the native white American, but is frequent among negroes living in cities. Local causes are of the utmost importance in the production of rickets. They are to be found in faulty food, especially in artificial feeding, particularly when the food is of excessive quantity or unduly farinaceous. The effects of improper feeding are exaggerated by poor hygienic surroundings, as cold, dampness, and absence of sunlight and fresh air. It has been suggested that malaria may be of importance in the etiology of rickets.

---

* Most important of the latter is congenital or *fœtal rickets*. In this affection, according to Kaufmann, there is an arrest of the growth of the epiphyseal cartilage and a degeneration of the cells. Thus the longitudinal growth of the bone is checked, while the periosteal growth continues. In consequence of the shortening of the base of the skull the root of the nose is depressed and the vault of the cranium heightened. The extremities are short and thick. The deformity thus resulting resembles that occurring in sporadic cretinism or congenital myxœdema. Fœtal rickets, however, is not common where cretinism prevails, and does not especially occur in goitrous families. There is no enlargement of the thyroid as in cretins, or atrophy as in congenital myxœdema. Furthermore, there may be no mental disturbance in fœtal rickets.

MORBID ANATOMY.—The changes in the growth of bone characteristic of rickets proceed from the epiphyseal cartilage and the periosteum, and are regarded by Kassowitz as inflammatory. They consist in a luxuriant growth of the epiphyseal cartilage and an excessive elongation in it of the osteoid trabeculæ, with the formation of marrow-spaces in the cartilage unusually remote from the diaphysis. The ends of the bone become thickened, and long after calcification has taken place islets of cartilage may remain in the ends of the long bones. The periosteum becomes thickened. New-formed blood-vessels arise in its deeper layers, and the osteoid trabeculæ are thin and elongated. An absorption of bone also takes place from the marrow-spaces. In consequence of this excessive growth of osteoid tissue without simultaneous calcification, the bones become thick and soft, are easily bent and broken, the fractures being of the green-stick character, and thus various deformities arise. When recovery takes place the bones are unusually strong, since the lime salts are deposited in those which are abnormally thickened, and the pre-existence of rickets may thus be apparent even in old age. In the cranium the alterations may take place in the occipital and parietal bones, and are manifested by delayed ossification as well as by irregularities in growth into the sutures, and in the closure of the fontanelles. Deformities of the spine, of the thorax and pelvis, and of the bones of the extremities, especially of the lower, take place. Enlargement of the liver and spleen is frequent.

The fontanelles are large, and the anterior fontanelle may remain open until the third year instead of being practically closed early in the second year. Various degrees of hydrocephalus may arise, and the head may become squared in consequence of thickening of the frontal and parietal bones. As a result of the alterations of the intervertebral cartilages, curvatures of the spine occur. The sternum protrudes from weakening of the costal cartilages, and the child becomes pigeon-breasted.

If the sacro-iliac synchondroses are especially diseased, the sacrum and symphysis are approximated and the lateral diameter of the pelvis is widened. If the pubic cartilages are especially diseased, the antero-posterior diameter may be elongated. The thickening of the epiphyseal ends of the bones gives rise to the term double-joint, while the yielding of the weakened bones to the weight of the body is manifested by the deformities known as bow-legs and knock-knee. The enlargements of the epiphyseal ends of the bones of the arm are more frequent at the lower end of the radius and ulna, and are attributable to the use of the hands and arms in crawling. Such deformities may prevail in the cranium, thorax, and spine, or in the extremities, or all these regions of the body may become affected.

SYMPTOMS.—The symptoms of rickets are usually of gradual development as the period of dentition approaches, and are often attributable to teething. They are more likely to begin in the winter months. The infant becomes fretful and restless, and perspires readily, especially from the head and neck. The stools become frequent, green, and slimy, although the appetite remains good. The infant awakes suddenly from a sound sleep with loud cries as if terrified, and convulsions and tetany and various nervous disturbances may occur. (See page 441.) Spasm of the glottis, bronchitis, and broncho-pneumonia are frequent, and irregular elevations of temperature take place. The abdomen becomes distended, although emaciation is usual, and the patient is weak and pale. The significance of these general symptoms is apparent when physical examination discloses the soft spots in the occiput (craniotabes) or parietal bones, or the beaded condition of the costal cartilages, the last forming the rosary of rickets, which is due to the thickening of the cartilaginous ends of the ribs. With the advance of the disease the child objects to being handled, through fear of pain, and refuses to sit up or walk. Dentition is delayed, and the fontanelles remain open. The head becomes large, and deformities of the spine and extremities arise. Such deformities may be greater in the head, thorax, and spine or extremities, or all these regions of the body may be affected. The child remains pale, the tissues flabby, and the intelligence dull. Intercurrent inflammatory affections, especially bronchitis, broncho-pneumonia, and atelectasis, are frequent, particularly when the thorax is affected. The course of rickets is usually slow, but rapid cases with pronounced fever occur.

The duration of the disease varies, and in accordance with the age at which it begins and the time it continues do the localization and extent of the deformities vary. Rickets beginning in the early months of life and continuing for months or years results in deformities of the head, trunk, and extremities. Rickets beginning at the same time and lasting but a few weeks may produce little alteration of the skeleton, or may cause changes localized in the skull, spine, and thorax while the extremities are but slightly affected. On the contrary, rickets coming on after the first year of life produces deformities of the extremities, thorax, and spine, while the cranium shows but little change.

DIAGNOSIS.—It is a matter of the gravest practical importance to recognize the first beginnings of rickets. When, during the second half-year of life, there are obstinately recurring digestive disturbances, soft, white, tender flesh, and delayed, irregular dentition, the nature of the case should be recognized before local sweating of the head, the peculiarities of the fontanelles, and bony deformities give precision to the diagnosis.

The only disease which can produce changes in the bones similar to those of rickets is hereditary syphilis. This disease is to be differentiated by the presence of other syphilitic changes. Although craniotabes is one of the early manifestations of rickets, it may occur independently of this affection. More characteristic is the presence of the rosary, which usually appears before conspicuous alterations in the cartilaginous ends of the long bones. It is to be remembered that one or several of the costal cartilages may be affected. The bruit which can often be heard on auscultation over the anterior fontanelle is not diagnostic, as it is liable to occur in chronic hydrocephalus or whenever from any cause closure of the fontanelles is delayed; indeed, it is sometimes audible in perfectly healthy children.

PROGNOSIS.—Rickets is a disease which may be arrested at any stage by appropriate treatment. The prognosis thus is favorable as to an arrest of the process, although the existing deformities may persist. During the progress of rickets death is frequent from the complications in which the respiratory apparatus is involved: hence the frequency of death in rachitic children from measles, whooping-cough, and bronchitis. The permanent alterations occurring in protracted rickets are frequent sources of the permanent stunting of the growth of the individual, shown by some of the dwarfs to be found in most large communities. In the female the rickety deformity of the pelvis proves a source of serious, if not of fatal, interference with labor, Cæsarean section often being necessary in the effort to save the life of mother and child.

TREATMENT.—In rickets it is essential that the child be put under the most favorable hygienic surroundings possible. The question of feeding is one of the greatest importance. Good healthy breast-milk is the proper food for an infant under one year of age; but, in our experience among the well-to-do, bad milk upon the part of the mother or of the wet-nurse is not a rare cause of a rachitic condition. It is essential that nurses be healthy, be sufficiently but not over-fed, have abundant exercise, and furnish a plentiful supply of milk with the proper physical characteristics. In any case of rickets occurring in a wet-nursed child the question of the milk used should be most carefully investigated, even by chemical examination. Moreover, the amount of milk should be sufficient. According to the researches of Lewis Smith, the child should have in the first year of infancy,—

	At each Feeding.	Number of Daily Feedings.	Total Daily Amount.
First week	1 ounce.	10	10 ounces.
Third week	1½ ounces.	10	15 "
Sixth week	2 "	8	16 "
Third month	3 "	8	24 "
Fourth month	4 "	7	28 "
Sixth month	6 "	6	36 "
Tenth to twelfth month	8 "	5	40 "

Under no circumstances should a pregnant woman be allowed to suckle a child, as either the offspring or the mother must be starved. When breast-milk cannot be procured, the best results are probably obtained by a very careful admixture of cow's milk and Mellin's, Nestlé's, or other artificial infants' foods.

In older children the diet should consist of an abundance of milk, easily digested farinaceous food (not oatmeal, unless the child's digestion is extremely good), and meat once or twice a day. Disturbances of the digestive organs should be carefully treated by the use of pepsin, sodium phosphate, bismuth, or other remedies, *pro re nata*.

The rachitic child should always be warmly clad; should be kept in the sunshine in the open air as much as possible, even on cold days, proper protection being afforded by wraps; should be bathed daily, and after the bath should be freely anointed with cotton-seed, olive, or other bland vegetable oil, or preferably with cod-liver oil, which should be well rubbed in. Great care should be taken to prevent deformities by keeping the child off its feet, or in extreme cases even from sitting up, and by the use of splints or other mechanical contrivances. In older children massage may be useful to replace natural exercise.

When it can be digested, cod-liver oil is a most important remedy: it is usually best given in the form of the emulsion with lactophosphate of lime. Iron seems to be demanded by the existing anæmia, and, unless it disturbs the digestion, is generally serviceable. It should, however, be given in small doses, and its effect upon the alimentary canal most carefully watched. Phosphorus is a very valuable remedy in rickets; one two-hundredth of a grain may be given three times a day after food, in the form of the official elixir, of which one teaspoonful contains one-sixty-fifth of a grain of phosphorus, or dissolved in oil. Owing to the avidity with which phosphorus takes oxygen, such minute quantities of it frequently suffer destruction: hence the practitioner should always see that the preparation has been freshly made and has a distinct odor and taste of the drug. The dose should also be increased as borne.

The numerous nervous, pulmonic, and digestive complications of the rachitic state should be carefully treated by remedies which would be suitable for the same conditions not arising in the general rachitic disturbance of nutrition, but it should always be remembered that the pulmonic, nervous, or other accident of the disease is best cured by curing the basal disorder.

HEMORRHAGIC RICKETS. INFANTILE SCURVY. BARLOW'S DISEASE.

This affection, although classified by some authorities as a variety of rickets, and by others as a variety of scurvy, is perhaps rather a disease *sui generis*, both resembling and differing from scurvy and rickets. The occurrence of its symptoms and lesions without any manifestations of rickets demands its consideration independently of that disease. Its frequent association with rickets, and its occurrence at the same period of life and in connection with a similar diet, suggest an intimacy of relation to rickets. It resembles scurvy in its origin from too exclusive a diet and from the conspicuous hemorrhagic lesions. It differs from scurvy in being found in regions where the latter disease is rare, in its absence from countries, as Russia, where scurvy prevails, and in being a disease limited to infantile life, occurring in solitary cases and not as an epidemic. In scurvy hemorrhages in the skin, gums, and joints are the rule, while in hemorrhagic rickets the bleeding is largely periosteal. It is generally admitted that the first case of the kind was published by Möller in 1857, who regarded it as an instance of acute rickets. Cheadle in 1878 considered the affection as rickets complicated with scurvy. Barlow in 1883 published his important paper on the subject which has led to the general association of his name with this affection.

ETIOLOGY.—Hemorrhagic rickets, like rickets, is a disease of infantile life, and is due to improper food. Particularly injurious has proved the exclusive use of condensed milk, or of various prepared infants' foods, although freshly prepared cow's milk and even breast-milk have given rise to the disease. Unlike rickets, it prevails among children of the well-to-do or of those in moderate circumstances with good hygienic surroundings, and is frequently seen in the country. Its occurrence in congenital syphilis has also been observed. Fürst suggests that the faulty diet produces a cachexia, either rachitic or hemorrhagic or a combination of both, according as there is a lack of calcium phosphate or of potassium phosphate or of both salts.

MORBID ANATOMY.—The characteristic changes are found in connection with the bones, especially of the extremities, the femur being oftenest affected. The cranium and jaw are rarely attacked. The periosteum is thickened, injected, and is separated from the shaft of the bone by more or less extensive hemorrhage. When rickets is associated the epiphyses are swollen and may be separated. Hemorrhages and œdema may occur in the intermuscular tissue. In fatal cases cutaneous hemorrhages may be found.

SYMPTOMS.—The symptoms of this disease are of most frequent occurrence towards the end of the first year of life, although they have been observed in infants of six months. They may develop suddenly or gradually, the child appearing well nourished and even plump, although somewhat pale. The patient is fretful, cries when touched, dreads to be moved, and becomes extremely weak and unable to sit upright. The extremities are semiflexed, and fusiform swelling, especially of the thighs, appears. Similar thickenings, though less extreme, may occur in the upper extremities and upon the scapulæ. The enlargement is resistant, the skin tense. Eventually the swelling becomes elastic, perhaps flattened. In the further progress of the disease the pale skin may present a faint yellow tint. If rachitic changes are associated, the epiphyses, especially of the femur and tibia, when separated yield to the touch. Hemorrhages from the mouth often occur at dentition, and more rarely cutaneous hemorrhages are observed. The temperature is usually slightly elevated while the hemorrhage is taking place, and there is then considerable perspiration.

DIAGNOSIS.—The rapid occurrence of pain on motion of the lower extremities, followed by swelling in the vicinity of the joints, which are themselves not affected, in infants artificially fed, should suggest hemorrhagic rickets. The local symptoms may be confounded with those of articular rheumatism, but the temperature is less elevated, sweating is less considerable, and the appetite of the child is but little affected. The rarity of acute rheumatism in infancy is also opposed to this diagnosis.

PROGNOSIS.—The prognosis is favorable, although fatal cases have occurred. Of one hundred and sixty-six cases referred to by Fürst, sixty-seven per cent. recovered, eighteen per cent. died, and in fifteen per cent. the result was unknown. Recovery is usually rapid with the early recognition of the disease and the use of appropriate diet.

TREATMENT.—Antiscorbutic treatment, with careful regulation of the diet and hygienic management of the child, should first be tried : if this fail the treatment should be that of rickets. Barlow recommends locally the use of wet compresses and avoidance of movement during the acute stage, followed after a time by careful shampooing.

OSTEOMALACIA.

DEFINITION.—A chronic disease of the bones of the adult, resulting in their softening and characterized by a transformation of bone-tissue into marrow.

ETIOLOGY.—Osteomalacia is a rare disease, especially prevalent in certain countries, particularly in Germany, Switzerland, and Italy, and especially in certain localities such as the borders of the Rhine and mountain valleys ; hence it has been regarded as endemic. It is found nearly ten times as often in women as in men, and usually in those from twenty to thirty years of age. Faulty hygienic surroundings are regarded as predisposing causes, but pregnancy is generally agreed to be of especial importance. The disease is more likely to occur in those in whom pregnancies take place at short intervals, and twin pregnancies often occur in women with osteomalacia. Fehling maintains that ovarian disease is a cause of osteomalacia, and that the favorable results of castration demonstrate the truth of the theory. A distinction is drawn between puerperal osteomalacia and senile osteomalacia. The former variety includes nearly three-fourths of the cases. Senile osteomalacia affects rather the bones of the thorax and pelvis, while in puerperal osteomalacia the entire skeleton may be attacked. The term infantile osteomalacia has been applied to certain cases of rickets.

MORBID ANATOMY.—The changes resemble those found in rickets, but are not present in the vicinity of the epiphyseal cartilage. The trabeculæ of the bones are largely composed of osteoid tissue, owing perhaps to decalcification, perhaps to new formation of osteoid tissue. The marrow is hyperplastic and excessively vascular. With the advance of the process the affected bone becomes so softened that it may be cut, and cavities of considerable size arise within the shaft by a cystoid metamorphosis,—their walls being composed of a thin layer of osteoid tissue and periosteum, while the contents are a gelatinous, transformed marrow. The weakened bones get bent and broken, and Orth pictures the healing of such fractures with osteoid tissue in which are no lime salts. The spine becomes curved, the ribs flattened and depressed, the antero-posterior diameter of the pelvis elongated, the transverse diameter shortened, while the bones of the extremities, especially the lower, are shortened, distorted, and brittle. The bones of the cranium and face are usually unaffected. Atrophy and cystic degeneration of the ovaries have frequently been observed.

SYMPTOMS.—Pain is the first symptom complained of, and in the osteomalacia of pregnancy usually makes its appearance during the later months of gestation. It is then dull, persistent, increased by prolonged muscular action, and is referred to the pelvis and sacrum. In the non-puerperal cases the pain is referred to the spine, chest, or legs. The pain may be localized in certain bones, and is perhaps aggravated on pressure. In puerperal osteomalacia relief to the pain usually takes place between successive childbirths, and aggravation occurs during pregnancy, sometimes at menstruation. With the persistence of the disease the pain eventually disappears, but the walk of the patient is affected. The gait is stiff, somewhat spastic, and the patient easily gets tired. The stature of the patient becomes lowered from curvature and shortening of the spine. The functions of the body other than those of respiration and parturition are but little affected. Deformity of the thorax may cause dyspnœa and palpitation, while deformity of the pelvis prevents the passage of the fœtus. Fibrillary muscular twitchings occur. The patients lose flesh. The skin is pale and the hæmoglobin is diminished. Various examinations of the blood have been made, but no characteristic appearances have been found. Albumose has frequently been detected in the urine. The disease is ordinarily of long duration, exacerbations and remissions occurring, and puerperal osteomalacia often ceases at the climacteric.

Diagnosis.—In the early stages the diagnosis may be difficult, since the pain in the back or pelvis may be regarded as lumbago or neuralgia. The suggestion of osteomalacia as a cause is based upon the association of the pain with pregnancy and upon the nationality and previous surroundings of the patient. In the later stages the deformity of the bones is sufficiently characteristic.

Prognosis.—Until recently this disease was regarded as almost inevitably fatal, although periods of temporary improvement might occur and the patient might live for years. It is now recognized that under appropriate treatment a cure may take place, provided the disease is early recognized. In cases of recovery the bones may become abnormally dense from an excessive deposition of lime salts.

Treatment.—The treatment of osteomalacia consists in putting the patient under the best possible hygienic surroundings; in the especial giving of foods, such as whole wheat flour, which contain the natural phosphates; and in the administration of tonics, of iron or cod-liver oil, of the calcium phosphates, and of phosphorus itself. Warm baths sometimes afford much relief. Numerous cases of arrest and cure of the disease have followed ovariotomy and Porro's operation. Married women suffering from the disease should always be warned against the danger of pregnancy.

OBESITY.

A tendency to excessive corpulence occurs in certain races of men, as the Jews, and is also a characteristic of certain families. Probably in the majority of cases, however, it is due to one or more of three causes: first, over-eating; second, under-exercising; third, the excessive use of alcohol. In judging of the individual case it should always be remembered not only that corpulence may exist without being excessive and without requiring rigid treatment, but that what is excessive in one individual may not be so in another; and even in the same individual the amount of allowable corpulence varies at different ages. In the young any superfluity of fat, unless in a person of pronouncedly corpulent inheritance, is usually the result of over-eating and physical indolence and must be looked upon as pathological. In the middle-aged a moderate amount of fat is physiological, and to be rather encouraged than discouraged. Again, women normally carry more adipose tissue than do men. Whenever the weight becomes so burdensome as to interfere with walking, or to cause puffiness or shortness of breath in going up-stairs or ascending heights, treatment should be instituted. The symptoms of cardiac disturbance and shortness of breath due simply to excessive fat in the system and about the heart may be so severe as to simulate organic disease; and there can be little doubt that in many of these cases there is an actual deposit of fat between the muscular fibres in the heart-wall which interferes with the action of the viscus.

Excessive corpulence is to be combated by exercise and regulation of the diet, and sometimes by medication. In the use of exercise it is essential to remember that the corpulent person is usually feeble and often has a softened heart and arteries. The amount of exercise, therefore, must be graded to the individual, whilst the form of it is almost always dominated by surrounding circumstances. The essentials are, first, that the exercise should involve most or all of the muscular system; second, that it should be sufficiently severe, if not at first, as soon as possible, to produce free perspiration; third, that it should never be enough to produce an exhaustion which will show itself in sleeplessness or in markedly increased debility the day following; fourth, that it should be steadily persisted in day after day, and gradually increased as fast as the patient's strength will allow. In all severe cases, when practicable, the exercise should be followed by a bath, a thorough rubbing down, and rest in bed. Outdoor exercise is preferable to in-door; exercise that gives variety and pleasure to the patient is much superior to exercise which is irksome. A careful, cautious trainer, if prevented from overdoing by rigid medical supervision, will often aid greatly.

In regulating the diet in a case of obesity it should be remembered that the fat contained in the food may be directly deposited in the body, but that albuminoid foods in their decomposition and destruction in the body certainly yield fat, although it is practically demonstrated that they do not increase corpulence as much as do the carbo-hydrates. It is evident, therefore, that those systems of dieting which consist simply in the withdrawal of fats and carbo-hydrates and their substitution by albuminous nitrogenous foods are not completely satisfactory, and that usually it is essential for the physician to regulate the quantity as well as the quality of the food.

The severity of the diet should depend upon the severity of the condition to be combated. In mild corpulence withdrawal of all alcohol and avoidance of excess of eating, with increased exercise, may suffice.

Bantingism, or the method of treatment of corpulence which consists especially in putting the patient upon an almost entirely meat diet, as advocated by Harvey, encounters in practice several difficulties. There is always the danger of an excessive strain upon the kidneys in the elimination of nitrogenous educts through an over-abundant supply of meat. This, however, is usually to be averted by rather decreasing the hydrocarbons in the food than increasing the nitrogenous elements. The physician should examine the urine of the patient once every ten days, so that the diet may be altered if albumin be found. In order not to be misled, the practitioner should bear in mind the fact that violent exercise may of itself produce albuminuria.

More valid objections to Bantingism are found in the chilliness, the weakness, and the ever-increasing repugnance to meats produced by a too rigid restriction to albuminous foods. Moreover, in some cases severe dyspeptic and especially gouty arthritic symptoms are produced by a rigid flesh diet. To meet these objections Professor Ebstein modified the plan of Dr. Harvey by adding fatty food, and allowed the following dietary:

Breakfast.—Tea without sugar or milk; one and a half ounces of white bread, with plenty of butter.

Lunch.—Fatty soup made from a marrow-bone; four to seven ounces of flesh containing much fat; some vegetables; stewed fruit without sugar; two or three glasses of wine. Later in the afternoon, one cup of tea without milk or sugar.

Evening.—One cup of tea without milk or sugar; one ounce each of bread and butter; one egg, or a piece of fat ham, or fat roast meat, or cheese; fresh fruit. No alcohol.

The following dietary is a modification of that of Harvey which we think may ordinarily be followed:

Breakfast.—Grape-fruit or orange; four ounces of lean meat or five ounces of fish; four ounces of bread; tea or coffee.

Lunch.—Two ounces of lean meat, oysters, or fish; three ounces of brown bread; raw fruit *pro re nata*, except grapes and bananas.

Dinner.—Half a pint of soup; two ounces of fish; three ounces of lean meat; two ounces of gluten bread; six ounces of green vegetables.

In some cases of corpulence it is not necessary to enforce the diet as strictly as that given, only to cut off potatoes and sugar and reduce the amount of white bread. In all cases the weight should be taken from week to week, and care be exercised that the loss is not too rapid. When the repugnance for meat becomes pronounced it is very often well to intermit the rigidity of the diet. The question of the amount of water to be used is a very difficult one to answer. So far as our present physiological knowledge reaches, there appears to be no scientific reason for reducing the ingestion of liquid, but from time immemorial trainers have insisted upon abstinence from drinking. There is no reason why alcohol should be allowed; but, if the patient insist on it in some form, thin sour wines are the best.

A mistake which we have very frequently seen made is the overdoing of the thinning process: there are many persons who are not in good health unless they carry with them more fat than is needed for æsthetic purposes or than would seem to be useful for comfortable living.

In making a modified diet list for a case of obesity, lean meat, including game, chicken, fish, lobsters, and oysters, young green vegetables, such as peas, beans, spinach, cabbage, cauliflower, asparagus, salads, celery, and tomatoes, gluten bread, and almonds, may be freely allowed. Articles which should be used in very small quantities or altogether forbidden are butter, cream, fats, sauces, tame geese and ducks, eels, salmon, pastries and all kinds of confectionery, and potatoes. The foods to be taken in small quantities are bread, buckwheat cakes, milk, and eggs.

It is doubtful whether any known drugs have the power of reducing the amount of fat in the body, except by purging, interfering with digestion, or producing in some way a diseased condition. There are, however, many corpulent persons who are habitually anæmic. Under such circumstances the use of iron in moderate quantities may be of service. As a restricted diet has a great tendency to produce constipation, it is essential that laxatives be employed when needed. In Germany certain springs, especially Marienbad and Carlsbad, have a great reputation in the treatment of obesity. We believe that the results obtained are largely due to the diet and exercise enforced, but it is probable that the free purging which these waters produce brings about a normal condition of the digestive apparatus, prevents the absorption of food by hurrying it out of the intestines, and probably aids in the reduction of corpulence. It is also possible that the excess of alkali may aid in removing the fat. The spring treatment may be imitated with success by giving a mixture of salines and alkalies, or of salines with iron in anæmic subjects. (See formulæ 2 and 3.)

Thyroid extract is stated to have done good in certain cases of excessive obesity ; and when the fat-making tendency of the body amounts to a disease, so that the condition cannot be overcome by the use of the normal methods of restricting fat-production in the body, the extract should be tried as in myxœdema. (See Myxœdema.)

ACUTE ARTICULAR RHEUMATISM. RHEUMATIC POLYARTHRITIS. RHEUMATIC FEVER.

Definition.—An acute febrile disease, believed by many authorities to be of infectious origin, characterized by inflammation of various joints in succession, profuse sweating, and a tendency to endocardial inflammation.

Etiology.—Acute articular rheumatism is a disease widely distributed throughout the world, especially in regions where there is considerable moisture. It occurs most frequently in the colder months, least often during midsummer. Both sexes are alike affected, especially during early adult life, and it is rare in infancy and old age. It prevails in certain families, and some authorities are of the opinion that a gouty inheritance strongly predisposes the young to attacks of acute rheumatism, while later in life muscular rheumatism, and still later unmistakable gout, become manifest. According to others, gout and acute rheumatism rarely occur in the same person, and members of gouty families are not especially prone to acute rheumatism, nor are families showing a strong hereditary predisposition to rheumatism particularly liable to the manifestations of gout. The endocarditis of rheumatic fever is acute and usually associated with the presence of bacteria, while that of gout is chronic, without bacteria and with degenerative aortic changes. Rheumatic fever is especially found among persons whose occupation exposes them to sudden and extreme changes of temperature, when profuse perspiration is quickly checked by cold draughts of air. Local conditions are important, since numerous cases occur in limited localities, and Dalton has observed an apparent etiological importance in leaky drains. A certain resemblance of the symptoms and lesions to those found in gout has suggested that obscure modifications in the metamorphosis of tissue may result in a toxæmia, oftenest attributed to an excess of uric or of lactic acid in the fluids of the body, but thus far no such excess has been found. Richardson affirms that he has produced rheumatism by the administration of lactic acid. This theory and his observations lack efficient confirmation.

The theory of the infectious origin of rheumatism is the most popular at the present time. It is based upon the resemblance and similarity of distribution of many of the lesions to those found in septicæmia and pyæmia, the frequency of relapses, and the occasional occurrence of arthritis in such infectious diseases as scarlet fever and dysentery; additional support is derived from the occurrence of apparent epidemics at certain seasons in limited localities, especially in households, and the discovery of bacteria in the fluids from the joints and from the inflamed endocardium and pericardium. Sahli has recently found throughout the body a coccus morphologically identical with the *staphylococcus citreus*, and Singer finds a variety of bacteria in the urine from cases of acute rheumatism.

According to this view, the joints, like the spleen, lymph-glands, and bone-marrow, represent a structure especially susceptible to the action of bacteria or their toxins. A suppurative inflammation of the joints readily occurs in pyæmia and puerperal fever, and the introduction of pyogenic cocci into an injured joint is followed by suppuration, and in the inflamed joints in gonorrhœa pure cultures of the gonococcus have been found. It is thus plausible that a rheumatic arthritis may represent a mild variety of pyæmia or septicæmia, the milder character of the lesions perhaps resulting from the action of an enfeebled bacterium or attenuated toxins. The observation made by Sahli suggests that the various local lesions of acute rheumatism may result from a multiple localization of bacteria. No specific bacterium has been found, but it is possible that various bacteria may be concerned, and that other factors may be necessary.

MORBID ANATOMY.—The anatomical changes consist essentially in a sero-fibrinous inflammation of the joint and the neighboring tissues. The synovial membrane is swollen, injected, hemorrhagic, and the synovial fluid is increased in quantity, opaque yellow, flocculent, and contains red and white blood-corpuscles. The exudation is sometimes sufficiently opaque from the presence of leukocytes to be regarded as purulent, although true pus rarely exists. An opaque-yellow infiltration of the tissues near the joint, including the bursæ, tendon-sheaths, and connective tissue, is frequent, and hemorrhage may occur. Abscesses in these tissues are rare, and disease of the bone and cartilage is infrequent, if the occasional termination in chronic articular rheumatism is excluded.

SYMPTOMS.—According to the nature and severity of the symptoms a distinction is made between acute, subacute, and chronic articular rheumatism, although the last, in most cases at least, is probably a different disease in etiology, symptoms, and results, and will therefore receive independent consideration. Acute articular rheumatism is of rapid onset, sometimes appearing within a day or two after the sudden exposure of a heated person to cold. The patient complains of chilly sensations, which are soon followed by a fever with morning remissions and evening exacerbations, the extreme elevation rarely being above 104° F. Tonsillitis is at times present, and may precede or accompany the arthritis. The joints now become red, swollen, and painful. Symmetrical joints are usually affected, and recurrences are frequent. At first the articulations of the lower extremities are usually inflamed, then those of the upper, more rarely the hip, jaw, vertebræ, and pelvic symphyses. The swelling of the joint is largely due to the exudation into the synovial cavity, which may be sufficient to cause fluctuation, but it is partly dependent upon œdema of the surrounding tissues. The pain is often severe, and may become intense on motion of the joint; it is usually worse at the outset of the inflammation and diminishes as the exudation increases. It may be limited to the joint or may extend along the course of the neighboring tendons or nerves. One or many joints may be inflamed, the inflammatory process tending to move from joint to joint without following any definite order, diminishing in the one as it increases in the other. In the milder cases the affected joint is freed from the inflammatory disturbances in the course of a few days. In the severer cases the arthritis may persist for several days. Profuse sweating accompanies the inflammation of the joints, increasing as new joints are attacked, and the perspiration has a sour odor and an acid reaction, but does not contain lactic acid. Headache, loss of appetite, and nausea accompany the fever. The pulse is quickened, its tension diminished. The respiration is somewhat accelerated. The range of temperature at first shows but little daily variation, but exacerbations take place as new joints are attacked, and remissions of temperature occur as the inflammation subsides. A continued high elevation of temperature remains for some time after the swelling of the joints subsides. The urine is scanty, high-colored, its specific gravity 1025 to 1030, and there are abundant uric acid and urates. Uric acid may be absolutely increased or diminished; urea is often diminished. A trace of albumin may be present. As the fever subsides, the urine becomes abundant and pale, and peptone is said to be present as the inflammation of the joints lessens.

Austin Flint has shown that the duration of acute articular rheumatism may be but little affected by treatment. In uncomplicated cases the disease lasts from one to six weeks, and in cases of moderate severity at least four weeks of illness may be expected. In severe cases, especially those in which complications exist, despite all treatment a period of several months may elapse before the patient is convalescent. The term *subacute* rheumatism is applied to cases of rheumatic fever of a mild type. Fewer joints are affected, pain, redness, and swelling are less extreme, the temperature is lower, and the sweating less, but the course is often protracted. Complications may occur, and it is to be remembered, especially in the febrile rheumatism of children, that the complications may be more serious than the arthritis, although the range of temperature may follow a subacute course.

Among the most frequent and important of the complications of rheumatic fever is endocarditis, which takes place in nearly one-fourth of the cases, whether mild or severe, especially in the young. In a certain number of cases pericarditis is present, with or without endocarditis, and inflammation of the myocardium may also occur. These cardiac complications may intervene at any time during the course of the disease, but are more likely to be made manifest during the second week. Localization of the rheumatic inflammation in the heart may be indicated by pain, palpitation, an irregular pulse, rapid breathing, a sense of oppression referred to the heart, and a rise of the temperature, although these symptoms are no necessary evidence of affection of the heart. The presence of a pericardial rub or an endocardial murmur may be the only symptom, and time is often necessary to determine the nature of the murmur in virtue of the relative frequency of hæmic murmurs in acute rheumatism. The mitral valve is oftenest diseased, either alone or with simultaneous affection of the aortic valve. Rheumatic inflammation limited to the latter is rare. The pericardial exudation is both serous and fibrinous, and its extent is to be determined by the physical examination of the heart. (See Pericarditis, Section III.) The exudation may be rapidly absorbed or may be so excessive as to be the immediate cause of death. Delayed absorption of the exudation is likely to result in permanent adhesions.

Pleurisy, more rarely pneumonia, may occur during the progress of acute rheumatism. The former prevails on the left side, and is frequently associated with pericarditis. It is probable that the occurrence of pneumonia is the result of an independent infection. The occasional occurrence of tonsillitis at the outset of acute articular rheumatism is considered by some as evidence of the infectious origin of the latter. Iritis, nephritis, and cystitis are rare complications.

Cerebral complications are sometimes so conspicuous as to give rise to the term *cerebral rheumatism.* Restlessness, delirium, convulsions, and coma may rapidly develop with subsidence of the joint-inflammation, usually during the second week, and be associated with hyperpyrexia and rapid pulse, the temperature even exceeding 110° F. The hyperpyrexia may prove the cause of death and no abnormal appearances be found in the brain. Various mental disturbances may occur during the course of rheumatic fever, in mild or severe cases, at the outset or during convalescence. The patient may be delirious, even maniacal, with suicidal intentions, or suffer from melancholia. Hemiplegia from cerebral embolism is a rare complication. Chorea, especially in children, sometimes occurs after the subsidence of the acute symptoms. Attacks of chorea may alternate with attacks of rheumatism, and many cases of chorea occur in persons who have suffered from rheumatism. A rheumatic neuritis of nerves in the vicinity of the inflamed joint may develop, and pain, numbness, or prickling follow. The muscular atrophy which sometimes follows a rheumatic inflammation of the joint is occasionally attributable to the associated neuritis.

A variety of cutaneous eruptions may arise. Most frequent are sudamina during the stage of excessive sweating, but urticaria, erythema, pemphigus, and purpuric spots have been seen. The purpuric spots in rheumatic fever should not be confounded with the rheumatic symptoms in purpura, the further consideration of which is to be found in the article on Purpura. That the disease is rather rheumatic than purpuric is to be inferred from the extent and severity of the articular inflammation and the relief derived from antirheumatic treatment. Attention has been called, particularly of late years and most recently by Futcher, to the presence of subcutaneous nodules, first especially described by Jaccoud as occurring in infants and young children, and regarded by some as pathognomonic of this disease. They appear during the progress, or more frequently towards the end, of the rheumatic attack as sharply defined nodules of the size of peas. They are few or many, and are situated upon the tendons and ligaments, especially near the elbow and knee, and upon the pericranium and periosteum. They may rapidly develop, disappearing in the course of a few weeks, or remaining for months. They are to be found in mild or severe cases, and may appear in the absence of inflammatory symptoms.

DIAGNOSIS.—Acute articular rheumatism is usually readily recognized from the grouping of the symptoms. Difficulty is sometimes experienced, especially among children, in discriminating between acute osteomyelitis and acute articular rheumatism, particularly when the former is multiple or in the vicinity of the large joints. The intensity of the pain, the extreme sensitiveness of the bone, and the typhoidal symptoms are of especial importance in the diagnosis of osteomyelitis. Secondary inflammations of the joints occurring in the various infectious diseases are preceded by the characteristic symptoms of these diseases. In gouty arthritis the small joints, especially the great toe joint, are usually affected; pain and redness are more considerable; sweating is absent; the fever is slight.

PROGNOSIS.—Acute articular rheumatism generally terminates favorably, although recurrences even after the lapse of years are frequent, and chronic articular rheumatism may result. Recurrent attacks of acute rheumatism may be as severe as the original attack. The mortality is estimated at three per cent., the fatal cases being usually due to the complications enumerated.

TREATMENT.—In acute rheumatism the patient should be dressed in a flannel night-dress (which should be so made that it can be frequently changed without exposure to the patient), and should sleep between blankets and be carefully protected from draughts of air. In most cases the diet should be at first restricted to milk, or to barley, oatmeal, or other gruels, the food being given in moderate quantities at short intervals. If milk cannot be borne, broths, raw eggs, and various farinaceous foods may be substituted or given in connection with the milk. Mellin's or other similar food is often of service. As the disease progresses the diet must be made more sustaining; but, unless the symptoms assume an adynamic type, highly nitrogenous food should be avoided until convalescence is assured.

The affected joints may be wrapped in cotton, or, better, in wool batting, and should be kept as quiet as possible by means of sand-bags or closely moulded well-fitting splints, but the bandages should never be tightly drawn.

Various local applications to the joints have been employed by practitioners for the relief of pain, but in our experience they have rarely seemed to be effective. The injection of from ten to fifteen minims of a one per cent. solution of carbolic acid into a joint from one to three times a day has been strongly recommended; we have had no experience with it. Simple warm water, concentrated solution of sodium carbonate (1 to 10), diluted tincture of aconite, laudanum, saturated solution of ammonium chloride, Fuller's lotion (sodium carbonate, six drachms; tinctura opii, one ounce; glycerin, two ounces; water, nine ounces), are among the most used local applications. In Germany the ice-bag or compresses of cold water are much employed. Blisters applied either above or below the joint sometimes allay pain and seem to do good: they are, however, especially useful in the advanced stages of the disease, when the inflammation lingers after the rheumatic tendency has been largely overcome. Under such circumstances their repeated application to the joint itself may be essential to a favorable result.

There are two more or less specific treatments of acute rheumatism. The older of these is the treatment by alkaline potassium salts. In carrying out the alkaline treatment, one ounce of the potassium salt dissolved in at least one pint of water is to be given during the twenty-four hours in divided doses. As the potassium citrate is converted in the system into potassium carbonate, and as it is much less disagreeable to the palate and irritating to the stomach than are the carbonates, it should always be preferred. Potassium nitrate is very irritant and very inefficient. The citrate may be given in a strong lemonade, the lemon juice concealing its taste and assisting its action; or, more agreeably, one drachm of the citrate in half an ounce of lemon juice may be put into a tumbler for each dose, and diluted at the time of taking with carbonic acid water from a siphon. It is usually necessary after from three to seven days to lessen the dose of the potassium salt, on account of its depressing influence.

The salicylic acid treatment is, however, much more effective than the alkaline method. The acid itself, sodium salicylate, ammonium salicylate, or oil of wintergreen may be employed. The acid given in capsules is the least disagreeable of the preparations, but is so apt to irritate the stomach that it can rarely be used with advantage. Sodium salicylate is exceedingly disagreeable, and is more depressing, and more apt to nauseate, than is ammonium salicylate; the latter salt is therefore the preparation which should be ordinarily exhibited. Oil of gaultheria contains methyl salicylate in such proportion that one hundred and sixty-nine parts of the oil represent one hundred and thirty-eight parts of the acid. It affords an excellent method of giving the salicylate, but has seemed to us upon the whole more apt to disturb the stomach than the ammonium salt, whilst its decided flavor makes it agreeable to some and extremely disagreeable to other patients. In some cases it may be combined with ammonium salicylate, but in the majority of instances the best results are to be obtained by giving the latter salt alone, in milk or in carbonic acid water. Twenty grains may be administered from three to six times a day. Decided cinchonism should be produced, but its appearance should be followed by the reduction of the dose. Delirium and a peculiar disturbance of the respiratory function, in which the movements become very rapid and deep, are spoken of as sometimes caused by the acid, but we have never seen them.

Marked alleviation of the symptoms usually follows salicylic acid tinnitus, but return of the symptoms, or, in other words, distinct relapses, are very common. Various opinions are held by authorities in regard to the power of small doses of salicylic acid in preventing these relapses; our own opinion is that it is better not to continue the drug in small doses, but after a decided result has been obtained to put the patient upon alkalies, giving overwhelming doses of the salicylates whenever symptoms of relapse occur. We have seen apparently excellent results from the continuous local application of the salicylates, especially of oil of gaultheria, to the inflamed joints. It is probable that under these circumstances sufficient absorption takes place to specifically affect the part. Salol, which has been commended, must, in order to be effective, be broken up by the intestinal alkaline juices, and is therefore uncertain in its action. Moreover, it contains about thirty-six per cent. of carbolic acid, so that if enough is given to produce a full salicylic influence there is danger of carbolic acid poisoning. It is therefore unfit for use in acute rheumatism. Salicin is a very feeble preparation, probably dependent for any influence upon its conversion into salicylic acid in the system, and is evidently to be condemned.

Antipyrin (ten grains) and antifebrin (five grains), two or three times a day, are alleged by various practitioners to have a specific influence like that of salicylic acid, and probably whatever power they may have is shared by phenacetin. These drugs are, however, much less efficacious than the salicylate, and are certainly fully as depressing. In obstinate cases, however, they may be tried.

Potassium iodide is sometimes used, but has seemed to us of very little value. Colchicum has been effective only when given in such large doses as to produce purgation. Quinine has no specific action, but is often of service when free sweating and evidences of weakness are manifested in persons who have been reduced by alkaline or salicylate treatment.

Violent cerebral symptoms are said sometimes to be present in rheumatism without high temperature, as the result of brain congestion. In all cases which we have seen, however, the nervous symptoms have depended solely upon the high bodily temperature, and have subsided at once upon the immersion of the patient in a cold bath and the subsequent reduction of temperature. Whenever the temperature is 108° F. or above there should not be the slightest fear in the use of the cold bath. The subsidence of the nervous symptoms under the use of external cold has been in our experience followed by the immediate return of the joint-inflammations, which had previously disappeared.

GONORRHŒAL RHEUMATISM.

A title applied to the occurrence of symptoms and lesions resembling those occurring in acute articular rheumatism, but due to gonorrhœal infection. The probability of various bacteria being concerned in the etiology of many cases of acute articular rheumatism has already been mentioned, and the frequency of the occurrence of rheumatoid symptoms in various infectious diseases has been stated. Especial importance is to be attached to the occurrence of such symptoms from infection by gonococci, from the close resemblance which they bear to the symptoms occurring in rheumatic fever.

ETIOLOGY.—That gonococci are the cause of the rheumatoid lesions is suggested by the repeated recognition of their presence in the fluid from inflamed joints, as shown by Petrone and Kammerer, and in the pus from tendon-sheaths, and by their presence in the diseased valves in acute ulcerative endocarditis (Leyden), and their discovery by Councilman in myocardial abscesses. Osler records that gonococci were cultivated from the blood of a patient with malignant endocarditis, and others have reported the presence of gonococci in the blood. Although the gonococcus alone may produce the lesions found in certain cases, other bacteria may also be concerned, and it is maintained by some that gonorrhœal rheumatism represents the accidental concurrence of acute articular rheumatism and gonorrhœa. Men are oftener affected than women, and urethral or vaginal gonorrhœa is the usual means of infection. Gonorrhœal ophthalmia and vulvo-vaginal catarrh in infants and children may also be followed by gonorrhœal rheumatism. It is stated to occur in two per cent. of the cases of gonorrhœa in adults, and may arise during the acute stage of the disease, although more frequently some weeks after the infection, and it may appear in gleet.

MORBID ANATOMY.—The lesions closely resemble those found in rheumatic polyarthritis. The fluid, although thin, contains more pus-corpuscles, and the adjoining bursæ and tendon-sheaths may become inflamed. The large joints are especially liable to be affected, the knee-joint being diseased in nearly three-fourths of the cases. One or many joints may be inflamed.

Endocarditis sometimes results, and may present the characteristics of a malignant endocarditis. Gonococci alone are to be found in the diseased valves, or other bacteria may be present. The aortic valves are said to be more often diseased than the mitral.

SYMPTOMS.—There is no essential difference between the symptoms of acute articular rheumatism and those of gonorrhœal rheumatism, with the exception that in the latter they are less severe and more obstinate. This relation may in part be due to the fact that fewer joints are usually simultaneously affected. The disease may be indicated by fleeting pains in the vicinity of the joints without fever, or by moderate redness, swelling, and pain of one or more joints, with slight elevation of temperature. In other cases sudden inflammation of the joint occurs with severe pain and marked swelling, especially in the knee-joint, but with moderate fever. The symptoms usually extend over a period of weeks or months, with exacerbations and remissions and possible complications, as endocarditis, pericarditis, pleurisy, or enteritis. The local inflammations ordinarily terminate in resolution, but when suppuration takes place adhesions may occur, with permanent deformity. The prognosis is generally favorable, even when endocarditis is associated. If the pyæmic symptoms of ulcerative endocarditis arise, the prognosis becomes grave.

DIAGNOSIS.—A gonorrhœal cause may be assumed for the rheumatic symptoms provided a recent infection has occurred. In obscure cases of gleet a gonococcal cause for the rheumatic symptoms may be overlooked. In general, fewer joints are affected in gonorrhœal rheumatism, the fever and pain are less extreme, the swelling persists longer, and antirheumatic treatment is of but little avail.

TREATMENT.—The treatment of gonorrhœal rheumatism is very unsatisfactory. We have never found the salicylates, colchicum, the iodides, or the mercurials to exert a distinct influence for good. In the acute cases, rest, fixation of the joints by splints, and blisters or the application of the thermo-cautery over the joints constitute the major part of the treatment.

In chronic cases, careful attention to the general health, the best possible hygienic surroundings, the administration of tonics and of arsenic, and the use of massage and passive movements, comprise about all that can be done by the physician. The utmost importance should always be attached to the local treatment of the genito-urinary organs; the obstinacy of the disease often depends upon the existence of a slight chronic gleet. The surgical treatment of the inflamed joints, by opening and irrigation, is said to have yielded satisfactory results.

CHRONIC ARTICULAR RHEUMATISM. CHRONIC RHEUMATIC ARTHRITIS.

DEFINITION.—A disease of obscure etiology characterized by stiff and painful joints.

ETIOLOGY.—Although chronic articular rheumatism may result from severe, protracted, or recurrent attacks of acute articular rheumatism, it usually presents no obvious relation to the latter condition. It occurs chiefly among the poor, especially among those living or working in cold, damp places. It is most frequent in adults after middle life. Men are more often affected than are women. It occurs at all seasons of the year.

MORBID ANATOMY.—The capsule of the joint is thickened, and adhesions are formed between it and the surrounding structures. The synovial membrane is thickened, vascularized, spreads over the cartilage, and adhesions form between the opposed surfaces. The articular cartilage is in part absorbed, and the articulating bones may become anchylosed, even consolidated, as is seen in the fusion of vertebræ.

SYMPTOMS.—Stiffness and pain in the diseased joint are the conspicuous symptoms. Swelling is usually but trifling. The stiffness is more marked in the early part of the day or after rest during the day, while pain is complained of on motion, or becomes persistent towards evening. The stiffness and pain are often more severe in wet weather. Acute exacerbations of the inflammation of the joint may occur, associated with slight elevations of temperature, compelling temporary rest. The longer the inflammation persists the more likely are the joints to creak on motion, and the degree of motion is more and more impaired. The extremities are flexed in various degrees, the rigidity is only partially overcome by passive motion, and in extreme cases the patient is bedridden because of the rigidity of the joints. Atrophy of the muscles follows, and the sufferer is often extremely emaciated. Several joints, both large and small, are usually affected, and complete disappearance of the symptoms from a diseased joint is rare. Complications are infrequent.

DIAGNOSIS.—The method of development of the symptoms of chronic rheumatic arthritis is sufficiently characteristic to enable the diagnosis to be made in most instances. Gouty arthritis is easily excluded by the history of the case, the absence of acute attacks in the great toe joint, and the usual polyarticular affection. The sometimes difficult differentiation from rheumatoid arthritis will be considered in the article on that subject.

PROGNOSIS.—The disease usually lasts throughout life, although for a long time the symptoms may be comparatively slight. Temporary improvement is likely to follow the occurrence of exacerbations.

TREATMENT.—Whenever in chronic rheumatism it is attainable, the subject should live in a mild, equable, dry climate, such as is found in Texas and other parts of the dry belt which runs northward from San Antonio and in certain parts of Southern California; if this be not possible, he should be most carefully protected from damp, from changes of temperature, and from the vicissitudes of the weather. The general bodily health should be assiduously maintained by careful management of the digestive organs, and by the abundant use of nutritious, especially of fatty and farinaceous, articles of food, and, whenever practicable, of cod-liver oil. The salicylates, the iodides, and the various alkaline carbonates may be used from time to time to subdue exacerbation, but the chief reliance must be upon the use of various baths, as in chronic gout.

The treatment of the joints by massage, and by the application of blisters, of ichthyol ointment, of mixed mercurial and belladonna ointment (equal parts), of oil of gaultheria, of weak alkaline solutions, and of other local remedies, is very important. In our experience good has chiefly been produced by massage and counter-irritation.

ARTHRITIS DEFORMANS. RHEUMATOID ARTHRITIS. RHEUMATIC GOUT.

DEFINITION.—A chronic progressive inflammation of the joints characterized by alterations of the capsule, cartilage, and bone.

From the resemblance which the symptoms in certain cases of arthritis deformans bear to those of chronic articular rheumatism, and from the fact that the former disease in rare instances follows the latter, it is probable that in such cases the etiology is the same. Frequently there is no such sequence, and the exciting causes are usually unknown. From the fact that severe destruction of the tissues of the joint and spontaneous dislocations follow injury and disease of the spinal cord, and that in rheumatoid arthritis the disease is frequently symmetrical and associated with hyperæsthesia, paræsthesia, and disturbances in the nutrition of the skin, hair, and nails, a neuropathic origin has been considered probable.

The disease usually begins in adult life, but becomes pronounced after middle life. In rare instances it may be found in young children. It is more frequent in women than in men, and heredity, hardship, and mental strain are of etiological importance. The disease may affect one or more joints: the monarticular form has directly followed injury to the joint. From its frequent occurrence among the poor, and from a certain resemblance in its symptoms to those occurring in gout, it has been called "poor man's gout."

MORBID ANATOMY.—Few or many joints may be affected, the larger, as the knee, hip, shoulder, and elbow, being oftenest diseased, although those of the hands and feet not infrequently first suffer. The vertebral joints sometimes show a marked degree of alteration. In extreme cases the capsule and ligaments become thickened, and the synovial membrane forms a series of fringes, at times transformed into polypoid excrescences, in which fibrous and fat-tissue may be found, and which when detached form free bodies. The synovial fluid is usually diminished, though sometimes it is in excess. The articular cartilage becomes fibrillated and softened, and cartilaginous outgrowths project beyond the edge, forming fungoid excrescences, sometimes of considerable size, portions of which may also become detached and form free bodies. The cartilage is also destroyed in places, especially in those exposed to the greatest friction. The surface of the bone is laid bare, and is transformed by sclerosis into an ivory-like material,—eburnation. In other parts absorption or new formation of the bone takes place, the latter perhaps extending into the cartilaginous excrescences, and extreme deformity of the joint results. Absorption of the bone may cause a nearly complete disappearance of the head of the femur or of the humerus, and the vertical diameter of the vertebræ may be shortened. The vertebræ may be thus consolidated by the union of bony excrescences from the contiguous surfaces. Partial dislocations are frequent, and especially at the hip a false joint may be formed at some distance from the acetabulum. The tendons near the affected joint may become thickened and the muscles atrophied, perhaps fibrous. Lateral bending of the fingers towards the ulnar surface of the hand frequently occurs, and the great toe may be inclined towards the little toe.

SYMPTOMS.—The disease usually develops slowly, beginning in a few joints, but extending in the course of years to many, with variations in the severity of the process. The first symptom is usually impaired mobility of the affected joint or joints, especially on rising or after prolonged rest. The incipient stiffness of the joint is relieved by exercise, but is increased as the joints become painful. The pain varies in degree, ordinarily being well borne, although sometimes very severe. Temporary exacerbations of the inflammation lasting a few days often occur, during which there may be slight swelling and tenderness of the joints. With the progress of the deformity motion becomes more impaired, and is associated with creaking, but is never absolutely restrained, although extreme flexion of the thigh, leg, and forearm may occur. The extreme impairment of motion occurs in the vertebræ, which when consolidated form a rigid spine. There is no elevation of temperature, except a slight rise during the acute exacerbations.

Closely allied are the arthropathies following injury to the nerves and spinal cord and occurring in locomotor ataxia and poliomyelitis. Their course, however, is rapid and usually painless, and, although associated with growth of the synovial membrane, is characterized by extensive destruction of the cartilage and absorption of the bone.

Another affection of the joints closely allied in its results to rheumatoid arthritis is the *malum senile*, or, from its frequent localization, *morbus coxæ senilis.* It is found exclusively in old people, and consists in fibrillation, softening, and destruction of the cartilage, absorption and eburnation of the bone, and corresponding deformity of the joints. Cartilaginous outgrowths are lacking, and the capsule of the joint becomes indurated. The pain is moderate. There is no tenderness, and the motion of the joint is usually lessened, although sometimes exaggerated, and is accompanied with creaking.

DIAGNOSIS.—The symptoms of rheumatoid arthritis are usually sufficient to permit the diagnosis to be made. During the earlier period of its development it cannot always be differentiated from chronic articular rheumatism. In the progress of the two diseases the former results in more extensive deformity of the joint, but without complete obliteration of its functions. Persistent chronic articular rheumatism tends towards anchylosis of the joint. Rheumatoid arthritis generally is limited to few and large joints; chronic rheumatic arthritis affects many and symmetrical joints.

PROGNOSIS.—Although the disease may come to a stand-still in its earlier stages, complete recovery does not occur. As a rule, the disease progresses, extends from joint to joint, and persists throughout the life of the patient, who may attain an old age.

TREATMENT.—In arthritis deformans, whenever it is possible, the patient should live in a dry, warm, equable climate; when this is not possible, every effort should be made by proper clothing to protect against damp and cold. The diet should be abundant and nutritious. Tonics and other drugs may be used to meet the symptoms as they arise, but the only remedies which seem to have influence upon the disease are iodine, preferably in the form of potassium iodide, and arsenic, which should be commenced in small doses and kept up continuously for months in ascending doses, great care being taken not to derange the digestion. The salicylates are sometimes useful in an acute exacerbation. Cod-liver oil is of great importance when there is a tendency to emaciation and loss of strength.

There can be no doubt as to the value of careful massage in affecting inflammatory exudations, and in combination with Swedish and other gymnastic movements in maintaining the mobility of the joints, the general health, and the nutrition of the muscles. Baths are very much used, especially sulphur baths; and annual resort to sulphur springs (in this country Sharon, Richfield, and the various sulphur springs of Virginia) often greatly protracts life and conduces to comfort. Hot salt-water baths are sometimes useful, and Strümpell very strongly recommends hot sand-baths. Counter-irritation over the joints by means of blisters or iodine may be exceedingly useful, but must be tried with caution.

MUSCULAR RHEUMATISM. MYALGIA.

DEFINITION.—Attacks of pain referred to certain muscles or to the tendons and fascia with which they are connected.

ETIOLOGY.—The term muscular rheumatism is largely one of convenience, it being probable that various affections have thus been designated. In certain cases a wry neck will immediately follow a tonsillitis, and the same infection is the presumable cause of both. The act of stooping may be followed by a tearing sensation in the lumbar region, and severe pain follow not differing from that occurring in a person whose back is exposed to a draught of air. To the latter condition alone is the term muscular rheumatism strictly applied. Its use should be more especially limited to muscular pains which are independent of any obvious anatomical lesion of the affected region or of any unquestionable cause. On the one hand, it is considered that exposure to cold and wet and the rapid cooling of a heated perspiring skin are exciting causes, a view which is favored by its more frequent occurrence among laboring men and, at times, in sufferers from chronic articular rheumatism; on the other hand, it has been regarded as a neuritis of the sensitive nerves of the affected region.

SYMPTOMS.—Pain is the conspicuous symptom, and may be a dull ache, or a piercing or tearing pain. It is aggravated and may become insupportable on motion of the muscles concerned, and may be relieved when they are relaxed, thus compelling the patient to assume the most extraordinary positions from the rigid contraction of the antagonistic muscles. The pain may be increased or relieved by pressure. It is unaccompanied by swelling, although an appreciable rigidity of the muscles is sometimes felt, and fever is absent. Single muscles or groups of muscles may be affected, and the myalgia may shift from one part to another. According to the localization of the pain various terms are applied, as *torticollis*, stiff- or wry-neck, in which case the sterno-mastoid and sometimes the trapezius are the seat of the disturbance, *lumbago*, when the muscles of the loin and their attachments are concerned, *pleurodynia*, the pectoral and intercostal muscles being involved, *omodynia*, with involvement of the scapular group of muscles, and *cephalodynia*, when the occipito-frontalis is affected.

A distinction is drawn between acute and chronic myalgia. The former is more likely to result from unknown infection or a single exposure to a draught of air. It may be accompanied with slight fever, and usually continues but a few days. Chronic myalgia is rather the result of repeated or prolonged exposure, lasts several weeks, and shows a tendency to recurrences. In such cases fibrous thickenings of the muscles have been found, especially in elderly people who have suffered from frequent recurrences perhaps continued over a period of years.

DIAGNOSIS.—This affection is usually easily recognized by the known exposure and the nature of the attack. Myalgia is to be distinguished from neuritis, which is limited to the course of the nerve. The nerve when inflamed is sensitive to pressure as it nears the surface of the body or overlies bony prominences, but is commonly unaffected by muscular action.

Torticollis is relieved by friction, and does not become insupportable on motion as does cervical spondylitis, which is tender to the touch. In lumbago the absence of fever excludes the backache of acute infectious diseases, and the examination of the urine and of the pelvic contents permits the exclusion of renal, rectal, uterine, or ovarian disease. Its usual brief duration enables inflammatory or neoplastic disease of the subjacent parts to be differentiated. Pleurodynia may be confounded with intercostal neuralgia, but sensitive points are lacking. Caries of the rib produces a tender as well as painful swelling, and pleurisy is characterized by physical signs. The pain from muscular rheumatism of the abdominal muscles may bear a slight resemblance to that of peritonitis, but the characteristic symptoms of the latter disease rapidly develop.

TREATMENT.—A severe acute attack of muscular rheumatism can often be immediately relieved by a free sweating. (For method of production, see Influenza, page 160.) The local application of heat by means of hot-water bags or sand-bags is often of service. Local blood-letting by cups or leeches will usually bring great relief. Blisters are even more powerful, but only in rare instances is such heroic treatment necessary. Sinapisms and irritating liniments are often useful. The application of a rapidly interrupted faradic current is frequently effective, especially after the subsidence of the first acute symptoms. The salicylates given for a few days in large doses (preferably ammonium salicylate) in rheumatic cases will generally produce an immediate cure. In protracted cases, with a marked tendency to recur, the basal condition is ordinarily gouty, and a more or less rigid application of the rules of treatment for chronic gout will usually be effective. Localized myositis of a non-rheumatic character sometimes occurs, and is scarcely to be distinguished from a rheumatic myositis: such a case would not, of course, yield to anti-rheumatic remedies, but should be treated by means of counter-irritation. When with a myositis, whether of rheumatic or of other origin, marked contraction of the muscles occurs, as in wry-neck, the one-hundredth of a grain of atropine sulphate should be injected directly into the muscle itself; severe pain may be immediately relieved by the addition of morphine sulphate to the atropine.

GOUT. ARTHRITIS URATICA. ARTHRITIS URICA.

DEFINITION.—A constitutional affection characterized by inflammatory and degenerative changes in various organs and tissues of the body, especially in the joints, associated with the deposition chiefly of sodium urate and by various disturbances in the functions of numerous organs.

ETIOLOGY.—Our knowledge of the pathological processes concerned in the causation of gout is but elementary. Numerous hypotheses have been offered, but none give satisfaction. The discovery by Garrod of uric acid in the blood in cases of gout led to the view that the formation of an excess of uric acid and its deposition in the tissues were the essential features of gout. He observed that there was a diminished elimination of uric acid during and an increased formation after the close of the attack of gout. The causes of such excessive formation were not ascertained. Garrod assumed that there was a diminished elimination of uric acid by the kidneys, and that in consequence of a lessened alkalinity of the fluids a precipitation of the urates was accomplished; while Roberts asserted that a relatively insoluble biurate was formed from the excess of uric acid in the blood. Pfeiffer maintains that there is no such excessive formation of uric acid in gout, and that the important characteristic is the presence of modifications of uric acid soluble with difficulty in various organs and tissues, therein practically agreeing with the view of Roberts. Charcot suggested that an increased formation of uric acid takes place in gout in consequence of hepatic disturbance, and Cantani, Ebstein, and Senator consider that there is an increased formation of uric acid in various tissues and organs of the body. Horbaczewski, however, asserts that uric acid is formed from the nuclein of the white blood-corpuscles, and Von Noorden states that the elimination of uric acid in gout is not essentially different from that in health.

Of importance in the origin of gout is heredity, a tendency to this disease having existed in many families through centuries. Hutchinson observed that the younger children were more prone to typical gout than the elder, and that the inherited tendency was more often transmitted by the father than by the mother. It has been suggested in explanation of these observations that the manifestations of gout usually appear towards the close of the child-bearing period, and hence the older children are begotten and born before active gout exists in the parents. Although the symptoms of gout appear most frequently in middle life, rarely young children may be affected. Men are more often diseased than women, especially wealthy persons of sedentary habits living in luxury, although those suffering from poverty and hardship are not exempt. The disease affects rather the fat than the lean, and is not infrequently associated with glycosuria.

Of especial importance in the production of gout are the habits of the individual. The free use of alcoholic drinks, especially port, madeira, sherry, burgundy, champagne, ale, and porter, is often the exciting cause. The prevalence of gout in England, France, Holland, and Germany is attributable to the frequent use of these beverages, while in the United States, where strong wines, ale, and porter are not in general use, typical cases of gout among the native-born are rare. The abundant use of red meat has generally been regarded as an exciting cause, but recent investigations (Pfeiffer) tend to disprove the correctness of this view. The relation between lead poisoning and gout to which Garrod calls attention has been both advocated and disputed. Recent as well as early observations in France, England, and America confirm those made by Garrod as to the occurrence of typical attacks of gout in cases of lead poisoning independently of the usual exciting causes. The rarity of gout among lead-workers in this country suggests that exposure to lead alone is not a sufficient cause. The immediate attack may be induced by alcoholic or sexual excess, mental excitement or depression, exposure to cold, or injury. The usual localization in the great toe joint is attributed to its frequent exposure to injury and its comparatively feeble circulation. Garrod observed that in persons who had previously suffered from rheumatism the rheumatic joints were the first to be attacked with gouty inflammation, and Charcot found that in a gouty paralytic the joints conspicuously affected were those of the paralyzed side.

MORBID ANATOMY.—The changes characteristic of gout are found within and in the vicinity of the joints, especially of the metatarso-phalangeal joint of the great toe. The synovial membrane is thickened and villous. A chalk-like deposit of urates, especially sodium urate, takes place in the capsule of the joint, its cartilages and ligaments, and eventually in the neighboring tendons, fasciæ, bursæ mucosæ, periosteum, and bone. The cartilage becomes fibrillated. The alterations of the joints are more frequent in those of the hands and feet, although the larger joints are not spared. The chalky masses in the vicinity of the joints are called tophi, and may be as large as the tip of the finger. Tophi are also formed in the auricular cartilages, more rarely in the cartilages of the nose. Pfeiffer regards Heberden's nodules, the rounded knobs of bone projecting from the second and third phalanges, as a result of gout, maintaining that, although these may be found in persons who have had no attacks of gout, the urine of such patients has the gouty characteristics. He also asserts that in chronic articular rheumatism the joints above mentioned are unaffected. In chronic gout the kidneys are frequently in a state of fibrous atrophy, the urates being deposited both in the pyramids and in the cortex. Hypertrophy of the heart is associated with this gouty kidney, and coronary sclerosis, fibrous myocarditis, and chronic endocarditis, especially of the aortic and mitral valves, are of frequent occurrence. Gastro-enteric catarrh, inflammation of the serous membranes, and pneumonia are frequent complications.

SYMPTOMS.—Gout is conveniently divided into the articular and visceral varieties. Articular gout is primary, typical, acute, or chronic, while visceral gout is atypical and chronic in character. Acute articular gout or gouty arthritis occurs in the form of attacks which are usually recurrent, the intervals being long or short, separated by months or years, and the attacks are preceded by digestive disturbances, muscular pains, headache often severe, chilly sensations, slight elevation of temperature, and scanty urine of high specific gravity with a lateritious sediment. In the course of a number of days, usually after midnight, the patient is aroused by a sudden attack of severe pain of a boring character, oftenest in the metatarso-phalangeal joint of the great toe,—*podagra*. More rarely other joints may be involved, either the small joints of the feet or hands, or the knee, hip, shoulder, or elbow joint. The affected joint becomes red, swollen, and tender, and its superficial blood-vessels are congested. The temperature rises three or four degrees, and the pulse is moderately accelerated and at times irregular. The pain diminishes towards morning, and during the day the inflamed joint is comparatively comfortable, the relief to the pain being associated with lowering of the temperature and a sour perspiration, although the digestive disturbances, especially an acid stomach, constipation, or diarrhœa, are present. A recurrence of the pain and other symptoms takes place during the successive nights for a week or more, the severity of the symptoms gradually diminishing as the attack ceases. The redness, heat, and swelling of the joint disappear, the skin itches, and the epidermis over the inflamed joint separates in scales. The premonitory diminution in the flow of urine persists throughout the attack, and there is a diminished secretion of uric acid, while the blood contains an excess of urates. In the intervals between the attacks the flow of urine increases and an excess of uric acid is formed. The presence of uric acid in the blood may be determined by the method suggested by Garrod. From one to two drachms of blood-serum obtained by venesection or wet-cupping are placed in a watch-glass with a few drops of acetic acid. A thread is placed in the mixture and allowed to remain for twenty-four hours, at the end of which time crystals of uric acid are seen attached to the thread. A like result occurs if serum obtained from a blister is used instead of that formed in the clotting of blood.

In chronic articular gout the attacks are of longer duration, although less severe, and numerous joints may be diseased. This variety may be chronic from the outset, especially in debilitated persons. Permanent deformity of the joints results, evinced by persistent thickening of the tissues, the formation of tophi, partial dislocation, and muscular atrophy.

The skin overlying the tophi may become ulcerated, and a watery discharge escapes containing chalk-like granules composed of acicular crystals of sodium urate. The resulting fistulous ulcers may heal or extend into the joint, and in extreme cases the phalanges of the fingers and toes may become detached.

The term visceral gout is applied when persons of gouty antecedents or those suffering from chronic articular gout are affected with chronic catarrhal affections of the various mucous membranes, especially of the respiratory, intestinal, and urinary tracts. In them the arterial tension becomes increased. Arterio-sclerosis, especially of the vessels of the heart and kidneys, often occurs. Symptoms of chronic endarteritis and valvular endocarditis, especially at the aortic and mitral orifices, are frequent. The heart becomes hypertrophied, interstitial myocarditis is often present, and the kidneys atrophy. In the latter case the urine is copious, of low specific gravity, and contains a large trace of albumin, and occasionally hyaline casts. Fibrous atrophy of the kidney, with the deposition of urates in this organ, may occur with normal joints in persons of gouty antecedents. Sand, gravel, or calculi of uric acid may form, and symptoms of vesical or renal calculi follow. Attacks of mild or severe uræmia may result, and fatal inflammation of the serous membranes, especially of the pleuræ and the pericardium, occur. Meningitis, diphtheritic enteritis, pneumonia, asthma, eczema, transitory and permanent glycosuria, keratitis, and iritis are occasional complications.

The designation metastatic, retrocedent, or retrograde gout is used when there is a rapid improvement in the arthritic condition associated with severe symptoms of visceral disease. In such cases the gout is said to attack the stomach when severe epigastric pain, nausea, vomiting, and diarrhœa, followed by collapse, occur. Metastasis is thought to affect the heart when palpitation, dyspnœa, and angina pectoris take place. The presence of delirium, convulsions, paralysis, or coma is attributed to involvement of the brain; while intense sciatica and a sense of constriction around the chest or loins suggest a metastasis to the spinal cord. Such retrocedent attacks are more likely to occur in chronic gout, are not attended with the deposition of crystalline urates, but in many cases are probably due to coronary sclerosis, cerebral hemorrhage, or uræmia, according to the seat and nature of the manifestations.

DIAGNOSIS.—Typical attacks of acute gout are unmistakable in virtue of their seat and course. Chronic articular gout is easily to be recognized provided tophi are present. If they are absent, the history of the earlier attacks and the characteristics of the blood and urine may permit the diagnosis to be made. Arthritic tophi when not recognizable as chalky masses are to be distinguished from the excrescences of rheumatoid arthritis by their frequent superficial seat and less resistance. The recognition of visceral gout is based upon the association of the symptoms with evidence of articular gout, or upon their occurrence in persons of gouty ancestry.

PROGNOSIS.—The attacks of primary arthritic gout are usually free from danger, especially when they occur in persons in the prime of life. Recurrent attacks are always to be expected, and are not incompatible with a long and active life. Chronic gout in elderly and debilitated persons is a source of danger from the associated weakness and the liability to diabetes, pneumonia, cerebral hemorrhage, and incurable disease of the heart and kidneys.

Lithæmia.—This term was introduced by Murchison to characterize an assumed condition of the blood attributed to temporary or persistent functional derangement of the liver. This derangement was considered to be indicated by disturbances of digestion, circulation, and secretion, but especially by an excess of uric acid in the urine and the frequent deposition of uric acid and urates. Although the symptoms described were those often found in gout and in the children of gouty parents, yet they might occur in persons wholly free from the suspicion of gout. It was Murchison's view that gout was a result of lithæmia. These symptoms in gouty persons were those to which the term gouty dyspepsia, or latent, suppressed, anomalous, or irregular gout, was applied. Da Costa has recently given increased popularity to the term lithæmia, which is not likely to yield to its most recent substitute *uric-acidæmia*.

It is recognized that the causes important in producing typical attacks of gout are also influential in the production of the symptoms now under consideration. These relate primarily to disturbances of digestion, such as an irregular appetite, coated tongue, a disagreeable taste in the mouth, acid eructations, flatulence, a sensation of epigastric weight and fulness, and constipation alternating with diarrhœa. Palpitation of the heart, and a slow, irregular, or intermittent pulse, are frequent. The patient suffers from vertigo, occasional attacks of frontal headache, blurred vision, neuralgic pains, and other disturbances of sensation, and from muscular cramps in various parts of the body. The mental condition is likely to be affected, the patient being restless, irritable, hypochondriacal, perhaps hysterical. The gouty origin of such symptoms may be admitted when there is evidence of gout in the joints, or when the cardio-vascular and nephritic disturbances mentioned under chronic gout are present, or when the symptoms occur in the children of gouty parents. The demonstration of uric acid in the blood by Garrod's test would also be evidence in favor of a gouty origin of these symptoms, although an excess of uric acid in the blood, the *uricæmia* of Flint, also exists in leukæmia and chlorosis, in which the enumerated symptoms are absent. The presence of a lateritious sediment in the urine, to which so much diagnostic importance is often attached, even Murchison admits may exist for years without discomfort. Da Costa, although reiterating the importance of the frequent occurrence of high-colored, acid urine with high specific gravity and brick-dust sediment in the diagnosis of lithæmia, would retain this term even if the urine were of low specific gravity and without excess of uric acid, the "lithæmic" symptoms in such cases being attributed by him to the effects of other products of tissue-metamorphosis than uric acid.

Lithæmia is, therefore, to be regarded purely as a term of convenience, by preference applied to the association of few or many of the symptoms above mentioned, with frequent or persistent lithuria in contradistinction to the association of these symptoms with calcic oxalate in the urine,—oxaluria,—or with a phosphatic sediment,—phosphaturia,—or without any sediment whatsoever. When such symptoms occur in persons with typical manifestations of gout or with an inherited tendency to this disease, they should be regarded as evidence of gout, pure and simple, neither "latent" nor "suppressed," but perhaps alternating with or eventually to be followed by typical attacks of arthritic gout.

TREATMENT.—In the acute gouty paroxysm, large doses of the salicylates sometimes give relief, as in rheumatism, but are less effective than is colchicum; vinum colchici radicis (ten to fifteen drops) or the alkaloid colchicine (dose, one-hundredth of a grain) may be given three or four times a day. Antipyrin, antifebrin, and phenacetin are sometimes useful in allaying the pain. When the patient has already suffered from repeated paroxysms, depleting or sedative treatment may be dangerous, and alcohol, digitalis, and other stimulants may be required. Under any circumstances we believe it to be bad practice to attempt to shorten very greatly a gouty paroxysm by the use of large doses of colchicum, but it is especially so when the paroxysm takes other form than that of a podagra. We have seen fatal metastasis to the alimentary canal under such circumstances apparently produced by moderate doses of colchicum.

The non-medicinal treatment of the acute paroxysms of gout consists in enforced rest in bed or on a couch, restriction to light diet, and the use of mechanical protection to the inflamed joint, which should be kept well elevated and wrapped in cotton wool, dry, or saturated with laudanum and tincture of aconite if this afford relief. It is usually well to begin the medicinal treatment by a free mercurial purge, and during the whole course of the attack the bowels should be kept loose by salines or other laxatives. Pain should be relieved, but rarely suppressed, by opium, aided at night by chloral, trional, or sulphonal to obtain sleep. The depurant remedies consist of the alkaline salts of lithium and potassium, which may be given in the form of natural mineral waters, which are, however, generally of such feeble constitution that they must almost universally be artificially reinforced. Moreover, these mineral waters have no curative properties other than those of similar artificial preparations, their apparent superiority resting chiefly upon the large amount of water which is taken with them: so that expense can be saved and as good results achieved by the use of artificial alkaline waters made of such strength that the patient should take at least a quart a day.

A very excellent combination, which is especially useful when there is uric acid gravel, is that of formula 4: the benzoic acid converts the uric acid into the soluble hippuric.

The remedies believed to be directly useful in chronic gout and between the paroxysms of acute gout are the alkalies, especially the lithium salts, the salicylates, colchicum, and piperazin. Of the lithium salts the citrate is the one usually employed (dose, five to ten grains three times a day). Colchicum given for a length of time is often very effective. In those cases in which colchicum does most good the salicylates are usually of little service, and *vice versa.* Of the salicylates the strontium salt is of special value, because it can be given in capsules (five to ten grains three times a day) without causing irritation of the stomach; although its influence as a salicylate is slower than that of other salts, it acts steadily and persistently. Moreover, it very commonly improves instead of disordering digestion, and is especially useful when there is a tendency to fermentative changes in the alimentary canal with consequent intestinal flatulence. The long-continued and free use of salicylates may produce cardiac weakness. Piperazin is believed to act simply by rendering uric acid soluble; in many cases of chronic gout it seems to achieve very little, but in some cases when given in large doses (ten to fifteen grains three times a day) its influence is marked. The oncoming of giddiness and weakness should be the signal for its withdrawal.

In lithæmia, in chronic gout, and between the paroxysms of a podagra, the chief reliance must be upon exercise and the proper regulation of the diet. The exercise should be carefully adapted to the strength of the individual case, and as the strength increases should be steadily increased in severity. It should always be sufficiently violent to cause free sweating, and should be followed by a bath. In a subject of fair degree of power regular training under an instructor is of the greatest advantage; in many cases habitual hard out-door exercise can alone secure comfort and health.

The local treatment of gouty deposits in the muscles and fibrous structures and of gouty arthritis is often of great importance. Massage may be very serviceable or harmful as it is employed skilfully or unskilfully. Counter-irritation by means of blisters often does great good, but requires care in its use. Faradization with a very rapidly interrupted current is frequently of great service in gouty myositis of a not too acute type; the continued galvanic current to the gouty joint is recommended by some, but we doubt whether it ever does any good. (See also Neuritis, page 603.) Local hydro-therapeutic treatment is often of great value; water as hot as can be borne thrown very forcibly by means of a needle-spray and followed by careful massage is not rarely effective in dispersing local gouty or rheumatic exudations. The application of tightly fitting wool coverings, kept on day and night, frequently brings great relief, especially when the larger joints, such as the knee, are in question.

In all classes of gouty patients fresh eggs, milk, the white meats (except pork), chicken, and game, should constitute special articles of food. Strong stock soups and alcoholic stimulants are usually to be entirely forbidden. When in feeble cases alcoholic stimulants are required, well-diluted whiskey or other spirit is always preferable to any malt liquor or wine. The diet of gouty or lithæmic patients must be various. When the subject is robust and vigorous it is highly important to reduce the amount of food taken to just sufficient to prevent loss of strength and weight. When the nutrition and vital forces are on a very low level it may be necessary to urge the taking of more food. When there is a great tendency to the accumulation of fat, with robustness of constitution, the fat should be largely removed by exercise; but there are fat anæmic gouty subjects in whom starchy foods must be withdrawn almost as rigorously as in the diabetic. The use of cane sugar should be reduced to the minimum; in fatty cases all sweetening except with saccharin should be interdicted. Starchy food should always be largely reduced. Potatoes should be used very sparingly, if at all. Bread is not to be denied, but, at least in part, gluten, bran, or whole wheat bread should be used. Most acid fruits and vegetables, such as strawberries, tomatoes, sorrel, etc., should be forbidden, but grape-fruit and lemons may be allowed, as well as sweet apples, oranges, and grapes. Whenever the fruit deranges the digestion it should be withdrawn. In robust acutely gouty people red meats should be permitted in small quantities, but in feeble chronically lithæmic people should be used freely. When the digestion and the strength are good, a diet largely composed of green vegetables is often of service. In severe chronic gout or lithæmia, especially if there be digestive disturbances, the exclusive milk diet should be essayed. From five to seven pints of partially skimmed milk may be given daily. Separator milk should never be allowed. The milk should always be taken slowly, a mouthful at a time,—warm or cold, but never boiled. In most cases in from two to four weeks it is necessary gradually to take the patient off the milk diet, but in some instances the best results are achieved by a continuance of the milk. We have seen the hardest labor performed for months by gouty individuals who were enabled to do their work and freed from suffering by restricting themselves to an absolute milk diet. Any constipation which may be produced must be overcome by the daily use of laxative remedies; in some instances oatmeal or other laxative farinaceous foods may be employed with advantage, and very frequently their addition to the milk diet should be the first change when the latter is being withdrawn.

In the treatment of chronic gout the use of baths is of the greatest importance. Every lithæmic subject should form the habit of taking the Turkish bath once a week, even if there be no very pronounced symptoms of the diathesis. Under such practice the glands of the skin become habitually active, so that the kidneys are assisted not only by the sweating at the time of the bath, but by the free perspiration which occurs at other times. If the Turkish bath be not attainable, ordinary hot baths may be used at regular intervals, but under all circumstances the body should be sponged down with cold water after taking the hot bath. When the lithæmic symptoms are acute, the Turkish bath, the steam bath, or the simple hot or medicated water bath, may be used daily, or more frequently or more seldom according to the needs and strength of the individual. The pine-needle bath, which is made by passing steam through the leaves of the *Pinus sylvestris*, is especially serviceable in sciatic and other forms of rheumatic nerve disease. In Europe the so-called mud—really the peat—bath is much used, and is undoubtedly often effective. It is probable that all these medicated baths act rather by increasing the excretion from the skin than by any specific influence of their constituents, though the sulphur baths may be an exception to this rule.

The climatic treatment of gout and rheumatism is very important; in many cases the disease can be kept in abeyance after failure of treatment in a bad climate by removal to some dry, equably warm region, such as the interior of Southern California, or the lower part of the dry belt which runs from San Antonio, Texas, northward to Colorado Springs. In the medicinal treatment of chronic gout it is essential to regulate carefully the action of the digestive organs by the use of such remedies as are called for by the symptoms in the individual case. Thus, in plethoric gouty subjects with constipation frequent purgation is of service; on the other hand, in feeble subjects with a tendency to gastro-intestinal catarrh, silver nitrate, bismuth with carbolic acid, and similar remedies, may be required.

In chronic gout, life may often be prolonged by an annual visit to some mineral spring. The good results are largely due to the change of scene, freedom from care, and the enforced diet and exercise, but natural mineral waters have some especial value. All springs may be divided into the simple hot spring, the alkaline-saline spring, and the sulphur spring. There is no reason why better results should be achieved in Europe than in America, except in the superior organization of the European health-resorts, a superiority which is disappearing year by year. Indeed, the mineral springs in Europe do not in their power and variety equal those of this continent. There can be no doubt that patients are better suited by one class of springs than by another, but we have never been able to frame any *a priori* rules of selection which were entirely satisfactory. When in any cases the habit is plethoric, with constipation and other evidences of torpor of the hepatic and other glandular apparatus of the alimentary canal, the very active saline springs, such as Carlsbad and Contrexéville, are especially effective. If in any case the abdominal condition just spoken of is conjoined with a tendency to weakness, the feebler saline springs, such as Wiesbaden, should be preferred. Sulphur springs have seemed to us especially useful when the joints are largely affected or when there is a marked tendency to anæmia and weakness. In any individual case, however, experience is the most important guiding principle, and the different springs may be tried at different times until one is found to be especially effective. When there is an anæmic tendency, excellent results are obtained by following the course of saline waters by residence at a ferruginous spa.

DIABETES MELLITUS.

DEFINITION.—A chronic disease characterized by the persistent presence of sugar in the urine, and, in severe cases, by an increased flow of urine, excessive thirst, digestive disturbances, and progressive loss of flesh and strength.

Sugar is always present in the blood and lymph, and Baumann has shown by means of delicate tests its constant presence in normal urine. Such sugar is in part absorbed directly by the intestine, but more largely results from the transformation into glucose of the carbo-hydrates, and to a less extent of the albuminous constituents, of the food. Such transformation is effected by the pancreatic and intestinal juices, and the glucose is absorbed by the portal vein. If more is absorbed than is needed for the immediate performance of the functions of the body, the excess is stored as glycogen in the liver and muscles, to be subsequently utilized when needed, and the remainder is transformed into fat. Some of the sugar in the blood may also be formed in the muscles. An excess of sugar in the blood may thus result from the increased supply of sugar or sugar-forming food in the diet, when an alimentary or dietetic glycosuria results, ceasing upon attention to the diet. An excess may also result from the failure on the part of the body to decompose or store that which is introduced. This may be due to a disturbance of the portal circulation dependent upon faulty innervation of the blood-vessels or to disturbances in the function of the liver, in consequence of which more glucose enters the blood without being transformed into glycogen in the liver, or an excessive transformation of glycogen in the liver takes place, setting free an excessive amount of sugar. (For the further consideration of this subject see Melituria.)

ETIOLOGY.—Diabetes is a disease most frequently found in middle life, although it may occur in children and, very rarely, in infants. It is more common among men than among women, and heredity often appears of importance. All authorities recognize its considerable frequency among Jews. Syphilis and gout are generally considered as of etiological importance. Grube states that in one hundred and seventy-seven cases gout or ancestral gout was of frequent occurrence. Fat persons appear predisposed, and Cantani assigns importance to the excessive use of sweetened food and drink.

The importance of affections of the nervous system in the production of diabetes is shown in the frequency with which shocks and strain of the nervous system are immediate antecedents. In like manner injuries, especially those to the head, cerebral hemorrhage, and tumors of the brain, insanity, epilepsy, and hysteria, also locomotor ataxia, are at times intimately connected with diabetes. In a number of cases diabetes has so immediately followed an acute infectious disease and exposure to cold that these have been considered as of etiological importance.

Disease of the pancreas has been found, especially of late years, to be of importance in the etiology of diabetes. Cowley in 1788 first noted this concurrence, and since then it has been frequently seen, especially in consequence of the observations of Lancereaux in 1887. Hansemann states that during ten years there were examined at the Berlin Pathological Institute forty cases of diabetes in which pancreatic disease was present, nineteen cases of pancreatic disease without diabetes, and eight cases of diabetes without pancreatic disease. Proof of the etiological importance of pancreatic disease in the production of diabetes was furnished by the experiments of Von Mering and Minkowski in 1889. They showed that the complete removal of the pancreas in dogs was followed within twenty-four hours by the characteristic symptoms of severe diabetes, ending fatally in a few weeks. This result did not follow ligature of the duct. If a very small portion of the pancreas was left in the animal, mild diabetes occurred. Sandmeyer observed that when about four-fifths of the pancreas were removed a mild diabetes resulted. which later became severe and ended in the death of the animal. These experiments suggest that it is a function of the pancreas to control the metamorphosis of sugar in the body, and that when this function is destroyed the sugar is eliminated by the kidneys. There is no satisfactory explanation of this result, although Lépine suggests that the pancreas produces a ferment which is necessary to the normal metamorphosis of sugar.

MORBID ANATOMY.—In many cases of diabetes no lesions are to be found. In others the alterations observed are to be regarded as complications of the disease, while in a certain number of cases lesions of the pancreas and tissues in its vicinity are present. Although glycosuria has been repeatedly observed in the presence of lesions, especially tumors and hemorrhage in the vicinity of the calamus scriptorius in the fourth ventricle, it is questioned whether the accompanying glycosuria should be regarded as necessarily indicative of diabetes. The lesions of the pancreas which have been found in diabetes are various. They consist of atrophy, fatty degeneration, suppurative and fibrous inflammation, concretions, cysts, and tumors. The liver is often hypertrophied, in part from the enlargement of the cells, in part from the presence of an increased quantity of blood. The periphery of the lobules frequently gives a reddish-brown color when tincture of iodine is applied, in consequence of the presence of glycogen. The kidneys are often enlarged and injected, and the presence of glycogen may be recognized by the application of iodine to the tubes of Henle near the bases of the Malpighian pyramids. At times fatty degeneration of the tubular epithelium is present. Numerous instances have been reported of fat-drops in the blood and of fat-emboli in the lungs, especially after death from coma. Glycogen has been found in the polynuclear leukocytes. The lungs are frequently diseased in diabetes, evidences of tuberculosis being often observed. The appearances of acute fibrinous pneumonia are frequently present, and gangrene of the lung is not a rare occurrence.

SYMPTOMS.—The onset of diabetes is usually gradual, the symptom first attracting attention being an increase in the quantity of urine. Various manifestations of disturbance of digestion or of the nervous system may precede polyuria, and are likely to follow this symptom. The abundant flow of urine is associated with a frequent desire to pass water, an abnormally large quantity being evacuated at each effort. The quantity secreted in the twenty-four hours may vary from three quarts to several gallons. It is diminished during intercurrent febrile affections, and a sudden diminution in quantity may immediately precede an attack of coma. Exceptionally there may be no increase in the flow of urine. Next in importance to polyuria as an early symptom is excessive thirst, often compelling the patient to have a pitcher of water at the bedside, from which frequent draughts are made during the night. The intensity of the thirst is in direct proportion to the frequency of micturition and the quantity of urine passed. This craving for water is a nervous symptom, and may be regarded as an attempt of the organism to eliminate the excess of sugar present in the blood. The appetite is usually excessive, and, despite large quantities of food, the sensation of hunger persists. Polyuria, polydipsia, polyphagia, and glycosuria continue throughout the disease, and are associated with various disturbances of nutrition. The patient loses flesh and strength. The skin becomes dry, and is frequently the seat of pruritus, especially near the anus and the genitals. Eczema, boils, and carbuncles often occur. In the later stages of diabetes gangrene of the skin is frequent. Hanot and Chauffard in 1892 reported the occurrence of cutaneous pigmentation, and since then, according to Mossé and Daunic, eight cases have been reported. A pigmented fibrous liver is associated. The gums swell and bleed easily, and the teeth become carious, the presence of sugar favoring bacterial growth and its destructive influence upon the teeth. The odor of the breath is often fruity. Cataract occasionally occurs, usually in the later stages of the disease. During the loss of flesh and strength various disturbances of the nervous system arise. The patients are depressed, suffer from headache and sleeplessness, and are especially prone to attacks of neuritis, resulting in pain and other disturbances of sensibility. Localized muscular paresis and absent patellar reflexes are relatively frequent. Disturbances of vision may be connected with retinitis or muscular paresis. The loss of sexual power in male patients often occurs, and may be an early symptom.

Diabetes is unaccompanied by fever. There is no disturbance of circulation, except a weakening of the pulse in the later stages of the disease. The respiration is usually undisturbed, except in diabetic coma or in complicating pulmonary affections or in asthma. The lungs are frequently the seat of tubercular processes, and become readily infected by pneumococci; hence chronic pulmonary tuberculosis and acute pneumonia are complications of relative frequency. A complication of occasional occurrence, especially late in the disease, is nephritis characterized by albuminuria and dropsy. Cystitis is occasionally met with, and gas, presumably carbonic acid, has been observed to have formed in the bladder, attributed to fermentation of the sugar in the urine from the presence of fungi. The urine is acid, its specific gravity from 1025 to 1050 and upward, although when diabetes is complicated with a fibrous nephritis the specific gravity may be below 1020. It is clear, of a pale yellow color; the odor at times is fruity, and has been compared to that of new-mown hay. This peculiar odor is due to the presence of acetone, which is sometimes accompanied by diacetic acid, the urine then becoming of a reddish-brown color on the addition of a few drops of dilute ferric chloride solution. The presence of these constituents as well as that of β-oxybutyric acid, according to Weintraud, is due to the disturbed oxidation of the albuminous substances in the food and in the body. The high specific gravity is owing to sugar, of which ten per cent. or more may be present. The tests for glucose are given under Examination of the Urine. The quantity of sugar eliminated is generally in proportion to the quantity of urine passed, and may vary in twenty-four hours from several ounces to two pounds. It is usually increased when the food contains abundant starches and sugar, and diminished when such articles of food are excluded from the diet, when excessive muscular exercise is taken, or when intercurrent attacks of fever occur. The elimination of urea and of the phosphates is markedly increased. In a case reported by Kobert fat was periodically passed in the urine. Traces of albumin are frequent, even in the absence of nephritis. The fæces are usually less abundant than in health, and in rare instances contain either liquid or solid fat.

Diabetes is conveniently divided into the mild and severe varieties, although this distinction is not absolute, since mild cases may become severe. The mild form may exist for years in apparently healthy, often well-nourished individuals, the glycosuria being accidentally discovered. The morning urine may be free from sugar, while that voided a few hours after a meal of carbo-hydrates shows its presence. Severe diabetes is oftener found in the young, or in those mild diabetics who from inability, ignorance, or negligence have not lived upon an antidiabetic diet. Emaciation is conspicuous, the grave symptoms above mentioned are present, and the patient is likely to live but a few years. In mild diabetes, when carbo-hydrates are taken, there is usually less than one per cent. of sugar in the urine, and this disappears in the course of a few days when an antidiabetic diet is adhered to. The longer the time necessary to produce the disappearance of the sugar the more closely allied is the case to the severer type. In severe diabetes the percentage of sugar is higher, and glycosuria persists despite the use of a diet largely restricted to albuminates. Naunyn has observed in certain cases that more sugar is excreted with a diet of abundant meat than when but little meat is taken.

The term diabetic coma was first used by Kussmaul to include a series of severe symptoms ending in coma which immediately precede death in certain cases of severe diabetes. The coma might occur suddenly and unexpectedly, or be preceded by increasing debility, excessive mental or physical exertion, digestive disturbances, and inflammatory affections, especially pneumonia. A brief period of headache, restlessness, wakefulness, or excitement precedes the drowsiness, sopor, and coma, which are in rapid sequence. Diabetic dyspnœa is often associated, and is characterized by frequent, deep, and somewhat noisy respiration, perhaps accompanied with a dusky skin. The breath presents the odor of acetone. The ferric chloride test indicates the presence of diacetic acid in the urine, and Kulz and others state that peculiar casts are frequently to be found in the urine before and during the attack of coma. According to Von Mering, abundant oxybutyric acid or ammonia in the urine is indicative of a threatened coma. The cause of diabetic coma is unknown, although probably in considerable part an auto-intoxication from a variety of chemical substances, while in certain cases fat-embolism may be of importance, especially in the production of dyspnœa, as suggested by Sanders and Hamilton. The coma lasts a few hours or a few days, and generally ends in death.

Diagnosis.—A suspicion of diabetes should arise when the patient complains of polyuria,and even in a young child, if there is incontinence of urine, diabetes is to be considered as the possible cause. The diagnosis consists in the determination of the persistent, not the transitory, presence of sugar in the urine. For this purpose the examination of the urine voided several hours after a meal containing carbo-hydrates has been taken should be made in doubtful cases, and cases of intermitting diabetes are to be borne in mind in which periodical examinations of the urine may be necessary owing to the temporary absence of sugar from the urine even after farinaceous food. The routine examination of the urine for sugar will often explain the origin of mental or physical debility, persistent neuralgia, and genital or anal pruritus without apparent cause. It is also to be remembered that designing persons, the hysterical for instance, may add sugar to the urine after it is passed. When in any case of diabetes there is a large excretion of sugar without a corresponding polyuria, the existence of organic disease at the base of the brain should be suspected.

Prognosis.—Diabetes mellitus is essentially an incurable disease, although under appropriate treatment the milder cases may live throughout a period of many years. In severe diabetes death usually takes place in from one to three years. Even in mild cases the prognosis must be guarded, from the difficulty of adhering to a restricted diet and the inability to avoid mental and physical strain or excess. The younger the patient the worse the prognosis. Fat diabetics usually suffer less and live longer than the lean. The course of a mild diabetes may suddenly or rapidly become serious from an intercurrent pneumonia or a complicating tuberculosis. The odor of acetone in the breath, the ferric chloride reaction in the urine, and the unexpected appearance of casts are bad prognostic signs, the successive occurrence of each being of the gravest import.

TREATMENT.—Diabetes due to some gross lesion encroaching upon the medulla oblongata is to be relieved by curing the original disease or not at all. Thus, we have seen antispecific treatment by curing a cerebral syphilis cure the diabetes which was its chief symptom.

In gouty diabetes the basal treatment should be that of gout. The diabetes may yield entirely, but is very apt to recur, and finally to require diabetic treatment.

In idiopathic or true diabetes the chief reliance of the therapeutist must be upon the removal of the vegetable carbo-hydrates from the food; neither sugar, starch, nor food containing them is to be allowed. Saccharin may be employed as a harmless substitute for sugar, but unfortunately it is almost impossible to still the natural craving for starchy food. The so-called gluten and washed bran always contain starch, and the ordinary commercial gluten contains a large quantity of starch; nevertheless, bread made of these substances is much less harmful than that made out of flour from which no attempt to remove the starch has been made; it is, however, less satisfying, and in some cases it is better to allow a crust of bread to be taken. As further substitutes for bread, cakes of an almond flour from which the sugar has been removed, and also bread or cakes of inulin, may be used. The following diet-list, based upon that made by Austin Flint, indicates the allowable articles of food:

Breakfast.—Oysters or clams stewed, without flour; beefsteak, beefsteak with fried onions, broiled chicken, mutton-chops or lamb-chops, kidneys broiled, stewed, or devilled; tripe, pigs' feet, game, ham, bacon, devilled turkey or chicken, sausage, corned beef hash without potato, minced beef, turkey, chicken, or game; all kinds of fish, fish-roe, fish-balls without potato; eggs cooked in any way except with flour or sugar, scrambled eggs with chipped smoked beef, picked salt codfish with eggs; omelets plain or with ham, with smoked beef, or with kidneys; asparagus-points, fine herbs, parsley, truffles or mushrooms, radishes, cucumbers, water-cress; butter, pot-cheese; tea or coffee with a little cream but no sugar (glycerin or saccharin may be used instead of sugar if desired); light red wine for those who are in the habit of taking wine at breakfast.

Lunch or *Tea.*—Oysters or clams cooked in any way except with flour; chicken, lobster, or any kind of salad except potato; fish of all kinds; chops, steaks, ham, tongue, eggs, crabs, or any kind of meat; head-cheese; red wine, dry sherry, or Bass's ale.

Dinner.—Raw oysters or clams.

Soups.—Consommé of beef, of veal, of chicken, or of turtle; consommé with asparagus-points; okra, ox-tail, turtle, oyster, or clam, without flour; chowder without potatoes; mock-turtle, mullagatawny, tomato, gumbo fillet.

Fish.—All kinds of fish, lobsters, oysters, clams, terrapin, shrimps, crawfish, hard-shell crabs, soft-shell crabs. (No sauces containing flour.)

Relishes.—Pickles, radishes, celery, sardines, anchovies, olives.

Meats.—All kinds of meat cooked in any way except with flour; all kinds of poultry without dressings containing bread or flour; calf's head; kidneys, sweetbreads, lamb-fries, ham, tongue; all kinds of game; veal, fowl, sweetbreads, etc., with curry, but not thickened with flour. (No liver.)

Vegetables.—Truffles, lettuce, chiccory, endive, cucumbers, spinach, sorrel, beet-tops, cauliflower, cabbage, Brussels sprouts, dandelions, tomatoes, radishes, oyster-plant, celery, onions, string beans, water-cress, asparagus, artichoke, parsley, mushrooms, and all kinds of herbs.

Sweets.—Brandied peaches and other fruits; omelets and calf's-foot jelly, sweetened, if desired, with saccharin.

Miscellaneous.—Butter, cheese of all kinds, eggs cooked in all ways except with flour or sugar, sauces without sugar or flour, almonds, hazelnuts, walnuts, cocoanuts, tea or coffee with a little cream but without sugar.

In bad cases of diabetes the absolute skim-milk, buttermilk, or koumiss diet, which has been strongly recommended by Donkin and by Tyson, may be tried: it is asserted that milk-sugar, as well as mannite and levulose or fruit-sugar, is incapable of conversion in the system into true sugar. When, however, milk or any substance containing levulose is given, the effect of the food upon the sugar elimination should be carefully watched.

The diabetic patient should always be warmly dressed, should be very carefully protected against exposure, and should sedulously avoid excessive mental or physical labor; whilst by means of frequent bathing the skin should be kept thoroughly active. Certain mineral springs, especially Vichy and Carlsbad, are much resorted to by diabetics, but are probably chiefly of service in the gouty forms of the disease and where there is evidently habitual engorgement of the liver and other viscera. Very often, however, alkaline waters are useful as adjuvants.

Strychnine, laxatives, cod-liver oil, and other remedies may be useful to meet symptoms as they arise. Arsenic, especially the arsenite of bromine (one-sixteenth of a grain a day increased to one-sixth of a grain in ascending doses), antipyrin (especially in neurotic cases), potassium bromide, and numerous other remedies have been in vogue, but rarely exert a distinct influence for good. Jambul has undoubtedly the power to check the action of diastase upon starch, and has been used in India in diabetes almost from time immemorial: we have seen the sugar disappear from the urine under its administration, but think it will generally be found to fail. The fluid extract should be used in a commencing dose of ten minims in capsule, three times a day, increased to forty or fifty minims. Extract of ergot has been largely used and lauded; if given at all it should be in full dose, one or two drachms a day in capsule. Opium seems to be the only remedy which is capable of distinctly checking the progress of the disease in most cases. It may be used in the form of the extract, or of morphine, or of codeine. The codeine is, we believe, the least successful of the opiate preparations; as, however, it produces much less disturbance of the general system than does the opium, it may be tried first; half a grain should be given three times a day and rapidly increased until thirty to forty grains a day are taken. Opium is better than morphine, and the extract is preferable to the opium itself; one-quarter of a grain of the extract should be given three times a day at first and slowly increased until ten or even more grains are taken daily. At no time should sufficient of the opium be given to produce narcosis. Preparations of pancreas have been used, in accordance with the pancreatic theory of the disease, but have not been sufficiently tested to warrant any definite conclusion; thus far the success does not seem to have been brilliant. The glycolytic ferment isolated by Lépine from the pancreas and from malt diastase is also worthy of further trial.

Diabetic coma is almost hopeless. Intravenous or subcutaneous injections of a three per cent. solution of sodium bicarbonate have been practised, but their use seems warranted only by the therapeutic despair which surrounds the case.

DIABETES INSIPIDUS.

Definition.—A chronic disease characterized by the excretion of a large quantity of urine which contains no sugar.

Etiology.—Diabetes insipidus is a disease more often found in males than in females; it is more frequent in the first half of life, and may occur even in young children. Heredity at times seems to be of importance in the etiology. Claude Bernard found that injury to the floor of the fourth ventricle near the seat of the sugar-puncture, and also section of the splanchnic nerves, produced temporary polyuria. Peyrani has caused polyuria by irritation of the cervical sympathetic, and Kahler has induced permanent polyuria in rabbits by injury to the cerebellum and medulla oblongata. The importance of affections of the nervous system in the etiology of diabetes insipidus is further shown by the occurrence of this affection after injury to the cranium, sunstroke, inflammation of the brain and its membranes, cerebral tumors and syphilis, myelitis, and fright. Kahler observed that these affections of the brain when focal were more frequently seated in the posterior cranial fossa. Diabetes insipidus has also developed in acute infectious diseases, in scurvy, in saccharine diabetes after the disappearance of the sugar, and as a result of excessive drinking. It has been noted in connection with abdominal tumors, especially when in the vicinity of the cœliac plexus, and as an accompaniment of chronic inflammatory processes in the same region.

MORBID ANATOMY.—The above-mentioned lesions of the nervous system and of the abdomen should be sought for, and, if found, as suggested by the results of experiments, offer a probable explanation of the disease. The kidneys are large, and in certain cases the pelves and ureters are dilated and the bladder is hypertrophied.

SYMPTOMS.—Polyuria is the characteristic symptom. It may be of rapid origin or may gradually and progressively develop. The daily quantity of urine excreted may be from one to two gallons and upward. The urine is pale and colorless, acid, the specific gravity usually below 1005. It is free from albumin, and contains no sediment. Inosite has sometimes been found, and the temporary occurrence of glucose has been observed. The absolute quantity of the solid constituents of the urine is generally unaltered. In certain cases or at certain times an increase of urea has been found, a condition to which the term *azoturia* has been applied.

Excessive thirst, polydipsia, is the usual accompaniment of the polyuria. In the mild cases there may be no further disturbance. The presence of headache, vertigo, mental excitability or depression, and neuralgia indicates a transition to the severer type, which is further manifested by hebetude, loss of flesh and strength, dryness of the skin, and weakness of the pulse.

DIAGNOSIS.—The diagnosis is based upon the occurrence of polyuria and the examination of the urine. Hysterical polyuria is spasmodic and not persistent, and therefore to be differentiated. The polyuria of chronic fibrous nephritis is to be excluded by the absence of albumin and casts, and the freedom from evidence of hypertrophy of the heart. In hydronephrosis, especially the intermittent variety, large quantities of urine may be occasionally voided. This affection, however, is to be excluded by the absence of a tumor diminishing in size with the abundant flow of urine ; the latter is either of relatively normal composition or presents the characteristics of the urine of fibrous nephritis.

PROGNOSIS.—In some cases diabetes insipidus continues for years, perhaps throughout life, without serious disturbance. Death in fatal cases is rather attributable to the cause or to complications than to the disease directly. Recovery is rare unless the exciting cause—*e.g.*, syphilis—is remediable, although temporary improvement may take place.

TREATMENT.—In the treatment of diabetes insipidus it is essential to obtain the best hygienic surroundings for the patient, to forbid excessive mental or physical work, and in every way possible to maintain the general health. The diet should be nutritious, but not special; the clothing should be warm, so as to keep the skin protected and active, and the thirst should not be fully satisfied. Extract of ergot (ten grains in capsule, three to six times a day), valerian (one-half to one fluidounce daily), and zinc valerianate (one to two grains in capsule, three to four times a day), are the most effective remedies, although the salicylates, arsenic, antipyrin and other coal-tar products, the bromides, belladonna, strychnine, and numerous other remedies, have been from time to time commended.

In giving any of these remedies the dose should be increased until some therapeutic or physiological effect is produced.

CHAPTER III.

INFECTIOUS DISEASES.

SCARLATINA. SCARLET FEVER.

DEFINITION.—A contagious febrile disease, characterized by a peculiar diffused eruption and a pronounced tendency to the development of serious sore throat.

ETIOLOGY.—Scarlet fever is probably always the result of a contagion, which may pass directly by contact with the person of the sick or be transmitted through the air, or be carried by fomites. The power of the poison to resist change is very great, as is also its ability to pass into milk and other articles of food, and to adhere to letters and other media of transmission. Various investigators during late decades have attempted to connect certain diseases of cows and of other lower animals with scarlet fever of man, but as yet there is no sufficient reason for believing that scarlet fever can be transmitted from man to the lower animals, although cats may act as carriers of the poison.

Although various pathogenic germs have been isolated from patients suffering from scarlet fever, the true nature of the contagion remains unknown. Streptococci, diplococci, micrococci, bacilli, and other organisms have, it is true, been found, but all appear to be the result of secondary infection. It is important to note that the diphtherial organism can often be obtained in abundance from the pseudo-membranous angina of scarlet fever; but, on the other hand, the most violent sore throat with abundant exudate may exist without the diphtheritic bacillus.

MORBID ANATOMY.—The anatomical changes in fatal cases of scarlet fever are most frequent in the throat and kidneys. The mucous membranes of the pharynx, soft palate, and tonsils are swollen, injected, perhaps hemorrhagic. There may be an adherent false membrane due to the presence of bacteria, sometimes of the diphtheria bacillus. Superficial ulceration and deep necrosis of the tissues are not infrequent. In some instances the neighboring lymphatic glands are swollen and injected and may contain abscesses, while the surrounding fibrous tissue is œdematous.

The kidneys are likely to show the characteristics of an acute nephritis in a mild or a severe form. The kidney is usually enlarged, the region of the convoluted tubes abnormally opaque. It may be injected, sometimes containing hemorrhagic spots beneath the capsule and on section, or it may be abnormally pale. The alterations of the kidney especially characteristic of scarlet fever are those which were first described by Klebs and Friedländer and designated as a glomerulo-nephritis. The changes consist in enlargement of the glomeruli and a thickening of the capillary wall, an increase of the nuclei, and a swelling and necrosis of the capsular epithelium. Collections of leukocytes in the interstitial tissue are to be found in various parts of the kidney. The diseased Malpighian bodies appear to the naked eye as pale gray translucent points.

Granular degeneration of the heart and liver and moderate acute enlargement of the spleen are present. The rash leaves no gross evidence of its presence in the skin except in hemorrhagic cases, when punctate hemorrhage is to be found. Hemorrhage may also take place in the mucous membrane of the intestine. Pseudo-membranous patches are sometimes to be found in the stomach and intestine. The lymphatic glands of the body may be enlarged, and Wagner has described the presence of a disseminated formation of lymphadenoid tissue in the liver and kidneys.

SYMPTOMATOLOGY.—The ordinary period of incubation of scarlet fever is from three to five days, though well authenticated and carefully studied cases have been reported by Trousseau and others in which it was twenty-four hours, whilst it may be prolonged to ten or even twelve days. The invasion of the disease is usually abrupt; in the mildest cases it is marked by nausea and chilliness, in the ordinary cases by one or more chills, and by vomiting, which is apt to be repeated and severe. The pulse becomes at once rapid and small (120 to 150), and the axillary temperature rises to 103°, or it may be 105° or 106° F. There is great dryness of the skin, mouth, and throat, whilst the tongue is covered with grayish fur, and the fauces are distinctly red. Conjunctival, nasal, or bronchial catarrh is very exceptional. In from twelve to thirty-six hours the characteristic eruption appears, in most cases first upon the upper chest and the back, but sometimes upon the extremities, and in exceptional cases upon the cheeks. The spreading of the eruption is so rapid that usually in twelve hours it covers the whole body. It consists of a scarlet or deep-red punctated or stippled efflorescence; sometimes it is minutely papular. Under pressure with the finger the color disappears, but reappears immediately upon removal of the pressure. Ordinarily the rash is nearly uniform, but it may be in patches. It is especially dark colored in the groin and in the folds of the skin made by flexion of the extremities. On the nose, lips, and chin it is often wanting, whilst it is always very pronounced upon the cheeks. When it is fully developed it is sometimes accompanied by an eruption of miliary vesicles, although the skin is very dry and pungent. The eruption also attacks the mucous membranes, so that the cheeks and the throat are brilliant red, swollen, and often distinctly punctated. The tongue, though red on the tip and edges, is covered with a whitish fur from which project the red papillæ (strawberry tongue). A few days later desquamation leaves the surface of the tongue red and rough, with greatly enlarged, very dark red papillæ (raspberry tongue); a condition which may last as long as five days. At this stage the tonsils are swollen, with their crypts distended with a yellowish-white creamy exudate, which often spreads over the surface to make a sort of false membrane. The contention of Lasègue, that there is a vesicular eruption upon the mucous membrane at this time, is plausible. The submaxillary glands and the surrounding cellular tissues are always swollen.

The coming out of the eruption is not followed by any immediate remission of the general symptoms; the fever remains high, the pulse rapid, headache is often extreme, and there is very commonly a nervous agitation which may rise to distinct delirium. There are extreme thirst, complete anorexia, more or less constipation; but vomiting is rare after the second day. The urine is, throughout the attack, of high specific gravity, dark-colored, and very commonly contains a trace of albumin as early as the second day of the disease. In favorable cases there may be a gradual abatement of the symptoms after the second or third day of the eruption; often, however, the constitutional disturbances do not subside until the sixth or seventh day, when desquamation has fully begun.

The characteristic course of the fever in scarlet fever is a sudden rise of temperature, with a maximum reached in the first twenty-four or forty-eight hours, followed by a continuous fever, with mild morning remissions and a general tendency to decrease slowly during the next five or six days, at the end of which time there is a rapid but not abrupt defervescence. Sometimes the temperature drops and the rash fades on the second or third day, both to reappear in a day or two. The quickening of the pulse and that of the respiration in most cases correspond to the rise of temperature, upon which they probably are largely dependent.

Although the protective power of an attack of scarlet fever cannot be gainsaid, it is certain that, especially in susceptible individuals, there may be repeated attacks, which may consist simply of a bad sore throat with some febrile reaction. Between such attacks (the mildest possible) and those in which the patient passes immediately into collapse and dies in a few hours overwhelmed by the poison, there is every grade or variety of scarlatina; but, in obedience to custom, three types may be recognized, —the simple, the anginose, and the malignant.

The simple scarlet fever is that which has already been described in the text. In the anginose scarlet fever the throat symptoms appear very early, and are attended with great swelling, and with the rapid formation of a membranous exudate which may extend upward into the nostrils, forward into the mouth, and downward into the pharynx and larynx. The excessive fetor, the rapid swelling of the glands of the neck, and the tendency to necrosis of the mucous membrane may make a picture indistinguishable from that of malignant diphtheria, and death may result from a septicæmia produced by the local disease of the throat, or the ulcers may open the carotids or other blood-vessels and cause fatal hemorrhage. Inflammations of the Eustachian tube and of the middle ear are common phenomena.

In malignant scarlet fever violent headache, vomiting, dyspnœa, cyanosis, convulsions, delirium, coma, and intense fever may end in the course of a few hours, without eruption, in death; or the attack may at first seem not overwhelming, but be followed in a few hours by violent adynamia, with great heart-failure, weakness of the extremities, excessive dyspnœa, and nervous disturbance; sometimes the malignant symptoms first develop after the appearance of the eruption, which may be intense and wide-spread. In most cases of malignant scarlet fever vomiting is pronounced, and not rarely there is diarrhœa. A sudden rise of temperature immediately preceding death is also frequent, even at a time when the extremities are very cold and the patient in collapse. In the hemorrhagic malignant scarlet fever epistaxis and abundant hæmaturia may precede or follow the occurrence of the purpuric and petechial eruption, and death may take place almost immediately in collapse, or be preceded by intense fever, violent dyspnœa, convulsions, and delirium. A rare form of malignant scarlet fever is that in which all the symptoms are lost in a furious choleraic diarrhœa.

Relapses in scarlet fever are rare, but do occur, with the reappearance of the fever, the sore throat, and the eruption. The time of their reappearance is from twelve to thirty-six days after the first attack.

COMPLICATIONS.—The complications of scarlet fever are often very serious. True diphtheria may develop, or there may be a wide-spread gangrene of the throat without diphtheria. Severe adenopathies are very common, especially in the submaxillary and sterno-mastoid region, and may end in suppuration during the height of the attack, or more frequently during the early days of convalescence, with a resultant severe or even fatal septicæmia. In some cases of scarlatina the enlarged lymphatic glands harden into a brawny mass, exceedingly intractable to all medical treatment. Middle-ear inflammation occurs probably in about thirty per cent. of the cases, and according to Burckhart-Merian a severe suppurative otitis media develops in about four and a half per cent., usually during the period of eruption, revealing itself by violent earache, insomnia, and excessive tenderness of the mastoid processes.

Among the complications or sequelæ of scarlet fever should be mentioned multiple abscesses, pyothorax, suppurative pericarditis, endocarditis, and certain arthropathies which have been incorrectly considered as rheumatic. Of *scarlatinal rheumatism*, so called, there are three forms: that in which the exudate is serous, that in which it is primarily serous and secondarily purulent, and that in which pus is formed from the beginning. The affection generally begins from the fifth to the seventh day of the fever, or rarely during the stage of desquamation. It sometimes attacks many joints, but is usually localized in a single articulation. Recovery in the course of a few days is common with the serous exudate;

recovery with more or less permanent change in the joint is the rule when the exudate is first serous and then purulent; but when from the beginning pus forms in the joints, death from pyæmia is the common result. It is probable that these complications are due to streptococcus poison from the throat.

The nervous sequelæ of scarlet fever are usually not severe, but chorea, hemiplegia, mania, and melancholia have all been reported, and probably multiple neuritis is more frequent than is generally believed.

Of all the complications of the disease nephritis is the most important. Not rarely, even in mild scarlet fever, when the temperature rises very high, albumin and even casts appear in the urine during the first twenty-four hours: they are the result of the hyperthermia, and have no greater significance than in various other febrile diseases, passing off without serious result. The characteristic nephritis of the disease develops most frequently in the second or third week, but may be delayed to the fourth or even sixth week. It may follow the mildest form of the disease, and come on when all the symptoms seem most favorable. The first evidence is usually an anasarca just below the eyes, which often is first detected in the early morning. The nephritis varies greatly in intensity; in the severest cases there are aching pains in the back, chills, vomiting, hæmaturia, and a partial or even finally complete suppression of the urine, with uræmic symptoms after some hours. Furthest removed from these cases are those in which the symptoms are so mild that they can scarcely be noted,—a little albuminuria, a few casts, some œdema. Between the two extremes may be found every variety of severity. Among the subacute cases are many in which without great care the symptoms, at first so slight as to be easily overlooked, progress to great seriousness, with profound alteration of the kidney. As œdema about the eyes may occur without the appearance of albumin in the urine, its presence is not a proof of nephritis. Again, there is much reason for believing that a dangerous nephritis may exist and yet the urine be temporarily free from albumin, so that repeated examinations are necessary for the detection of the kidney disease. Œdema of the lungs and acute œdema of the glottis are more frequent in severe than in mild cases of scarlatinal nephritis, but may suddenly appear in any case.

DIAGNOSIS.—The diagnosis of scarlet fever in the stage of incubation depends upon the severity of the symptoms, the presence of vomiting, and the rapid rise of temperature. In malignant cases without the development of the rash the diagnosis must be made by exclusion, aided by the history of exposure to cause. The rashes produced by antipyrin, belladonna, oil of copaiba, and some other drugs resemble somewhat the rash of scarlet fever, but the absence of fever and of sore throat, with the presence of other symptoms of poisoning, usually makes the diagnosis easy. Acute exfoliative dermatitis may develop without obvious cause, and, as it has a sudden onset, a brilliant exanthem closely resembling that of scarlet fever, and distinct fever with nervous phenomena, it may at first be impossible to distinguish it from scarlatina. The absence of throat symptoms and of the peculiarities of the tongue of scarlet fever, and the fact that the desquamation affects the hair and the nails, usually make the final recognition easy.

The diagnosis between diphtheria and scarlet fever is not always possible, because undoubtedly diphtheria is sometimes accompanied by a scarlatinal rash, whilst Loeffler's bacillus may be present in scarlet fever. In other words, a pure diphtheria may closely simulate a scarlet fever, and diphtheria frequently coexists with a scarlet fever, so that at times the most acute practitioner cannot unravel a case sufficiently to know whether he has one simply of diphtheria or one of mixed infection. In some cases a history of exposure may greatly aid in the diagnosis. Fortunately, so far as treatment is concerned, the diagnosis is of little practical importance, as the treatment of the scarlet fever complicated with diphtheria would be precisely that of a diphtheria simulating scarlet fever.

Prognosis.—The mortality in scarlet fever varies in different epidemics and under different circumstances from one to forty per cent. It is greater in hospital practice than in civil life, among the poor than among the rich. In children under one year the death-rate is very high, but it diminishes after the first year until it reaches its minimum between six and twelve years of age. Any previous disease or diathesis greatly increases the danger. The prognosis is grave in proportion to the severity of the early symptoms; high fever, great adynamia, restlessness, intensity of the sore throat, any of these occurring early are very serious, as are also extreme rapidity of the pulse and elevation of the temperature. A precocious painful swelling of the submaxillary gland is of evil import. The majority of cases of nephritis recover under careful treatment, but a complete early suppression of urine is very dangerous.

Treatment.—Absolute isolation in a fully ventilated room, with all the precautions as to bedclothing, etc., which are especially described in the article on typhoid fever, are essential in the treatment of scarlet fever. The activity of the contagion and the seriousness of the disease make it imperative that the physician should carefully supervise the disinfection of the sick-room.

Although belladonna, mercurials, salicylic acid, and various other drugs have been recommended as specifics, there is no remedy which has any power to affect the course of the fever. The symptoms must be met as they arise. When the vomiting is severe, carbonic acid water, lime water and milk, and bismuth, will often be found effectual. If these fail, a quarter of a grain of cocaine, in solution, every one or two hours, may be tried for a few doses. For the relief of nervousness and insomnia the bromides, trional or sulphonal, and chloral, used very carefully, are of value. Hyoscine will frequently control the delirium and produce sleep, but is an extremely dangerous remedy, as by increasing the dryness of the throat and probably also by producing paralytic weakness of the throat it tends greatly to increase symptoms of suffocation in anginose cases: we have seen it apparently cause death in this way. In order to maintain the secretions, and especially to lessen the strain upon the kidneys, the child should be encouraged to drink cold, simple, or carbonated water very freely.

Antipyretic treatment is in most cases essential. Phenacetin, antipyrin, and antifebrin will reduce the temperature, but certainly grave danger accompanies the free use of any of these remedies; although, on the other hand, small doses given at regular intervals may do great good by quieting the nervous disturbance and helping in the reduction of temperature. Quinine has been strongly recommended as an antipyretic by practitioners, but to have a distinct effect must be given in large doses, —a practice which, in our opinion, is not justifiable. On the other hand, when given in moderate dose it probably is of service in tending to reduce temperature, as well as by supporting the nerve-centres. The bisulphate should always be employed, as more easy of absorption, and if it irritate the stomach it should be given by the rectum, not in suppositories, but in slightly acidulated (tartaric acid) solution.

As the fever of scarlatina does not last over a few days, a temperature of 102.5° F. does very little harm. When, however, it rises to 103° F. or above, cold should be used externally, first by sponging, and, if this fail, by packing or by bathing. The severity of the baths must be proportionate to the resistance of the fever; probably in most cases the bath at a temperature of 85° F. gradually reduced to 80° or 75° F. is the best. Leiter's tubes applied to the head and to the abdomen, with iced water run through them, sometimes suffice. The cold pack, or the bath, or whatever means is employed, must be used until the desired effect is produced, and be repeated whenever the temperature rises to 103° F. If there be a tendency to relapse, alcoholic stimulants should be given freely just before the patient is put into the bath; and hot-water bottles or bags may sometimes be advantageously applied to the extremities whilst the patient is in the bath.

The treatment of adynamia in scarlet fever is similar to that of exhaustion from other fevers. (See Typhoid Fever.) As, however, there is a special tendency to irritation of the stomach and of the kidneys, ammonium carbonate and other irritating drugs must be avoided.

Through the whole course of the disease the throat must be very carefully treated. The local external use of ice by india-rubber bags fastened around the neck underneath the jaws is often advantageous, whilst small pieces of ice may be allowed constantly to melt in the mouth. Potassium chlorate has been very largely used in scarlet fever, and is sometimes of service as a local remedy to the throat; except for its local action it is of no value whatever, and it has without doubt aided in numerous cases in causing death by irritating the kidneys and increasing the danger of nephritis. Tincture of ferric chloride, solution of silver nitrate, glycerite of tannin, and various other astringent solutions are employed by different practitioners as local applications. Spraying the throat out, however, with a peroxide of hydrogen solution has seemed to us the best of all local treatment. (See Diphtheria.) The official preparation may be used of the full strength, or diluted one-half. When there is a tendency to closing of the nostrils, the spray should be thrown into the nostrils, if possible, from behind; if not, from the front.

If suppuration of a gland occurs, a free incision should be at once made. In order to allay the burning and itching of the skin, cosmoline, cacao butter, cold cream, lard freed from salt by washing, olive oil, or other bland fat should be freely applied to the surface of the body morning and evening, after the first or second day of the eruption. When the eruption retrocedes or fails to develop, the hot mustard bath, or, if there be high temperature, the cold mustard bath, will often be of service. The treatment of nephritis is that of acute nephritis from other cause. (See Acute Desquamative Nephritis.)

RUBEOLA. MEASLES.

DEFINITION.—A contagious eruptive fever, characterized by the early and severe development of catarrhal symptoms, and the appearance on the third or fourth day of a peculiar eruption, which may be vesicular, but is usually composed of very minute papules, arranged in irregular, more or less crescentic patches.

ETIOLOGY.—The cause of measles is always a contagion, whose nature has not yet been fully established. In searching for the supposed germ of the disease bacteriologists have found in various parts of the body streptococci, micrococci, bacilli, and other micro-organisms, which are without doubt due to secondary infection. The same appears to be true of the diplococcus obtained by Cornil and Babés, which is probably identical with the well-known pneumococcus of pneumonia. In 1892 Canon and Pfeiffer discovered an organism in the blood as well as in the catarrhal secretions of patients suffering from measles, which is believed by some to be the specific germ of the disease. This organism is remarkable for its variations in size and shape; it exists both as a diplococcus and as a bacillus as long as the diameter of a red blood-corpuscle.

Although measles is most frequent in children, yet adults unprotected by a previous attack readily take the disease on exposure to the contagion, and both sexes are equally affected. One attack of measles affords a very decided but not completely perfect protection.

MORBID ANATOMY.—In fatal cases of measles death is usually the result of complications, especially in the respiratory tract. These are present as foci of broncho-pneumonia or of lobular pneumonia, with more or less extensive patches of atelectasis from obstruction of the bronchi by catarrhal secretion. A persistence of such inflammatory conditions in the lungs is not infrequent in virtue of their infection with tubercle bacilli, and death may eventually result from pulmonary tuberculosis.

SYMPTOMATOLOGY.—The period of incubation is about ten days, at the end of which time an abrupt rise of temperature, to 102° or 103° F. the first day, with or without chill, occurs, and the characteristic catarrhal symptoms appear. The conjunctivæ become red and watery, there is frequent sneezing, with excessive nasal secretion, and not rarely epistaxis, laryngitis, tracheitis, and even a mild bronchitis, rapidly develop. Of these catarrhal symptoms those concerned with the eyes and the nose are most pronounced, but even at this time the least exposure is liable to produce severe bronchitis or pneumonia. The throat is sometimes a little sore, but never as it is in scarlet fever. In many cases during the stage of invasion the hard and soft palates and the throat itself are very red and covered with minute spots or points, which are sometimes spoken of as an eruption upon the mucous membrane. For the next three or four days there are headache, malaise, pronounced fever, anorexia, and not rarely vomiting or diarrhœa. Severe nervous symptoms are rare, though in very bad cases delirium and, especially in young children, convulsions may occur. In from three to five days the beginning of the second stage of the disease is marked by the development of the eruption, which almost invariably appears first upon the cheeks and forehead and around the mouth, and spreads rapidly downward over the rest of the body. It consists first of minute papules, which are surrounded by a pale-red, slightly elevated border and become confluent. When fully developed the eruption consists of dark-red macules, which by the finger can be found to be slightly elevated, and are of irregular shape and size, more or less crescentic, dentated, and often fantastically arranged in festoons or column-like groups. Under the pressure of the finger they lose their color at once, but regain it immediately upon removal of the pressure. With the appearance of the exanthem the fever usually increases, and the catarrhal symptoms become more manifest. In from thirty-six to forty-eight hours, however, in favorable cases all the symptoms begin to decline, and in from three to six days the fever has disappeared, desquamation has commenced upon the face, and a rapid convalescence has been entered upon.

In some cases the eruption of measles is very small and remains discrete, whilst in others the patches flow together so as to make a uniform covering of the surface, which resembles somewhat the rash of scarlet fever. Very rarely the measle rash becomes vesicular.

The departure of measles from the ordinary type is best studied under the heads of mild and malignant cases. Of the benign varieties the most remarkable is that in which all the symptoms of the disease are present except the eruption, producing a disease-picture whose true nature can be recognized only by knowledge that the subject has been exposed to the contagion of measles. In a similar way there would appear to be certain cases of measles in which the catarrhal symptoms are altogether wanting. There is also an abortive form of the disease in which the eruption appears with the ordinary symptoms, but fades immediately, with a rapid abatement of the fever, and a well-developed convalescence by the fifth or sixth day of the disease. These cases can be at once differentiated from those in which there is a sudden retrocession of the eruption by the immediate abatement of the constitutional symptoms.

Among the malignant forms of measles may be placed those cases in which the disease develops during the course of some other serious illness, as a tuberculosis, or even an acute disorder, like typhoid fever or diphtheria. Under these circumstances the course of the measles is very frequently irregular, the eruption imperfectly developed, the fever high, and the complications excessive. A form of measles which has been especially seen in the army, and in children's asylums, is that in which from the beginning there is violent dyspnœa with marked cyanosis, and usually rapid death from asphyxia. In many of these cases examination will reveal the fine disseminated râles of a capillary bronchitis, but sometimes the only departure from the norm to be made out is extreme feebleness of the respiratory movements. It is to this variety of measles that the name of *epidemic capillary bronchitis* has been given.

In the ataxic or adynamic form of measles the severe symptoms usually develop at the time of the appearance of the eruption. The pulse becomes very rapid, the respiration exceedingly hurried, the temperature rises to 104° or 105° F., and the dry tongue, typhoid face, great muscular prostration, and other symptoms of the typhoid state rapidly develop. In young children repeated convulsions are frequent and often end in coma. In adults, delirium, mild and muttering or fierce and maniacal, comes on. Death in such cases may occur in three or four days; or with the development of natural sleep and a great increase in the secretion of the urine the violence of the symptoms may abate. In some of these cases there is a sudden disappearance of the eruption, with a great increase of the symptoms. A rare form of measles is that known as "black measles," with hemorrhage under the skin and into the mucous membranes, terminating in death in two or three days.

COMPLICATIONS.—In measles as ordinarily seen the complications are very often much more serious and important than the original disease. The most frequent of them are connected with catarrhal irritation of the respiratory mucous membrane. Violent nasal catarrh may give rise to a serious otitis media; laryngitis may be followed by so much swelling as to produce symptoms of laryngeal obstruction; whilst actual membranous exudation is not very rare in the throat and larynx, and may be associated with the diphtheria bacillus. Bronchitis is almost universal, and is especially prone to pass into the small tubes and produce a capillary bronchitis followed by the formation of small infiltrated patches through the lungs, which by their confluence may produce wide-spread lobular pneumonia. Broncho-pneumonia also occurs. The pulmonary complications may develop at any period of the disease, but are more frequent and severe during the stage of eruption and also during convalescence. They are usually marked by increased fever, rapid respiration, and dyspnœa. Fine sibilant or more commonly fine moist râles may be heard upon auscultation through the whole chest, but a very severe disseminated lobular pneumonia may exist without dulness of percussion or without alteration of the breath- or the voice-sounds: only when the patches are confluent do these physical signs of consolidation become apparent. When severe pulmonary complications occur in young children the dyspnœa is extreme and convulsions are not rare; death from suffocation may occur during the second or third day.

In healthy subjects the conjunctivitis rarely ends in suppuration or serious trouble, but in delicate children suppurative conjunctivitis, diffused purulent keratitis, and ulceration of the cornea are especially common.

Tubercular disease often develops during the convalescence of measles. The resistive power of the system seems to be lowered out of proportion to the severity of the original disease, whilst the various catarrhal inflammations afford an excellent nidus for the bacillus.

DIAGNOSIS.—During the period of invasion the catarrhal symptoms separate measles from the ordinary eruptive fevers, but cause the attack to resemble very closely one of epidemic influenza. The most important difference in the affections is that in grippe the temperature is apt to reach its maximum in the first few hours, whilst in measles it mounts steadily for two or three days. The eruption of measles is sometimes simulated by the rashes produced by copaiba and other drugs, but under such circumstances the catarrhal symptoms are wanting, and fever, if present, takes a diverse course. (See also Rötheln and Small-pox.)

PROGNOSIS.—In healthy children, properly taken care of, measles is ordinarily a very trivial affection, almost free from danger to life. On the other hand, in some epidemics it has almost rivalled the plague in its destructiveness, fifty or even seventy per cent. of the cases ending in death. The mortality is excessive between the ages of one and four. In civil life, among adults well taken care of, the mortality should not be more than three or four per cent.; in armies and crowded prisons it may rise, as at the siege of Paris, to thirty-seven per cent. Race characteristics are of importance. Death is much more frequent among negroes than among whites; whilst the results of the inoculation of semi-barbarous people with the contagion have been frightful. In North America, South Africa, and Oceanica, half the population of a whole district has died in the course of a few weeks.

TREATMENT.—Owing to the great tendency to catarrh, the mildest case of measles should be put to bed and kept there until convalescence is established, and the greatest care should be subsequently exercised for some weeks until desquamation is completely over; exposure may cause death. The sick-room should be somewhat darkened so long as there is any photophobia, and should be well ventilated, but free from draughts. The conjunctiva should be washed once, twice, or oftener a day, according to the severity of the symptoms, with saturated solution of boric acid; whilst borax, potassium chlorate, and other local remedies should be used for mucous inflammation in the nose, mouth, and fauces. The diet should be light but nutritious, consisting of milk and milk purées, animal broths, and, as convalescence comes on, milk-toast, oysters, sweetbreads, chicken, and other light solid foods.

In the ordinary case of measles the only medical treatment required is the administration of moderate doses of potassium citrate, which has a tendency to favor perspiration, increase the secretion of urine, and favorably affect the bronchial inflammation. If constipation exist, it should be immediately relieved. Diarrhœa should not be interfered with so long as it is slight; if it be excessive, bismuth and carbolic acid, or other local remedies, may be used. If the eruption be delayed or if it retrocede, the patient should be put at once in a hot bath or a hot mustard bath (two teaspoonfuls to the gallon), or the hot mustard foot-bath should be used. A sudden rise of temperature, or even a very high temperature gradually attained, almost invariably indicates the coming on of bronchial or pneumonic irritation, and calls therefore for the employment of counter-irritation and the appropriate internal remedies. A temporary elevation of temperature does no harm, but, as any continuance of a severe pyrexia (103° F.) is dangerous, it must be met by the use of external cold or of antipyretics. Of the antipyretics phenacetin is probably the safest, next after it antipyrin. In no case should very large doses of these remedies be used. It is safer to reduce the temperature by means of the bath of the temperature of 90° F., which if essential may be further cooled, even as low as 80°. As the pyrexia is very seldom urgent, the bath should be used not only cautiously but also with slow increase of power, so that no greater application of external cold be made than is absolutely essential. After removal from the bath the patient should be rapidly dried, and if there be any failure of vitality whiskey should be given.

The treatment of the pulmonic complications of measles does not differ from that of similar condition of the lungs arising from other cause. Sedative remedies are, however, borne very badly, and stimulants are commonly necessary from the first. Small doses of ipecac may be given with potassium citrate, but stimulant expectorants, such as ammonium chloride, terebene, and oil of eucalyptus, are soon demanded. Extract of ergot is sometimes valuable in reducing the congestion; and the free use of hot poultices over the chest is of the utmost value. In malignant measles free stimulation should be employed from the onset, by means of whiskey or brandy, raw beef juice, and meat essences.

RÖTHELN. GERMAN MEASLES.

DEFINITION.—A contagious febrile disease, characterized by mild catarrh, a measles-like eruption, and enlargement of the lymphatic glands.

ETIOLOGY.—Rötheln, although long confounded with other exanthematous diseases, is without doubt distinct. It occurs almost universally in epidemics, and is due to a contagion of unknown nature, which is capable of being transferred in fomites and is given off by the subject from the period of invasion to well-advanced convalescence.

SYMPTOMATOLOGY.—The invasion period of rötheln is short, and marked only by slight fever, malaise, nervous disturbances, and some conjunctival catarrh. Even at this time, however, pressure upon the jugular and subauricular lymphatic glands will usually detect tenderness. The eruption is especially prone to develop during the night, and in more or less erratic ways. It may appear first on the face or upon the body, or on the inner side of the arms, etc. It is more or less polymorphous in color and size and form, as well as in dissemination. Upon the trunk, and especially upon places where there is continuous pressure, the spots may become confluent, whilst upon the hands and feet they are usually discrete. The eruption spreads rapidly, reaching commonly its full efflorescence and beginning to fade in from twenty-four to thirty-six hours, and disappearing entirely without desquamation in three days. Its color ranges from a pale rose to a deep red; and, whilst it varies greatly in its minute appearance, there are two typical forms,—one in which the spots are minutely papular, like measles, and one in which they are large, reddish plaques, suggesting scarlet fever.

During the stage of eruption the bodily temperature usually remains below 101° F., but may rise to 103°; the conjunctival and nasal catarrh persist until convalescence, but are rarely so severe as in true measles. The most distinctive feature of the disease is the glandular enlargement. This is most pronounced in the occipital, submaxillary, and carotid glands, and is often very severe. In some cases, however, the only glands apparently affected are those of the extremities or of the trunk, and these may not be visibly swollen, although tender upon pressure. The glandular enlargement does not always disappear with the exanthem, and sometimes may be detected two weeks after the first invasion. When the eruption has not been severe it is often impossible to demonstrate a true desquamation, but a furfuraceous shedding of the epiderm may sometimes be detected.

There is said to be a malignant form of the disease, but usually there are no complications and the cases pass rapidly to recovery.

DIAGNOSIS.—It is affirmed that the eruption in rötheln may exist without the enlargement of the lymphatic glands, and the enlargement of the lymphatic glands without the eruption. In such cases the diagnosis could be fixed only by a knowledge of exposure to the cause of the disease. In our experience the only affection which the disease resembles is measles, from which we believe it is especially separated by the lymphatic enlargement and tenderness, as well as by the mildness of the catarrh and of the general symptoms, and by the polymorphic character of the eruption.

Treatment.—Rarely is other treatment required than simple nursing. Any symptoms which may arise should be met on general principles.

VARICELLA. CHICKEN-POX.

Definition.—A specific contagious fever, occurring chiefly in children, and characterized by the presence of a vesicular eruption.

Etiology.—Various organisms have been isolated from the lymph of the vesicles of chicken-pox, and Bareggi asserts that he has discovered an ovoid micrococcus which exists in the white blood-corpuscles and whose cultures are capable of producing varicella in infants; whilst Pfeiffer has found an amœba-like parasite in the vesicular lymph. Varicella is certainly distinct from all other diseases, and is entirely incapable of protecting from small-pox or other affections. It occurs almost exclusively among children.

Symptomatology.—The period of incubation is from ten to fifteen days, followed by a period of invasion which in most of Steiner's inoculation experiments lasted four days, but which in the natural disease is ordinarily much shorter. The symptoms usually consist simply of a slight fever and malaise, although there are cases in which violent vomiting, delirium, very high temperature, convulsions, and excessive dyspnœa develop to an alarming degree. There is, moreover, no distinct relation between the severity of these primary symptoms and the gravity or prolongation of the whole sickness. The eruption usually first appears upon the trunk, only in rare cases upon the face, and is first a macule, then a transparent vesicle which becomes opaque and finally forms into a crust. The macular stage is so short that it is often overlooked. The vesicles are brilliant, rarely umbilicated, surrounded by a reddish areola, and varying in size from a tenth to a quarter of an inch: especially when scratched by the child, they may leave distinct, ugly scars. Fresh groups of the eruption may appear for several days, so that various stages of the pock coexist side by side. In cachectic cases the varicellar eruption is purpuric, and may be ecchymotic; even gangrenous ulcers sometimes result. The eruption may occur upon mucous membranes, producing a simple or an ulcerated stomatitis, angina, tracheitis, conjunctivitis, or vulvitis. During the eruptive stage there is generally a mild fever. In ordinary varicella severe complications are rare, though nephritis does occasionally occur. In purpuric or malignant varicella various hemorrhages, local gangrene, pneumonia, pleurisy, and abscesses have been noted; whilst severe nephritis is not infrequent.

DIAGNOSIS.—Varicella can usually be distinguished from varioloid without difficulty by the absence of serious prodromic symptoms; by the appearance of the first vesicles upon the trunk; by the absence of the hard, shotty feeling of the papules, or of umbilication in the vesicles; and by the failure finally to develop pustules. The varicella pocks are also more bleb-like, and the areolation around them is not so deep. When the pock of varicella becomes confluent and in some places umbilicated, the diagnosis may for a time be very difficult, especially if there be no distinct history of the earlier stages of the attack. Acute pemphigus, varicelliform syphilides, and certain other skin affections occasionally closely resemble varicella, but can usually be distinguished without difficulty by being apyretic or by the slow development of the vesicles.

PROGNOSIS AND TREATMENT.—Varicella ordinarily requires no further treatment than some restrictions of diet and of exposure. In the rare malignant cases the prognosis may be grave, whilst the treatment should be that of malignant febrile attacks of other nature.

VARIOLA. SMALL-POX.

DEFINITION.—An acute contagious fever, characterized by an eruption whose unit is at first a hard papule, then an umbilicated vesicle, then a pustule, and finally a crust.

ETIOLOGY.—The cause of small-pox is a contagium which is probably an organism. Late in the disorder secondary septic infection is very prone to occur, so that various species of staphylococcus, streptoccocus, and even a saccharomyces, have been found in different portions of the body. The nature of the original virulent organism still remains doubtful. Klebs has described a tetracoccus, whilst Pfeiffer and Van der Loeff affirm that there is a sporozoon. For the transmission of the contagium contact is not necessary; the fact that the crusts, which in China are preserved for the purposes of inoculation, retain their activity for two years, shows how tenacious of life the germ is; and any form of fomites suffices. The contagium chiefly finds entrance into the system through the respiratory organs, and there is much evidence to show great resistive power in the digestive organs. It certainly exists in enormous quantities in the pustules and scabs; but it probably escapes from the body with all the excretions, and is abundantly given off during the stage of invasion before the appearance of the eruption. Legroux and others have reported severe epidemics which originated in cases which died during the prodromic stage. The maximum activity of the contagium is said to be at the time when the pus-formation is most abundant.

Predisposing causes are of little importance in the history of small-pox; it attacks all ages and both sexes. Very few persons, unless protected by previous attacks, are insusceptible to the poison, though there appear to be certain families in which there is a distinct hereditary immunity. Certain races, notably the negroes, seem to be more susceptible than others; but the statements that have been made that other races, such as the Hindoos and the Australians, are insusceptible, is incorrect. Owing probably to the complete lack of protection by previous attacks, aboriginal tribes such as the North American Indians are liable to be reduced almost to extinction by small-pox epidemics. When inoculation was practised, the period of incubation was almost invariably from eight to nine days; the more usual time is from nine to ten days, but it varies from eight to fourteen and in rare cases even twenty-five days.

MORBID ANATOMY.—The skin presents the remains of the eruption either as crusts, pustules, or ulcers, which in hemorrhagic cases are infiltrated with blood. Pustules or ulcers may also be found in the mouth, pharynx, and œsophagus, and in the upper air-passages. In the latter a fibrinous exudation may be present. The dependent portions of the lungs are often collapsed, injected, and œdematous, and patches of lobular pneumonia or broncho-pneumonia are frequent. The heart is flaccid, of a pale-gray color from granular degeneration of its muscular fibres. The liver and kidneys also show evidences of parenchymatous degeneration, and the spleen is enlarged and soft from acute hyperplasia. Minute necrotic foci have been found by Weigert in the liver, spleen, kidneys, and lymphatic glands, and Chiari has described multiple minute spots of a grayish-yellow color rapidly tending to become necrotic. Weigert and Bowen have found focal necroses in the lungs, liver, spleen, kidneys, and lymph-glands. Chiari and Mallory observed similar lesions in the testes and bone-marrow.

In hemorrhagic small-pox the above-described changes are lacking, since time is required for their development. Characteristic are hemorrhages in the skin, in the mucous and the serous membranes, within the muscles, joints, and bone-marrow, and in the loose connective tissue of the mediastinum and along the spine. Hemorrhages are rare in the viscera, although present in the renal pelvis and calices and in the uterine mucous membrane. The heart and spleen are dense, of a dark reddish-brown color, somewhat translucent. Nothing abnormal is found in the appearance of the liver or kidney.

SYMPTOMATOLOGY.—Small-pox is among the more consistent of the eruptive fevers, but for the purposes of study it may be divided into Simple small-pox (Variola vera), Malignant or Hemorrhagic small-pox, and Varioloid or mild small-pox as modified by previous attacks.

Simple small-pox is divided into three varieties: the *Discrete*, in which the pustules remain distinct from one another; the *Coherent*, in which though at first distinct they finally come in contact and join at the edges; and the *Confluent*, in which almost from the beginning they run together. It must be remembered that these varieties represent simply distinct degrees of intensity, and that they are not sharply separated from one another.

The course of an ordinary small-pox is divided into four periods: first, that of invasion; second, that of eruption; third, that of suppuration; fourth, that of desiccation and desquamation.

Invasion.—The onset of small-pox is sudden and accompanied with a violent rigor or very frequently with a series of rigors, which may last many hours. Fever sets in immediately, and the temperature may rise within a day to 104° or 105° F. The pulse is rapid, full, rarely if ever dicrotic, and sometimes has a distinct hardness. Vomiting always occurs, and is commonly severe and repeated. The matters vomited are in no way characteristic, consisting of the stomachic contents and mucous bile. There is often a very painful sense of epigastric constriction, and in most cases acute constipation exists. The skin and the mucous membranes are dry. The nervous phenomena are very pronounced; not rarely in the young child the chill is replaced or accompanied by convulsions, and the neuralgic pains are in their severity almost diagnostic of the fever. Headache and backache are constant and extreme. The latter is especially in the lower lumbar region, sometimes spreading upward even to the neck, and radiating into the legs and more rarely the arms. It is often associated with a sense of difficulty of respiration or of feeling of weight upon the chest, which may rise to a violent dyspnœa, with a great acceleration of the respiratory movements, that cannot be explained by any condition of the lungs. Delirium is very common; in the mildest attacks it consists simply of a little wandering at night; in the more severe and adynamic cases it may be hallucinatory, and is often so associated with tremors as to present an appearance of delirium tremens. In the severest forms there may be the muttering delirious unconsciousness of profound exhaustion, or a wild and even violent mania.

Often on the second or third day initial rashes appear, especially affecting the distribution of the abdominal nerves,—that is, of the nerves arising from that portion of the back which suffers most from aching pains. The eruption may resemble that of scarlet fever or of measles, or it may be erythematous or erysipelatoid; sometimes it suggests roseola, or even an urticaria-like localized œdema; if it be purpuric or ecchymotic it indicates the hemorrhagic form of the disease. It is usually from one to two days in its development, and fades in about the same time, though the scarlatinal rash, which is more tenacious than the other forms, may last six days. The initial rash occurs in about one-sixth of the cases, but is more frequent in some epidemics than in others. It is usual for the subsequent characteristic variolous eruption to be least abundant in those parts of the body which have been especially occupied by the initial rash.

Eruption.—From the third to the fourth day in a case of discrete small-pox there is usually abatement of the constitutional disturbances, with the appearance upon the forehead, especially near the roots of the hair, of small red spots that rapidly spread to the face and then to the body and limbs, which in from twenty-four to thirty-six hours are completely covered. The eruption quickly changes into distinct papules, and these again into vesicles, which are usually fully formed upon the face by the third day of the eruption, but on the extremities do not arrive at this degree of maturity until two or three days later. The vesicles are of various sizes, always, however, in the discrete form larger than in the confluent variola, and very distinctly umbilicated except upon the face. They are surrounded by a red areola, and on the face are usually opaque and purulent by the seventh or eighth day. During the stage of eruption the mucous membranes of the conjunctiva, mouth, pharynx, and larynx, vulva, and prepuce are intensely red, and have frequently on them an eruption which is usually proportionate in severity to that upon the surface of the body. The mucous membrane vesicles are small, often brilliant, and apt to leave small round erosions.

The defervescence at the beginning of the period of eruption is often abrupt, and the temperature continues low until about the seventh day.

Suppuration.—The period of suppuration usually begins from the seventh to the eighth day, and lasts about four days: during this stage the vesicles are converted into swollen pustules, accompanied often by great subdermal swelling, excessive irritation of the skin, and great pain upon movement. In severe cases violent conjunctivitis, excessive salivation, dysphagia, dyspnœa from œdema of the glottis, or bronchial inflammation, may occur. The fever during this period is pronounced, headache is usually present, and the sleep is unquiet and not rarely interrupted by delirium.

The fourth period, that of *desiccation*, may be considered to commence at the eleventh day and to last from ten to twenty days or even longer. On the face, and sometimes on other portions of the body, the pustules break, discharging their contents so as to make a purulent mask, or each pustule in mild cases may form its own distinct scab. The surface, as the scabs fall off, is left of a reddish-wine color, often excoriated or ulcerated, so that cicatrices of various form and appearance remain after convalescence.

At any time, during even a discrete small-pox, various complications may set in: usually, however, they are wanting. During the stage of invasion there is habitually an increase in the specific gravity of the urine, which may rise to 1075, and is largely due to extreme elimination of urea, though extractives, creatinin, xanthin, tyrosin, indican, and the sulphates are augmented; the chlorides are diminished. During the stage of eruption and suppuration, however, the urea diminishes, while the chlorides are greatly increased. Defervescence is often accompanied by a critical discharge of uric acid.

During the stage of invasion in *confluent small-pox* the symptoms differ from those of the ordinary form only in their much greater intensity, and in the tendency, which is especially seen in children, to diarrhœa. The eruptive stage is especially marked by the failure of the constitutional disturbances to subside, and by the peculiarities of the eruption. The whole face becomes excessively swollen; an erysipelatoid rash appears; whilst the papules are in enormous number and rapidly coalesce, so that in the vesicular condition the eruption seems to be bullous. In certain parts of the body, especially in the lower abdomen, the papules are distinct from one another, but they are always smaller and more numerous than in the true discrete variety of the disease. The mucous membranes suffer greatly: salivation, glossitis, dysphagia, aphonia, dyspnœa, diarrhœa, and dysuria are common symptoms. The fever, though it may abate for two or three days, never disappears, and the pulse remains frequent. During the period of suppuration the swelling of the surface becomes enormous, the features of the face almost disappear, the eyes being closed, whilst the movements of the swollen extremities are extremely painful. If the patient survive, desiccation begins about the eleventh day, but the fever persists, and rarely disappears until the fourth week, by which time the face is usually desquamating. Death may occur at any time during the disorder: it may be due to adynamia and be preceded by violent delirium and coma, or may be the result of asphyxia, produced by a rapid congestion, by a bronchial pneumonia, or by an œdema of the larynx. Not rarely it is the result of the septicæmia; sometimes it is due to a sudden cardiac failure the result of a myocarditis.

In *Malignant*, *Hemorrhagic*, or *Black Small-pox* the stage of invasion is usually very short, accompanied with very violent vomiting, anxiety, dyspnœa, horrible backache, and epigastric constriction, whilst the rash which precedes the eruption is more constant and severe and has a much greater tendency to be purpuric than in the ordinary disease. The hemorrhages usually appear about the fifth day, first as petechial spots, then as phlyctænulæ and subconjunctival ecchymoses, accompanied by violent epistaxis, hæmaturia, and at last bloody discharges from the mouth, intestines, uterus, bronchial tubes, and ears. During the whole course there is great adynamia, with rapid feeble pulse, heavy malodorous breath, not rarely paraplegia with retention of urine, various anæsthesias or hyperæsthesias, diphtheroid exudations, tympanites, and sometimes enlargement of the liver and spleen. The eruption is always discrete, and of a brownish or black color, whilst the vesicles fill with blood and go into pustulation. The temperature is at no time very highly elevated. Delirium and convulsions and terminal coma are common, but sometimes consciousness is retained almost to the end; death occurs from syncope or asphyxia.

In foudroyant cases the end may be reached before the appearance of any rash; more frequently it occurs after the rash, but before the specific eruption has been well formed. In less malignant cases the hemorrhage may not begin until pustules are well developed.

Diagnosis.—In the suddenness of invasion small-pox may at first resemble pneumonia, but is to be distinguished at once by the absence of physical signs and by the intensity of the backache. Owing to the character of the initial rashes, not rarely mistakes of diagnosis between it and scarlet fever or measles have been made. It is to be distinguished from scarlet fever by the absence of sore throat, and by careful attention to the minute characters and especially to the topography of the initial rash, which in small-pox is always limited in its distribution, is especially abundant on the lower abdomen, and rarely, if ever, appears upon the face. The rash of measles appears later than does the initial rash, and differs also in its distribution. Further, in both measles and scarlet fever the backache is never so severe as in small-pox. Nevertheless, although the differences seem so clear, yet cases do arise in which the diagnosis must for a time remain uncertain, requiring the practitioner to wait for the appearance of the small shot-like papules on the upper forehead before sending the patient to the hospital. In rare cases of measles there may be some papulation, but the papules lack the intense hardness of those of small-pox.

From small-pox in the vesicular stage varioliform syphilide, which is often accompanied with a pronounced fever, is to be differentiated by the slowness of its evolution, by the absence of backache, and by the fact that the temperature does not fall on the appearance of the eruption. There is also a variolous form of acne, but it is apyretic and develops slowly. Chicken-pox is to be distinguished from varioloid and other mild forms of small-pox by the oblong form and greater size of its bullæ, by their irregular dissemination, by the absence of distinct umbilication and of suppuration, and by the lack of severe constitutional disturbances. There are, however, cases in which for a time the diagnosis between chicken-pox and very mild varioloid must remain in doubt.

The severity and universality of the hemorrhages and the abundance of the petechiæ distinguish malignant small-pox from malignant scarlet fever, cerebro-spinal meningitis, and other similar affections. If death do not occur before the fourth day, the papules, even if they be not plainly apparent in the deeply discolored skin, can be felt in the region of the upper forehead along the edge of the hair.

PROGNOSIS.—The prognosis in small-pox varies with the epidemic and with the age. In the very young almost all the cases die. Alcoholism, old age, general feebleness of constitution, and previous chronic disease, increase greatly the danger. Taking all the cases, the mortality of variola in the unprotected is from forty to fifty per cent. in different epidemics. Pregnancy, especially in its later stages, increases the fatality. Abortion is almost invariable after the third month, is accompanied with great hemorrhage, and usually ends in death. The fœtus in a majority of cases suffers from the disease.

TREATMENT.—Every case of small-pox should be isolated from the first moment at which suspicion of the nature of the disease is aroused.

Free, even violent, ventilation of the room should be insisted upon, so as to prevent any condensation of the poison. Further, everything which is capable of affording a resting-place for the poison, such as carpets or hangings, should be removed from the room, whilst the personal and bed linen should be changed frequently, and always dropped at once into a solution of corrosive sublimate or into boiling water. The surface of the body should be frequently bathed, with the free use of carbolic acid soap, and after the bath the water should always have added to it sufficient of corrosive sublimate (1 to 500) or of carbolic acid (1 to 200) to destroy all germs. All discharges from the body should be immediately disinfected. (See Typhoid Fever.)

During the whole course of the disorder, unless there be a tendency to subnormal temperature, the patient should be lightly covered in the bed. The diet should be easily digested but highly nutritious, milk, strong broths, raw eggs, and similar foods being relied upon. It must be remembered that the suppurative process is very exhaustive, and the patient should be fed up to the full power of digestion. The use of baths is of the greatest importance; in the stage of invasion the hot bath will frequently relieve the pains, whilst whenever the fever is high the cold bath will reduce the temperature and often moderate the nervous disturbances. If there be delirium and subsultus, with a temperature of over 102.5° F., the bath of 80° F. may be used every three or four hours, the temperature of the water being reduced if it be not low enough to cool the patient. Symptoms must be met as they arise. Opium is especially useful in the period of invasion, and when there is much vomiting should be given in the form of suppositories. It is also serviceable when in the advanced stages there is great irritation from the suppurating skin, or when there is insomnia combined with delirium.

Laxatives in most cases are required from the beginning; but if diarrhœa should exist, opium, bismuth, salol, and similar remedies may be employed. Chloral given in small doses along with opium and hyoscine is sometimes useful in controlling maniacal outbreaks. As prostration comes on and increases, alcoholic stimulants, strychnine, and other stimulant remedies should be used. It is doubtful whether in malignant smallpox any drugs have perceptible power for good; nevertheless, various stimulants may be freely used, and an attempt may be made to check hemorrhage by the use of ergot and other hæmostatic remedies, though it would seem more rational to struggle only for euthanasia by the use of opiates.

As the inflammation and ulceration of the skin in variola not only are a source of immense suffering and after-disfiguration, but often play an important part in the production of a fatal exhaustion, the local treatment is a matter of great importance. Numerous plans have been tried from time to time, but experience seems to show that many of them are harmful, and few, if any, of value. Opening the pustules and forcing out their contents; altering the disease-processes by the application of nitrate of silver or iodine or other substance to the individual pustule; the employment of mercurial, iodinic, and other alterative ointments, and all similar procedures, are generally condemned by recent authorities. It would seem, however, that there is some truth in the old belief that light fosters the development of the pustules: hence it is well to protect the face and hands by the constant application of patent lint.* The local application of cold wet lint is generally very grateful to the patient, and some good may be hoped for from the addition to the water of antiseptics, such as salol (1 to 10), sodium salicylate (1 to 10), boric acid (1 drachm to the pint), corrosive sublimate (1 to 5000), and carbolic acid (1 to 200). The sensations of the patient should be the guide in regard to the temperature of the compresses, and also, in a measure, to the strength of the solution. When tepid applications are preferred to cold they should be used. Very grateful and very useful during the stage of suppuration and desiccation are prolonged warm baths, in which the patient is immersed for two or three hours once in the twenty-four hours, and by which the local inflammation is often greatly reduced. As a substitute for these baths, in France the person is sometimes fully washed two to four times a day with a warm solution of corrosive sublimate (1 to 1000).

VACCINIA. COW-POX.

DEFINITION.—An eruptive disease of the cow, the virus of which is capable of producing in man a pock, associated with constitutional disturbance, and having protective influence against small-pox.

In 1798, Edward Jenner found that in Gloucestershire milkmaids and others accidentally inoculated by the cow-pox were afterwards insusceptible to small-pox, and was thus led to his immortal discovery. It does not seem worth while to occupy space in this volume with statistics proving the value of vaccination. The fact that before the days of Jenner small-pox killed in England as many persons as all other diseases put together, in contrast with the present mortality from small-pox, is sufficient. If thorough vaccination and revaccination of whole communities were possible, small-pox would almost disappear. The mortality-rate of the mild forms of variola (*varioloid*) which occur in those who are protected by vaccination is not more than eight per cent., while in the unvaccinated it probably exceeds forty per cent. W. M. Welch's mortality statistics (Municipal Hospital of Philadelphia) give, in persons with good cicatrices, eight per cent. ; with fair cicatrices, fourteen per cent. ; with poor cicatrices, twenty-seven per cent. ; post-vaccinal cases, sixteen per cent. ; unvaccinated cases, fifty-eight per cent.

---

* The demonstrations of Unna, Hammer, Widmark, and others that it is the chemical rays of the sun, especially the ultra-violet rays, which irritate the skin and produce "sunburn," led Finsen to try the effect of completely excluding these rays from the rooms of small-pox patients, and his assertion that the severity of the eruption is greatly abated has been confirmed by Feilberg, Svendsen, and other Scandinavian doctors. The red sheets which protect all the doors and windows should, if of muslin, be of four thicknesses, though heavy flannel is preferable: red glass does very well if it is thick and dark. The chemical rays must be shut out as absolutely as in a photographic dark room, and only protected lamps (red) allowed. Further, it is essential that the treatment be begun during the stage of invasion.

Concerning the explanation of the immunity conferred by vaccination there has been almost endless discussion. Whilst nothing can be considered positively determined in the matter, it is extremely probable that cow-pox is variola which has been altered by its passage through the cow but yet has retained the protective power of the original disease. It is true that Chauveau, of the Lyons Commission, Warlomont, Berthet, and others have failed in their efforts to produce the vaccine disorder by the inoculation of cows with matter from variolous pustules; but more recent investigators, especially Pfeiffer, Fischer, Eternod, and Haccius, agree in affirming that if the liquid from the small-pox vesicles be brought in contact with a large and thoroughly denuded surface in the cow there will be produced a pustular eruption which after the second or third generation of reinoculation becomes identical with that of cow-pox and is able to produce typical vaccine disease in infants.

Stephen C. Martin states that the germ of cow-pox is an organism which is in one stage of development a coccus and in another a bacillus, and that by inoculating with pure cultures of this organism he has produced typical cow-pox in the calf and also in man.

Vaccination in man may be produced with lymph taken from the human vaccine vesicles, with the scab which follows vaccination, or with lymph taken from the cow. Injurious effects from vaccination are extremely rare, but syphilis and other constitutional or bacterial diseases may be transmitted. Very severe epidemics of vaccino-syphilis, the product of a mixed infection, have occurred, especially in Europe.

The vaccine disease as produced by animal material is more severe than that caused by humanized lymph, but it is probably also somewhat more protective, and, as by its use all danger of specific infection is avoided, the employment of humanized vaccine is ordinarily unjustifiable. When animal virus cannot be obtained, the greatest care should be taken in collecting the human vaccine to see that it is from healthy infants who are free from acquired or hereditary taint, and that it is collected without admixture with blood or possible septic matter.

So far as the protection is concerned, it makes no difference upon what part of the body the vaccination is performed, but in female infants of the richer classes the leg should be selected, for æsthetic reasons. The skin, after it has been thoroughly washed, first with warm water and soap and then with alcohol, and dried, should be very superficially cross-hatched with a dull lancet or the ivory point, and the moistened or liquid lymph be well rubbed in: care should be taken to see that there is no bleeding. After quiet drying, the spot may be protected by a clean linen handkerchief, or, if there be any special reason for fearing infection, with a dossil of antiseptic cotton.

Vaccination in a fresh subject is followed in from one to twenty-four hours by a reddish blush, with, in the centre, a papule, which increases and becomes vesicular, so that by the fifth or sixth day it is a well-formed orbicular vesicle. By the eighth day the vesicle is distended with limpid fluid, and has a hard margin, a marked umbilication, and a wide red area. Usually from this time the inflammation begins to subside; by the twelfth day the vesicle is opaque, beginning to dry; by the fifteenth day there is formed a distinct brownish scab, which falls off on from the twenty-first to the twenty-fifth day, leaving a large circular or irregular, deeply pitted scar. Fever is usually present from the third to the ninth day, varying in severity and duration in different cases, and often in children associated with pronounced nervous irritability. Swelling of the neighboring lymphatic glands is common, and may be troublesome. In unhealthy subjects the vaccine vesicles may be accompanied with great inflammation and end in ulceration. Local dermatitis and erythematous or roseolous rash may accompany the vaccination, probably without being the result of any special infection; but when erysipelas or septic cellulitis or contagious impetigo or tetanus results, as has occurred, there has been a double inoculation.

Usually the subsidence of the pock ends the disorder, but in rare cases secondary pocks appear in the vicinity of the original vaccination, or even in distant parts of the body. In feeble children a fatal result is possible.

Vaccination does not confer an absolute protection against small-pox: the susceptibility slowly increases as the years go by, so that revaccination is essential at not longer periods than eight or ten years. Indeed, whenever any person is exposed to an epidemic of small-pox, revaccination should be insisted upon. The vesicle in revaccination is usually smaller than that of primary vaccination, and accompanied by less induration and resultant scar. In cases of failure to "take" there should be repetition of the vaccination almost indefinitely if there be positive exposure to small-pox.

MILIARY FEVER.

DEFINITION.—A contagious malady, characterized by an eruption of miliary vesicles, ending in desquamation.

Although during the Middle Ages epidemics of the *English Sweating Disease* or the *British Plague*, as it was termed on the continent of Europe, carried off large numbers of people (eight thousand, it is said, in eight days in Augsburg alone), of recent years the disease has occurred rarely and in limited localities: so far as we are aware, it has not been reported at all in North America.

The period of incubation is from one to five days. With or without prodromes, suddenly or more slowly, the patient passes into a condition of great weakness, with high fever, excessive sweating, and various nervous symptoms, which occur especially in nocturnal paroxysms. Violent headache, sudden attacks of excessive dyspnœa, with agonizing constrictions in the throat and chest, or even in the abdomen, intense insomnia, mild or sometimes raging delirium, and wide-spread coldness of the extremities, are among the most characteristic symptoms of the night attacks, and may occur during the day. After from one to four days, rarely as long as a week, the eruption appears, with an increase of the fever, and especially of the nervous symptoms. This eruption is twofold in character,—in exanthem, which may resemble that of measles or scarlet fever or be hemorrhagic or purpuric, and a miliary rash, which consists of minute acuminated papules, rapidly developing into vesicles, which may be very small or by coalescence may be formed into bullæ. Twenty-four hours are usually sufficient for the completion of a vesicle: on the third day desiccation begins, followed about the fifth day by a furfuraceous or sometimes a massive desquamation. The urine, which is ordinarily scanty during the attack, often becomes, by the thirteenth or fourteenth day, suddenly very excessive, and the disease may break up with a urinary crisis. Convalescence is apt to be long and uncertain.

The mortality-rate varies greatly in different epidemics. It has risen as high as thirty-three per cent., but in the recent epidemics it seems to have been about two per cent. The treatment consists in proper nursing and hygiene, the use of cold baths as they are indicated, and the meeting of the various symptoms as they arise.

TYPHOID FEVER.

DEFINITION.—A specific fever, due to the presence of a peculiar bacillus, and running a course of from three to four weeks, with fever, a rose-colored eruption resembling flea-bites, and cerebral, pulmonary, and abdominal symptoms, and accompanied by lesions of Peyer's patches, of the spleen, and of the mesenteric glands.

ETIOLOGY.—Typhoid fever is due to a short, actively motile bacillus, originally discovered by Eberth and by Gaffky. This bacillus is very closely allied to the bacillus coli communis, from which it is often distinguished with great difficulty. It is killed by a temperature of 60° C., but is capable of resisting repeated freezing and thawing. It has been known to retain its vitality after having been buried for nearly six months, and probably can so live for years. In the body it especially develops in Peyer's patches, in the mesenteric glands, and in the spleen, but it has been demonstrated in the liver, in the kidneys, and in other organs. As ordinarily inoculated, it usually fails to produce in the lower animals any series of symptoms or any lesions comparable to those of typhoid fever in man, but, according to Sanarelli, when rabbits, guinea-pigs, and mice have been previously poisoned with the growth-products of bacterium coli commune and certain other saprophytes, a rapid infection is produced by inoculation with the typhoid germ ; and Alessi asserts that typhoid fever can readily be produced in guinea-pigs and rabbits which have been caused to breathe for some time the gaseous products of organic decomposition. The bacillus is discharged in great numbers with the fæces from persons suffering from typhoid fever, and probably also escapes to some extent with the urine ; and it is alleged that it has been found in the saliva.

The life-history of the typhoid bacillus outside of the human body is known only in very small part. In the laboratory the cultures can be propagated almost indefinitely, but appear to lose their pathogenic power. Left to themselves, in sewage, in milk, and in other organic mixtures the bacilli grow rapidly at first, but are soon destroyed by other bacilli. Air and sunlight hasten the destruction of the fever germ. No spores have as yet been discovered, though they probably exist.

The bacillus probably always infects the human individual through the intestinal tract, which in the majority of cases it reaches in the drinking-water ; although in numerous instances it has been carried by infected milk, and some very severe epidemics have been produced by eating oysters which had been planted near the discharging mouths of sewers. It is also probable that the bacilli may be carried upon the hands to the mouth, as typhoid fever is very frequent among laundresses who have washed the clothing of patients.

It is evident that, whilst the bacillus is always the immediate exciting cause of a typhoid fever, the predisposing causes are of the most serious importance ; else why is it, as Peters asked. that when a million of people are drinking the infected waters of the Seine, only a comparatively few are affected ? Typhoid fever is undoubtedly much more frequent in the late summer and autumn months than in winter and spring, in the southern temperate zone than in other climates. and after hot, dry summers than when the ground-water is high ; but. as it occurs in every climate, at all elevations, and in all sorts of weather, it is probable that these circumstances are simply factors in favoring the development of the organism, and not predisposing causes. Sex is without influence ; age is of more importance. as the disease is comparatively rare under fifteen and over thirty years. though it may occur in the youngest infant or in the oldest adult. We have seen it at three months, and it has been discovered in the fœtus.

The influence of over-crowding, of filth, and of exposure in the production of typhoid fever is not pronounced, but it is probable that whatever lowers the vital power of the human individual lowers the capability of resisting the typhoid bacillus, and it would seem that a certain immunity is acquired by habitual exposure to the bacillus in a not very virulent form. At least this is the most plausible explanation of the notorious fact that young people moving from the country into cities are attacked with typhoid fever in many times larger proportion than are the older inhabitants of the cities. There would appear, also, to be an hereditary susceptibility or lack of susceptibility to the attacks of the germ, as certain families are especially prone to be attacked, whilst others escape through successive generations. Pepper believes that intestinal catarrh strongly predisposes by affording a nidus of growth to the organism.

Morbid Anatomy.—The lesions characteristic of typhoid fever are in the intestine and in the mesenteric lymphatic glands. The many alterations to be found elsewhere in the body are chiefly dependent upon the infectious nature of the disease and the variations in its course.

The intestinal alterations are those dependent upon the swelling, necrosis, and sloughing of the lymphatic glands,—namely, the solitary follicles and Peyer's patches. A hyperplasia of the cells takes place, producing an enlargement of the follicles or groups of follicles, which assume an opaque gray color and a soft consistency. This condition, to which the term medullary infiltration is applied, continues throughout the first ten days, gradually increasing in extent and involving more or less of the follicles and Peyer's patches, especially from the ileo-cæcal valve upward. In some cases these alterations are limited to the ileum,—ileo-typhoid,—in others to the lymphatic follicles of the colon,—colo-typhoid,—and in still other cases the lymphatic follicles of both ileum and colon may be affected. The mucous membrane of the affected portion of the intestine is swollen, injected, and somewhat opaque from the associated catarrhal inflammation.

As the infiltration extends and compression of the blood-vessels occurs, and perhaps also on account of the direct action of the bacteria present, necrosis takes place in the inflamed follicle or Peyer's patch, in the latter often in several places. With the increase of the necrosis, which is favored by a cellular infiltration of the perifollicular tissue, more or less of the enlarged follicle or Peyer's patch forms an opaque yellow or brown slough, discolored by intestinal contents or extravasated blood, and surrounded by a line of demarcation, the tissue limiting which is swollen and injected, sometimes gangrenous. At a later stage of the inflammatory process the slough is detached entire or in part, gradually or rapidly, exposing the deeper layers of the mucous membrane, the muscular coat, or the subperitoneal fibrous tissue. Necrosis of the inflamed follicles takes place during the latter half of the second week, while the detachment of the sloughs usually occurs during the third week. The inflamed Peyer's patch may also undergo resolution, with absorption of the hyperplastic cells, a trabeculated meshwork enclosing depressed spaces being left, the mucous membrane covering which is often perforated (reticulated patches). When absorption occurs, a flaccid, œdematous, somewhat translucent mucous membrane remains, its outline corresponding to that of the patch. This termination in resolution of the inflamed patches may be evident after years by the presence of groups of pigmented specks, the shaven beard appearance.

Healing of the ulcers begins during the fourth week and extends over a period of a fortnight, the scar becoming covered with epithelium and showing no tendency to contraction. In protracted cases the healing of the ulcers extends over a longer period, and in relapsing cases the stage of medullary infiltration may be renewed, and generally it ends in resolution.

From the detachment of the slough occurs the danger of hemorrhage, which is usually gradual, though sometimes profuse and immediately fatal. In the former case the blood is intimately mixed with intestinal contents, or forms a continuous or broken clot ; in the latter the intestinal contents are liquid blood.

Perforation of the wall takes place from the extension of the ulcer in depth, and is, as a rule, preceded by the formation of fibrinous adhesions between the peritoneum at the base of the ulcer and that contiguous. The base of the ulcer, if gangrenous, yields to the pressure of the intestinal contents or to intestinal peristalsis and is torn through : thus the intestinal contents escape into the peritoneal cavity, causing a general peritonitis. A localized peritonitis may take place when the sloughing ulcer is in the vermiform appendix, or when the peritoneal surface of the intestine is firmly attached to adjacent peritoneum.

The mesenteric lymph-glands, especially those near the lower end of the ileum, the ileo-cæcal chain, become hyperplastic. They may increase to the size of pigeons' eggs, and are soft, and on section of a reddish-gray color. Resolution of the inflamed gland usually takes place, although necrosis may occur, with softening and detachment of the overlying peritoneum, resulting in the escape of the necrotic material into the peritoneal cavity and a consequent peritonitis.

The spleen is also hyperplastic, and during the second week may become tripled in size, except when it is atrophied or its capsule indurated. As the spleen enlarges it is at first of a dark-red color, and later becomes pale red. The consistency diminishes with the increase in size, and on section, especially in the later stages of the disease, the pulp resembles dregs of paint, and the follicles and trabeculæ are indistinct. Hemorrhagic infarction and abscess of the spleen may occur as complications. During the later stages of the disease as the spleen diminishes in size the capsule becomes flaccid and opaque.

The heart, liver, and kidneys show the granular degeneration of protoplasm characteristic of infectious disease. The heart is opaque gray and flaccid ; the liver is enlarged, opaque gray, the lobular regions indistinct. At times both liver and kidneys contain opaque white specks, which are either accumulations of round cells or foci of necrotic cells.

Ulceration of the larynx, catarrhal bronchitis, and hypostatic lobar and lobular pneumonia are frequent. Gross alterations of the brain and its membranes are infrequent, although a meningitis at times follows a complicating inflammation of the middle ear or acute parotitis. Microscopical changes affecting the ganglion-cells have been described by several observers. Venous thrombosis, especially of the veins of the leg, is of not infrequent occurrence, and sometimes proves a cause of fatal embolism during convalescence from the fever. Hyaline degeneration of the voluntary muscles, especially of the abdominal rectus, was discovered by Zenker, and may prove a source of hemorrhage into the muscle,—hæmatoma,—by favoring its rupture. Orchitis sometimes occurs.

SYMPTOMATOLOGY.—Although typhoid fever may begin abruptly with a chill, in most cases its development is so insidious that it is almost impossible to fix the day of attack,—weariness, malaise, epistaxis, headache, slight aching pains in the legs, increasing weakness, and accelerated pulse, with perhaps slight diarrhœa, being the only manifest symptoms. During this stage there commonly is in the evening a slight elevation of the bodily temperature, which as the disease progresses takes upon itself the peculiar almost characteristic temperature rhythm of typhoid fever. Not rarely there is at first such tendency to remission and exacerbation of the symptoms as to suggest the presence of malarial disease. As the days go by, the symptoms grow more marked, the pulse becomes more frequent and feeble, the fever very distinct; tympanites, often with tenderness and gurgling in the right iliac region, especially under pressure with the fingers, appears; and the habitual hebetude of the disease is manifested. If there be not at this time diarrhœa there is commonly a peculiar susceptibility to the action of laxative drugs; but constipation may exist. The tongue, at first covered only with a fine whitish fur, coats itself more deeply and becomes brownish and often red at the tip. About the seventh day the peculiar eruption appears; it consists of minute rose-colored spots, resembling very closely flea-bites, disappearing under pressure and rapidly reappearing when the pressure is taken off, and not perceptibly elevated above the surface. The eruption in most cases appears first upon the abdomen, but may come out upon the chest, the back, or even the limbs. The spots may be found with difficulty, are in most cases few in number, widely scattered, and appear in successive crops, beginning to fade in one or two days after they develop. Sometimes peculiar bluish or slate-colored macules precede the development of the characteristic eruption, and when there is free perspiration sudamina are usually abundant. In severe cases, during the second week of the disease the tinnitus aurium begins to give way to hardness of hearing, the simple hebetude to stupor, often with muttering delirium; unless prevented by special care, the tongue becomes dark brown, often gashed and red on the edges or tips, whilst sordes collect upon the teeth. During the third week, and sometimes into the fourth week, the symptoms steadily increase; the pulse grows frequent and feeble, the temperature reaches its maximum elevation; subsultus tendinum, muttering delirium, and carphologia evince the general exhaustion; whilst great abdominal distention and diarrhœa, with perhaps bloody stools, mark the severity of the local intestinal disease. Usually in most cases ending fatally extremely rapid, feeble pulse, coma or coma vigil, absolute dryness of the mouth, with an almost colliquative sweating of the hot surface, enormous distention of the abdomen, hiccough, irregularly intense hyperexia, and finally the Hippocratic face, mark the passage, by insensible degrees, from life to death. Sometimes, however, the course of the disease is more stormy, the delirium becomes furious, and the restless motor excitement deepens into violent convulsions, in which the patient may die or pass into a fatal coma.

When the case terminates favorably, the return to health may be marked by an abrupt crisis; usually, however, it is gradual. The tongue grows more moist and begins to clean; hour by hour the nervous symptoms subside; the pulse lessens in frequency and gains in power; the local abdominal symptoms little by little become less pronounced; the temperature falls; and so the patient passes into a condition of great weakness without active symptoms, from which he slowly emerges. When the tongue as it becomes moist cleans gradually at the tip and edges, the convalescence is usually steady; but when the throwing off of the fur begins in the centre or towards the base, and the surface is left smooth, red, and shining, the convalescence is apt to be tedious and interrupted by various accidents. There is loss of weight during the whole period of a typhoid fever, but it is in the early stages of the convalescence that the emaciation most plainly shows itself.*

Especially during the early stages of convalescence, bodily exhaustion, mental or emotional excitement, or improper food may produce a sudden rise of bodily temperature, constituting what is sometimes spoken of as a recrudescence.† The rapid abatement of the symptoms sharply separates the recrudescence from the genuine relapse, in which there is a true return of the phenomena of the disease. The tendency to relapse varies very greatly in different epidemics of the disease; by some writers the proportion of relapses is placed as low as one per cent., by others as high as ten or even fifteen per cent. As the cases are commonly seen in this country we do not believe that true relapses occur in more than four or five per cent. The most common period of their development is the second week of convalescence, though they may come on earlier than this, or after many days of freedom from fever. Their development is usually abrupt, and the rising temperature, whilst resembling that of the first attack of the disease, is more rapid in its elevation. Eruption, enlargement of the spleen, and abdominal and nervous symptoms similar to those of the primary disease occur in regular sequence, but usually the course of the relapse is much shorter, and the changes in the condition of the patient are more abrupt, than in the primary attack. The relapse may be more or less severe than the primary attack, and may in its turn be followed by a second or even a third relapse, so that the entire history of the case may spread over the course of many months.

---

* The description of typhoid fever given in the text is rather as it was formerly seen than as it is witnessed at present in our hospitals; in other words, it is a description of the disease as it exists in nature, unmodified by the cold-bath treatment.

† The terms "recrudescence" and "relapse" are variously used by different authors. Recrudescence has been defined to be a relapse without an intervening period of normal or nearly normal temperature. Many authorities believe that a recrudescence depends upon a reintroduction or a renewed activity of the bacilli.

Headache may be a very early symptom of typhoid fever, and, although it sometimes abates as the disease progresses, may continue throughout the case. It may be occipital or frontal or without localization. In some cases it is so severe as to constitute the most prominent symptom, and it may be associated with vertigo, intolerance of light and sound, retraction of the head, tinnitus aurium, and even muscular contractions in the back and limbs, under which circumstances the case closely simulates one of meningitis.

The typical mental condition of typhoid fever is a peculiar hebetude or mental immobility, which is the basis of the so-called "typhoid face," whose characteristics are dulness and lack of expression. In some of the cases there is during the first eight or ten days a troublesome insomnia, but ordinarily a tendency to somnolence predominates from the start, and becomes marked after the first week, when it may gradually deepen into a stupor with or without muttering delirium. The delirium may in severe cases appear as early as the second or third day of the disease, but ordinarily it is not pronounced until the patient is well advanced into the second week. Cases have been recorded in which the delirious excitement came on so early and so completely masked the more ordinary symptoms of the disease that the diagnosis of acute mania was reached and the subject sent to an asylum. It is affirmed by some French writers that this primary delirium may closely simulate severe melancholia and other forms of acute alienation. Ordinarily the delirium is first manifested at night, and especially in periods of half-sleep. It is usually quiet and muttering, but may become furious, with outcries and attempts at violence. It is very apt to vary from day to day in the same subject, and in fatal cases may persist until death, though more commonly it is replaced by coma. Even in its milder forms it frequently leads the patient, especially at night, to rise and wander off in search of the ignis-fatuus of his dreams. It is generally associated with high temperature; but Liebermeister calls attention to the fact that in some instances it is concurrent with marked depression of the temperature. In most favorable cases it gradually disappears, but it may continue into convalescence and pass by insensible degrees into a confusional insanity.

On the other hand, confusional insanity may come on during convalescence from typhoid fever after intelligence has been regained.

It is often difficult to decide how far catarrhal changes and the peculiar depression of the nervous centres are the causes of the early dulness of hearing, which may deepen into profound deafness, and also of the loss of tactile sensitiveness. The taste is almost abolished in grave cases, and the vision is dulled.

In advanced typhoid fever of very severe type continuous rigidity and spasmodic contractions of all or a part of the muscles of the trunk and of the extremities may develop, and even such local spasms as those which produce strabismus or trismus may be pronounced without the existence of any meningeal inflammation. Violent epileptiform convulsions occasionally occur.

Special Symptoms.—Elevation of temperature is among the earliest of the symptoms of typhoid fever. In typical cases during the first week there is a peculiar ascent of the temperature which is almost characteristic of the disease,—the minimum temperature in the morning and the maximum in the evening being from half a degree to a degree and a half higher than the corresponding temperature of the day before. As the daily swing of temperature is from a degree and a half to two degrees, the morning temperature is about half a degree lower than the temperature of the evening before. By the eighth or ninth day a morning temperature of 102.5° to 103° F., with an evening temperature of 104° to 105° F., may be reached, and be maintained with more or less steadiness until some time in the third week. The first abatement of the fever is usually shown in the morning temperature, so that the daily swing will be two or even more degrees, and in the fourth week a fall and rise of three or even four degrees is not rare. The course of the temperature curve of typhoid fever is so modified by modern treatment that a typical temperature chart is rarely seen ; and even under the old methods great irregularities were often present during the second and third weeks. 105° F. is a common maximum ; 106° F. is not rare, but is always of very serious import ; when 107° F. or over is reached death is usually not far off. The time in the twenty-four hours of the highest temperature varies : it is generally about five or six in the evening, but may be much earlier or later. Rarely there are two temperature maxima in a single twenty-four hours, and cases of inversion of the temperature rhythms—*i.e.*, with the highest point in the morning—occur, especially in young children. The subsidence of fever is ordinarily gradual, but may be abrupt, and is commonly accompanied by great irregularities of the temperature.

The pulse in typical typhoid fever is accelerated from the very beginning, and throughout the course of the disease preserves a certain parallelism with the temperature, being also closely affected by the degree of exhaustion and by the various accidents of the disease. Cases have, however, been reported in which an early fall in the pulse-rate has persisted through the whole attack, and in some instances the patient has passed through the cycle of changes with a normal pulse-rate, notwithstanding a distinct elevation of temperature. At first the pulse may be full and offer a degree of resistance to the finger, but it soon becomes very soft, though large, and early in the disease is apt to take on a dicrotic character. In rare cases it may be tricrotic or even polycrotic. In bad cases the heart-sounds are altered in the accentuation, or there may be disappearance of the first sound whilst the second loses its sharpness and in rare instances may be duplicated; the præcordial impulse also may become so markedly diminished as to be replaced by a simple undulation of the chest-wall.

The respiratory movements are increased in frequency in typhoid fever to a degree corresponding with the height of the fever. Early in the attack there is often some cough, which may subside or may continue throughout the disease. In the advanced stages, especially of bad cases, increased rapidity of breathing may often be noticed, and be accompanied by lessening of the basal pulmonary resonance and also of the respiratory murmur in the posterior portion of the lung, with or without the development of a coarse crepitant râle. Under such circumstances there is present the so-called hypostatic pneumonia. True lobar pneumonia may also be developed at any stage of a typhoid fever, and give rise to symptoms which are similar to but less pronounced than those of the ordinary disease; it is to be recognized by the association of percussion dulness with bronchial breathing and increased vocal resonance. Hemorrhagic infarcts and pulmonary apoplexy, which were noted by Hoffmann in five out of two hundred and fifty autopsies on persons dead of typhoid fever, should be suspected when hæmoptysis occurs.

In most cases of typhoid fever the abdominal symptoms are pronounced; loss of appetite occurs early; and whilst in the majority of instances there is not much nausea or vomiting, these symptoms may be so continuous and severe as to suggest bilious fever. Diarrhœa is an almost universal symptom, but the bowels may not move frequently unless there be some laxity in the diet or a loosening medicine be given. In rare cases there may be constipation, and autopsies have been reported in which scybala were found resting upon the typhoid fever ulcers. The characteristic stool of typhoid fever is of a light ochre-yellow color, very watery, offensive, alkaline, and, it may be, ammoniacal. It separates on standing into an upper serous and albuminous layer and a lower flaky sediment. Brownish stools are common in the early stages; frothy, pultaceous, and even purulent stools sometimes occur. Intestinal hemorrhage happens in about five per cent. of the cases; it is very rare during the first week, and when present is probably the result of an oozing from the mucous membranes and is of small amount. Serious hemorrhage is most frequent in the third week; it may give rise to tarry stools or red bloody discharges with or without jelly-like clots; it is often recurrent. It is always a serious symptom, but our experience in civil life coincides with that of Trousseau and of Graves that it is rarely fatal. Statistics seem, however, not to be in accord with this, and epidemics have been reported in which nearly all the cases died. Liebermeister saw a death-rate of twenty-seven per hundred, Murchison fifty-three per hundred, and Homolle (four hundred and ninety-eight cases) forty-four per hundred. When the intestinal hemorrhage accompanies other hemorrhages and is the outcome of a general blood dyscrasia, the prognosis is very grave. When the bleeding is due simply to the ulceration opening one or more blood-vessels, the danger is usually in direct proportion to the amount of the hemorrhage. According to Murchison, perforation occurs much more frequently after hemorrhage than in other cases. A large intestinal hemorrhage is accompanied by a sudden drop in the temperature (even as much as seven degrees), pallor, free sweating, coldness of the surface, faintness, and failure of the circulation. A fatal internal hemorrhage may occur without the voiding of blood and cause collapse ending in death. The nervous symptoms are often temporarily abated by the hemorrhage.

Perforation of the intestines, according to Murchison, takes place in about eleven per cent. of fatal cases. In its typical form it attacks the small intestine, and is ordinarily simple, though it may be multiple. In rare cases it has been noted in every portion of the large intestine from the cæcum to the rectum. It is said to be more frequent in males than in females, and especially to occur in severe cases in which there has been an abundant diarrhœa, but it may suddenly end the scene even in walking typhoid. It has been noticed as early as the eighth and as late as the hundredth day of the disease, but is especially apt to occur in the early part of the third week. Its happening in a typical case is marked by a chill, very pronounced fall of temperature followed in a short time by hyperpyrexia, nausea, vomiting which may in rare cases be fæcal, lessening of the alvine discharges, increased meteorism and abdominal tenderness, and very anxious face. It may, however, occur without any pronounced symptoms, and be entirely masked. When perforation of the large intestine takes place, the general symptoms are much less severe than when the small intestine is the seat of the lesion, and the resultant peritonitis is often local, ending in an abscess which is usually fæcal.

Enlargement of the spleen, beginning from the third to the seventh day of the disorder, is almost universal, and, though liable to be masked by the tympanites, can usually be detected during life by gentle percussion and palpation. The smooth, not indurated, slightly tender organ may be three times its natural size, and generally begins to diminish at the close of the third week.

The urine of typhoid fever is usually less than the norm in amount, extremely acid, and of high specific gravity. When convalescence occurs there is an increase in the urinary discharge which may amount to polyuria, with a more than corresponding decline in the specific gravity, which sometimes falls to 1003. At the same time the urine becomes less acid, in some cases alkaline. During the period of increasing fever there is a notable increase in the amount of urea eliminated: Vogel noted the enormous daily output of seventy-eight grammes. During the second and third weeks the elimination of urea remains above the norm, but falls as convalescence becomes established, even to a point much below the norm. The uric acid is always increased during the febrile period, and decreases with defervescence. The chlorides, phosphates, sulphates, and carbonates are diminished during the fever, but increase with defervescence. According to the researches of Roque and Weil, the urine of the typhoid patient is extremely poisonous; Lépine and Guérin affirm that they have discovered in it a poisonous alkaloid. Teissier has found that whilst in mild cases of typhoid fever urobilin may or may not appear from time to time in the urine, in the severe cases it is persistently and abundantly present. In a proportion of cases varying according to different observers from twenty to fifty per cent. there is albuminuria, which usually appears in the second week, but may be developed in the first week, and may delay until the third or even the fourth week. It is more apt to occur in severe than in light cases. It may be due simply to the fever and the disturbance of circulation, or to nephritis. According to Bouchard, albuminuria appearing late in the disorder is especially prone to be connected with renal disease. Not rarely the presence of renal epithelium, of blood-globules, or of epithelial or granular casts renders the diagnosis of nephritis clear; and violent hæmaturia ending in death has been recorded by Duckworth, Greenhow, and others.

Varieties of the Disease.—The wide variations in the course and symptoms of typhoid fever. have led to the naming of a number of varieties of the disease.

The *gastric* or *bilious typhoid*, representing some of the cases of *gastric fever* of the older writers, comprises cases in which the vomiting is severe and prolonged. *Hemorrhagic typhoid* is a very deadly form of the disease, especially prone to occur in debilitated subjects suffering from scorbutus, alcoholism, etc. There is in it a rapid alteration of the blood, with profuse hemorrhage from the nose, mouth, intestines, and kidneys,—indeed, from all the mucous membranes,—and the formation of abundant ecchymoses, blotches, suggillations, etc. In these cases the adynamia is extreme from the beginning; the fever very high; the pulse very rapid and small; the heart-action greatly enfeebled; the tongue and the mouth loaded with a brownish deposit; the breath very fetid and even ammoniacal. Death occurs in a great majority of the cases, usually before the tenth day, and has been recorded as early as the third day.

Foudroyant typhoid is a very severe form, ushered in with convulsions and other evidences of intense nervous disturbance. *Ataxic typhoid* is that in which there is from the first profound exhaustion with high temperature.

In so-called *pneumo-typhoid fever* the first symptoms are complicated with, or replaced entirely by, those of pneumonia, so that it may be for a time impossible to decide whether the individual is suffering from a simple pneumonia with typhoid symptoms or from a typhoid fever complicated with early pneumonia. Gerhardt, in 1875, recorded an epidemic of this form, which is also said to occur sporadically, especially in young children. Typhoid fever commencing with pleurisy is spoken of by French writers as pleuro-typhoid fever, and is said to occur sometimes epidemically.

In the mildest cases of typhoid fever the symptoms may be so slight as to be overlooked, especially when the subject belongs to the poorer classes, among whom care of person and early consultation of a physician are rare. Slight headache, insomnia, a little diarrhœa, a feeling of malaise and weakness, may constitute all the symptoms. In this way arises the so-called *walking typhoid.* The latency of these cases is remarkable, and the disease may progress without care until a favorable termination is reached, or, more usually, until the sudden coming on of severe symptoms, or the occurrence of perhaps a fatal accident, enforces attention. We have seen in hospital practice a patient walk into the hospital, remain seated for some hours, and die suddenly during the subsequent night without a diagnosis having been made, and at the autopsy have found acutely ulcerated Peyer's patches with perforation of the intestines of at least four days' duration.

Apyretic or *afebrile typhoid fever* includes a class of cases in which the temperature does not reach at any time 100° F., and may remain normal or subnormal through the whole course of the affection. This form of typhoid fever usually represents the mildest degree of the disease; but epidemics have been described in which, although the other symptoms of typhoid fever have been pronounced, and even death resulted, there has been little or no fever. In some of these cases a daily rhythm of temperature can be noted, in which the diurnal variation is produced by a fall of the morning temperature rather than by a rise of the evening, so that the patient may be said to have an inverted fever.

Abortive typhoid is a condition to which, under the name of *typhus lævissimus,* attention has been especially called by Griesinger. The symptoms are those of a typhoid fever compressed into a few days,—perhaps three, perhaps ten or twelve. The onset is always abrupt, usually with a chill, which may be very intense; the temperature rises rapidly, and has been noted as high as 105° F. on the third day; splenic enlargement appears at once; the rose spots may be abundant, and any or all of the usual typhoid fever symptoms and accidents, such as diarrhœa, intestinal hemorrhage, albuminuria, bronchial catarrh, and hypostatic congestion, may appear. The defervescence is usually abrupt, accompanied by excessive sweat, sometimes by a critical polyuria. During convalescence the patient is said to be liable to the same sequelæ as after ordinary fever. Although the resemblance between this disease and typhoid fever is so marked, great hesitancy must be felt in considering the two affections the same; but in a case in which sudden death occurred during convalescence Laveran found the characteristic lesions of typhoid fever in Peyer's patches.

The effect of age upon the symptoms of typhoid fever is decided, and even the anatomical lesions appear to suffer alteration. Thus, in the infant the infiltration of the abdominal lymphoid glands is much less than in the adult, so that the ulcerations are very slight and perforations almost unknown. Indeed, according to Rilliet and Barthez, a large proportion of Peyer's glands do not ulcerate at all, but recover by resolution. The symptoms present in the young child sometimes resemble so closely those of a remittent fever that one form of the disease is known as *infantile remittent*, the chief manifestations being gastro-intestinal disorder with a remittent fever. These cases are usually mild in type; in the more severe forms of the disease in children the fever is apt to be very high and constant. It is worthy of remark that these high temperatures are better borne by the child than by the adult. The pulse is ordinarily very rapid, rising sometimes to 150 and 180, but is very rarely dicrotic. The abdominal symptoms are less marked than in the adult; very commonly the abdomen is flattish, and the diarrhœa is usually very mild; moreover, constipation is not extremely rare, and fæcal accumulation sometimes takes place. Intestinal hemorrhage is almost unknown. The rose-colored eruption is frequently absent, and only rarely abundant. The nervous symptoms of the disease are ordinarily well marked; convulsions, strabismus, inequalities of the pupil, irregularities of the pulse, and coma may give an almost complete picture of meningitis. The tendency to the occurrence of focal symptoms is shown by the fact that sometimes aphasia occurs. The mortality of the disease is distinctly less than in the adult, and in fatal cases death is commonly due to pneumonia.

In the very old, typhoid fever is rare, but extremely dangerous; the onset is usually very slow and insidious. The spots are commonly wanting; the splenic tumefaction and other abdominal symptoms are less marked than in the young. On the other hand, the adynamia is very pronounced, whilst the tendency to pneumonia is extreme. The intestinal ulceration is often severe, so that perforation is not rare, whilst severe hemorrhage is very common.

Complications and Sequelæ.—There can be no doubt that the typhoid bacillus may coexist and grow along with various other pathogenic germs in the human body, producing mixed forms of disease. Thus, although it is rare for a typhoid fever to occur in persons suffering from tuberculosis, the coexistence of the two diseases in active process was proved by Kiener and Villard by examinations made during life and after death. A number of cases have been reported of erysipelas developing during typhoid fever. Friedländer, Galliard, and others have reported cases, many of them with autopsies, in which typhoid fever and cholera coexisted. Murchison states that he has seen eight cases in which there was coexistence of typhoid fever and scarlet fever, and has five times seen typhus graft itself upon a typhoid.

Of all these mixed diseases, however, the one which has attracted the most attention and is of the most importance is the so-called *typho-malarial fever*, which has been especially studied in this country by Woodward, and in France by Colin. The recognition of the true nature of such a fever may be rendered easy by the occurrence in the typhoid of a manifest quotidian, tertian, or even quartan paroxysm; but very commonly the symptoms are so interwoven and obscure as to render the recognition of the two poisons difficult. When typhoid fever occurs in a highly malarial district, or in a person suffering from chronic malaria, or when the enlargement of the spleen is excessive, the blood should be carefully examined for the malarial organism. During the late civil war the mortality-rate of cases diagnosed as typho-malarial fever was a little over eight per cent., but it is probable that a very large proportion of the cases represented as instances of typho-malarial fever were instances of malarial fever with typhoid symptoms, whilst others were pure typhoid. Only when the typhoid fever eruption is plainly marked, or the ulceration of Peyer's glands revealed at the autopsy, or the bacilli of Eberth found in the stools or in the body after death, can the practitioner be sure that there has been a true typhoid fever infection, and absolute proof of double infection would require the detection of both malarial and typhoid lesions or organisms. In some cases the typhoid and malarial fevers follow closely upon each other, although remaining distinct: so that it is possible to have relapses of the typhoid with malarial attacks between them.

Not only may typhoid fever be combined with any one of the bacterial diseases, but almost any local affection may arise during its course. A few scattered cases in literature of hemiplegia, monoplegia, and aphasia, arising during the active course of the typhoid, show that the nerve-centres may suffer from hemorrhage, thrombi, and other organic affections. Abscess of the liver, infarction and abscess of the spleen, parotitis, orchitis, abscess in the muscles and cellular tissues, inflammation of the lymphatics, periostitis, osteomyelitis, arthritis, and local gangrenes, have all occurred from time to time during a typhoid fever. Much more frequent than any of them, however, is nephritis, which may be the cause of death. In its history and in its lesions this nephritis does not differ from that of scarlet fever, variola, or other infectious disease.

Convalescence.—The convalescence of typhoid fever is usually slow and protracted, especially after a severe case, and many months are often required for the gathering together of the strength, and especially the powers of endurance. Not rarely great mental inaptitude and inability to study or to do intellectual labor remain after the general strength has in great part returned. In our experience, however, the mental powers have always been finally recovered. Moreover, it is not rare for a patient in the end to gain strength and health beyond what was his previous norm. Sequelæ are exceptional. The most important are insanity of the confusional type, paralysis, and acute phlebitis, ordinarily affecting one leg and producing a true phlegmasia alba dolens. The paralysis is generally not complete, and is commonly attended with wasting of the muscles, and sometimes with contractures or other evidences of muscular irritation. The most frequent form is that of paraplegia, monoplegia being, however, not very rare. There is usually some disorder of sensation, and there may be complete anæsthesia. The bladder and even the rectum may for a time be paralyzed. The nervous lesion varies: in some cases it is a peripheral neuritis, while in other instances it is a myelitis. When there is paralysis of the bladder or rectum, with paraplegia and trophic disturbances in the legs, the lesion is probably always spinal, and the prognosis is more grave than in a peripheral monoplegia. The paralysis may develop suddenly; but usually its onset and its subsidence are alike gradual. The prognosis is much more favorable than in similar paralyses due to other causes than infection.

As has already been stated, periostitis or osteomyelitis may come on during a typhoid fever. More frequently the bone lesion manifests itself during convalescence, the first symptom being heavy, aching, localized pain, with slight swelling and some soreness. This may be followed by resolution, or, more commonly, after a remission which may last from six to eight months, by a return of the symptoms and a very slowly developed necrosis. As was first determined by Ebermaier, these bone lesions depend upon the local deposit of the typhoid bacillus. It has further been shown by Dmochowski and Janowski that the typhoid bacillus may be deposited in any tissue and produce suppuration, so that under certain circumstances it is a pyogenic organism. The typhoid bacillus has been found in the brain membranes in purulent meningitis after typhoid, and it is probable that milk-leg, neuritis, and other local lesions occurring during or after a typhoid fever are directly due to local deposits of the bacillus.

Diagnosis.—For the purposes of treatment it is essential that typhoid fever should be suspected at a time when the lack of development of the symptoms may make a positive diagnosis impossible. Whenever in a youngish or middle-aged person there is an acute and increasing malaise and weakness, without apparent cause, with an elevation of temperature in the evening, typhoid fever should be suspected. If at such a time it be found that getting the patient suddenly from bed into a standing position notably increases the pulse-rate, the case should be treated as one of incipient typhoid fever. The occurrence of epistaxis, diarrhœa, or other of the peculiar symptoms or of the regular ascending febrile movements of typhoid fever makes the diagnosis highly probable, although it can very rarely be positive until the appearance of the eruption.

There can be no difficulty in the diagnosis of well-developed typhoid fever, the characteristic symptoms being the progressive, ascending fever, with increasing weakness, nervous disturbance, diarrhœa, enlargement of the spleen, and the eruption. In aberrant cases of the disease, however, especially in those which resemble meningitis, it may be necessary to reserve the diagnosis until the rose-colored spots can be found or until time has made the matter clear. The typhoid symptoms are, however, in such cases usually so strongly pronounced as to give an inkling of the true nature of the disease. When pneumonia develops in the beginning of a typhoid fever it may not be possible to determine at once whether the case is one of pneumonia with typhoid symptoms or of typhoid fever with pneumonia. Here again a guarded opinion must be given until the appearance of the rose spots. Acute tuberculosis sometimes very closely resembles typhoid fever, and may offer any symptom of the disease except the eruption ; indeed, even this may be simulated, except in its recurrence in successive crops. A peculiar shifting character of the local symptoms in any obscure case is suggestive of tubercular disease : thus, if to-day the manifestations point towards meningitis, to-morrow towards pulmonary involvement, whilst the next day the abdominal symptoms are most pronounced, or if rapidly shifting pulmonic congestion come and go, tubercular disease should be strongly suspected. A regular typhoid fever ascent of temperature tells strongly in favor of a diagnosis of typhoid fever, but, unfortunately, irregularities of fever are no proof that the case is not one of aberrant typhoid.

Erhlich's test—the diazo-reaction—is of limited value in the recognition of typhoid fever. It is true that it is usually responded to after the first week of the disease, but it certainly is also responded to in tubercular disease, including acute tuberculosis, in septicæmia, in measles, in pneumonia, and probably in various other febrile diseases. When after a week or ten days of obscure symptoms there is still difficulty in differentiation between gastritis or malaria and typhoid fever, it may be of service. For the test a one per cent. solution of sodium nitrite and a half per cent. solution of muriatic acid saturated with sulphanilic acid are kept separate, but mixed in the proportion of forty to one just before using ; after mixture nitrous acid is liberated by the action of the hydrochloric acid upon the sodium nitrite, and produces with the sulphanilic acid diazo-benzene-sulphonic acid. In making the test, equal parts of the mixture of the two solutions and of urine are thoroughly shaken together, and ammonia is poured upon the top. At the line of junction a ring forms, which in normal urine is not reddish, but in typhoid fever urine is of a color varying from carmine to a deep garnet.

The presence or absence of the typhoid bacillus in the stools of a suspected typhoid case is of diagnostic import. According to Elsner, the typhoid bacillus is readily differentiated by making cultures of the typhoid stools on potato gelatin impregnated with one per cent. of potassium iodide. The latter agent is stated to kill all the organisms of the fæces except the typhoid and the colon bacillus. At the end of twenty-four hours the colonies of the colon bacillus are very distinct as largish, brown, coarsely granular spots, whilst those of the typhoid bacillus are evident only after forty-eight hours as transparent, small, colorless, finely granular spots.

Prognosis.—The mortality of typhoid fever varies very greatly in different epidemics and in different classes of individuals, and has recently been distinctly modified by improvements in methods of treatment. The statistics of the Paris Hospital from 1888 to 1894, inclusive, give an average mortality in nearly nine thousand cases of twenty and six-tenths per cent., whilst from 1866 to 1881 the mortality was twenty-one and five-tenths per cent. In the General Hospital of Vienna, from 1846 to 1861, the mortality was about twenty-three per cent. In the French army, from 1875 to 1891, about one hundred and thirty thousand cases of typhoid yielded an average of twelve and five-tenths per cent. of deaths, the mortality-rate varying from eleven per cent. in some years to fifteen per cent. in others. The same statistics show plainly the difference in the typhoid fever in different localities, the rate varying in the one hundred and sixty-two large garrisons of France from four and six-tenths to twenty-three and seven-tenths per cent. It seems fair to suppose that this great difference was the result not of differences of treatment, but of the original character of the disease.

In the Pennsylvania Hospital, out of six hundred and twenty-one cases, from 1862 to 1881, nineteen and five-tenths per cent. died, so that the mortality in this country is probably not far from what it is in Europe. In all these hospital reports are, however, included many cases which have come under medical care only at an advanced stage of disease, and some when moribund, so that in private practice upon the well-to-do classes the older mortality-rate was probably from ten to twelve per cent. The use of the cold bath has very sensibly reduced the average mortality-rate of the fever, some institutions reporting as low a rate as five or six per cent. In applying the general mortality-rate to the individual case it must be remembered that the danger of death is greatly increased by previous disease, old age, or obesity, and also by any neglect of early recognition and treatment of the disease. The more severe the symptoms in the first week the greater the danger. High temperature is always an unfavorable omen, especially high temperature which is persistent, has a high morning register, and cannot be controlled, except with the greatest difficulty by means of the cold bath. The early development of muttering delirium, especially with tremor and other evidences of marked adynamia, is a bad indication. Coma vigil or general convulsions are sometimes spoken of as fatal symptoms, but we have seen recovery from each. A very unfavorable symptom is the belief on the part of the patient that he is not sick, or that he is away from home and should go there. Rigidity of the limbs, with symptoms of meningitis, is a serious but not necessarily fatal condition. Great rapidity of the pulse, especially when combined with irregularity, is very ominous; in Liebermeister's statistics seventy per cent. of the cases in which the pulse was over 120 ended fatally. Slipping down in bed, marking as it does an extreme adynamia, great coldness of the surface and the extremities, and general cyanosis, often indicate the approach of death. Persistent dryness of the tongue with excess of sordes and excessive tympanites are much less serious than the symptoms just spoken of, but require a guarded prognosis.

Any of the accidents or complications of the disease add greatly to the gravity of the situation. Perforation usually ends in death; and peritonitis without perforation is only a little less fatal. (See also p. 128.) The significance of intestinal hemorrhage has already been spoken of. (See page 128.) Albuminuria indicates simply that the case is a grave one, but when persistently associated with tube-casts or other evidences of nephritis renders the prognosis doubtful. Hypostatic congestion of the lungs is serious, but is probably recovered from in the majority of cases; true consolidation of the lungs, occurring in typhoid fever, very commonly ends in death.

It must, however, be remembered that so long as there is life in a typhoid fever so long is recovery possible, and that therefore an absolutely hopeless prognosis is very rarely justified. We have seen recoveries after prolonged coma vigil, pulselessness, absolute inability to retain anything on the stomach, subnormal temperature, and a seemingly hopelessly moribund condition. On the other hand, sudden death sometimes occurs in typhoid fever without distinct warning; and any accident may in a moment entirely alter the aspect of the case. For this reason the prognosis should always be guarded. Pulmonary embolism from venous thrombosis, and cardiac failure, are the usual causes of sudden death, a termination which in our experience has especially occurred in cases with atheromatous arteries or "athletic heart," either of which conditions adds greatly to the danger of a typhoid fever.

The mortality of typhoid fever is greater in women than in men, and the existence of pregnancy notably increases the danger. Out of three hundred and twenty-four cases collected by Sacquin abortion occurred in two hundred and five,—that is, in sixty-four per cent. It takes place usually from the seventh to the fourteenth day, but may happen very early or be delayed even to convalescence. The fœtus is usually but not always dead before expulsion, and in a large proportion of cases probably both the death of the fœtus and the abortion are the result of the passage of the typhoid bacillus from the mother into the child. The mortality of typhoid fever with abortion is about fifty per cent.

Owing to the insidiousness of the invasion and the gradual defervescence of typhoid fever, it is usually impossible to fix the first and the last day of the disease. Moreover, there is no sufficient reason for believing that the time required for the working out of the fever process is the same in all cases. In its typical form typhoid fever may be considered to have a duration of from three to four weeks, but, as has already been shown, there are cases in which convalescence occurs at an early period, and according to our experience cases also occur in which the fever continues for five or six weeks without its being possible to detect any reinfection from outside of the body or any sufficient complication to account for the continuance of the fever. The most fixed point is the day of the appearance of the eruption, the seventh to the ninth day.

PROPHYLAXIS.—As in the vast majority of cases the typhoid bacillus finds access to the patient in the drinking-water, whenever there is an epidemic of the disease in a city, water which has been boiled for some minutes or which has been bottled from some distant spring should alone be used. The fact that the average yearly number of cases of typhoid fever admitted into the hospital in Munich was abruptly and permanently changed from five hundred and ninety-four to one hundred, and the annual number of deaths in the city from two hundred and eight to forty, by a reform in the drainage and water-supply, shows the immense importance of sanitary precaution.

The dejections should always be received into a bedpan which has previously had placed in it either one-quarter pound of chlorinated lime or half a pint to a pint of a ten per cent. solution of carbolic acid. When it is possible, the pan should be emptied into a receptacle containing a large amount of chlorinated lime, so that it shall be impossible for the bacillus to escape destruction ; if an ordinary cesspool or water-closet is used, the bedpan after use should be allowed to stand for at least half an hour before being emptied, so as to insure the killing of all germs. After emptying, the bedpan should be thoroughly washed with a solution of five to ten per cent. of carbolic acid and have put in it the new charge of germicide. There is probably little choice between chlorinated lime and carbolic acid, except that the chlorinated lime is less apt to produce accidental poisoning. Care is, however, necessary to see that the chlorinated lime is of good quality and is used with sufficient freedom.

The bed- and body-linen of the patient should be changed frequently, perhaps daily, and immediately whenever they are soiled. So soon as they have been taken off they should be tied tightly up in a clean sheet and be put without opening into a boiler, where they should be boiled for not less than half an hour before opening. If circumstances require that the linen should be washed away from the house in which the sick person is, the bundle should be thrown immediately after tying it up into a five per cent. solution of carbolic acid and allowed to soak for at least six hours before rinsing. In case of death, a ten per cent. solution of carbolic acid should be immediately injected into the rectum, and the corpse should be wrapped in a sheet wet with a three per cent. solution of carbolic acid.

When practicable, it is certainly preferable to protect the mattress and pillows by close-fitting rubber covers. If the mattress be fouled by the discharges, it should be soaked in carbolic acid solution and then taken to pieces. Under no circumstances should proprietary disinfectants be used: they are all more or less uncertain in action and greatly excessive in price.

TREATMENT.—The hygienic and general management of a case of typhoid fever has probably much more to do with the result obtained than has the medication. The trained nurse is so essential that no skill on the part of the physician will atone for her absence. So soon as there is the slightest reason for suspecting that a person is getting typhoid fever, absolute rest in bed, with the use of the urinal and bedpan, should be insisted upon. No harm will have been done if the suspicions be not confirmed, whilst incalculable harm may result from unnecessary loss of strength during the first few days of the attack. The bed upon which the patient rests should be neither too high nor too low, nor yet too wide for practical nursing; at the same time it should be wide enough for the comfort of a restless subject. It should always be a mattress, not too hard, preferably one upon springs. The room should be as well ventilated, and in summer as cool, as possible.

The question of food is most important, and should be carefully supervised in its details by the attending physician. The food should be chiefly liquid, but very early in the attack and during the beginning of convalescence semi-fluid food may often be allowed with advantage, provided it be given in not too large quantities. Thoroughly cooked farinaceous foods, custards, junket, very soft boiled, shirred, or raw eggs, and fine hashes, may be mentioned as fulfilling the indications. The feeding should always be at short intervals: semi-solid food should be given in small quantity at meal-time, purely liquid food being taken with such freedom between meals that the patient will want but little. Throughout the disease the chief reliance is milk, which ordinarily should be diluted with from ten to fifteen per cent. of lime water. When the digestion is extremely feeble, further dilution may be required: as a diluent barley water is distinctly preferable to ordinary water. Partially peptonized milk, when taken without repugnance, is advantageous. The substitution of koumiss, matzoon, or even buttermilk, for a portion of the milk, sometimes affords variety. Often when milk is repugnant to the patient's palate the addition of a little salt will cause it to be relished. Raw eggs given in sherry or with milk in the form of eggnog are often very well borne and very nutritious. The various animal broths may be used as adjuvants to milk, but are not to be relied upon as affording much nutrition; beef essence should be considered as stimulating rather than nourishing. All meat-essences or broths should be made fresh; "peptonoids,". Liebig extracts, and every other form of artificial meat-essences or extracts should not be allowed in the sick-room. In some cases peptonized oysters afford an innocent and highly nutritious liquid food, of which, however, the patient soon tires.

In all cases a record should be kept of food and medicines as administered during the twenty-four hours, so that the attending physician may know exactly what has been taken. During the day food should be given at intervals of two hours. In regard to the night, there is danger on the one hand of breaking the sleep of the patient, and on the other of exhaustion from want of food. If the patient be in a semi-stupor and fall asleep directly after being disturbed, the night intervals may be short; but if there be a tendency to insomnia and nervous restlessness, they should be long: the skill of the physician finds exercise in balancing between the difficulties. Very rarely is it proper to allow a typhoid fever patient to go more than four hours without food. The physician in charge should always make a written schedule for the twenty-four hours of the times of feeding, administration of medicine, etc.; and if this be so arranged that food is given at ten P.M., two A.M., and six A.M., there will be little disturbance of the patient. When the food and stimulants are given at these long intervals they should be in larger amounts; and if the exhaustion be severe, the intervals should never be longer than three hours.

It is impossible to lay down with any accuracy the amount of food; the object is to get as much digested as possible, and to put none in the alimentary canal which cannot be digested. Most patients will take two quarts of milk or milk-food, such as koumiss, with four eggs, in the twenty-four hours; some require much more, some less. Sick stomach, excessive tympanites, excessive diarrhœa, and above all the appearance of curds or other particles of undigested food in the stools, indicate that the patient is taking more food than can be digested. Under these circumstances peptonized foods are especially valuable, and it may become necessary temporarily to withdraw milk altogether, sustaining the patient with animal broths. Strong beef tea with an egg stirred into it whilst very hot may be advantageous. In some cases milk diluted with carbonic acid water is most grateful; and we have seen a mixture of milk and champagne taken by a stomach which would retain no other food. Coffee, tea, and even cocoa are rarely in themselves harmful, and may sometimes be used with great advantage for the purpose of getting a patient to take milk otherwise repulsive to the palate. Cold water may be given freely, but if the patient be taking already three quarts a day of liquid food there will be danger of upsetting the stomach with an excess of fluid, so that cracked ice is often preferable. In most cases the various liquid foods given cold will be more grateful to a patient; rarely the stomach does not tolerate them at low temperature.

Alcohol in some form, according to our belief, should be used in every case of typhoid fever from the beginning, unless there be some very strong moral reason for refusing it, as when there is a distinct heredity towards drunkenness. Given properly it is incapable of harm. There are two distinct uses of alcohol in typhoid and other adynamic diseases. Early in the attack, given in small amount with the food, it acts as a local stimulant to the digestive organs, and enables the patient to take more than would be otherwise possible; whilst in the advanced stages of the disease it is useful as a general stimulant, and should be given freely with the food and also at other times. In the first week of an ordinary case of typhoid fever a dessertspoonful of whiskey in a tumblerful of milk is a full dose, one ounce to two ounces of whiskey being given in the twenty-four hours; in the advanced stages of the disease twelve or even fourteen ounces of whiskey a day are sometimes necessary. Beyond this it does not seem to us it is wise to go. In deciding the amount of spirit to be given, the physician must be especially guided by its effects as well as by the degree of existing exhaustion: so long as the pulse under the use of the spirit becomes slower and steadier, and the tongue more moist, and the nervous symptoms less severe, so long is it probably doing good. Whenever the patient flushes or becomes nervously excited after the single dose, or whenever the odor of the liquor appears on the breath, or the pulse takes on the peculiar angry feel which every experienced practitioner must know, too much of the spirit is being taken. It should always be given at short intervals, in small quantities, usually every two hours during the day and every three or four hours during the night, the individual night portion being larger than the day allowance.

The most important part of the treatment of typhoid fever is that which has to do with the reduction of the temperature. Antipyrin, phenacetin, guaiacol, and similar antipyretic drugs will, if given in sufficient dose, certainly act efficiently in typhoid fever, but they may cause collapse. Moreover, they are powerful disturbers of the nutritive processes of the body, and the method of their action upon nutrition when altered by fever still remains a matter of pure conjecture. It seems to us established by experience that they are capable of doing great harm in typhoid fever, and that their most careful administration in antipyretic doses is not only more dangerous but much less beneficial than the external application of cold. If used at all, they should be employed in small doses, simply for the purpose of hindering the rise of temperature in those cases in which the baths would otherwise have to be given at too short intervals.

Whilst it is not true that the external use of cold will always prevent death in typhoid fever, it does seem to be true that, when used from the beginning with other judicious measures, it will reduce the mortality-rate to three or four per cent. So soon as a patient reaches the temperature of 102° F. he should be freely sponged with cold water, the person being freely exposed to the air whilst the process is being carried on. If the temperature rises to 102.5° F., more efficient hydrotherapeutic measures should be instituted. The least disturbing and the least efficient of these is the cold pack. The naked body of the patient, lying upon a rubber blanket above the sheet of the bed, is to be wrapped in a sheet wrung out of ice-water, and pieces of ice so placed that their outflow shall spread over the sheet and keep it cold. If a fever patient in a cold pack be wrapped in a blanket, the pack becomes a hot one. The cold pack in adults suffering from typhoid fever so generally fails to reduce the temperature sufficiently that it is scarcely worth while to try it; in children it frequently suffices.

Next after it in power is the bath at 80° F., cooled, if necessary, whilst the patient remains in it. Finally, there is the cold bath of 70° F., which will reduce any temperature rapidly. In using the bath the patient must be lifted from the bed with every precaution to prevent movement on his part. The bath should be repeated every two, four, six, eight, or ten hours, according to the necessities of the individual case, the bodily temperature never being allowed to remain above 102.5° F. The temperature of the patient sometimes continues to fall for some minutes after removal from the bath, so that it is usually advisable to take the temperature of the patient in the bath, by the mouth, or, better, the rectum, and to remove the patient when the bodily heat falls to 100° F.

It is essential that hospitals be provided with a movable bath-tub which may be taken directly to the bedside of the patient. In private practice portable tin or rubber bath-tubs are sometimes available. When the cold bath is used the sensations of the patient are commonly very unpleasant; there may be much shivering and excessive complaint of cold. Considerable relief is sometimes afforded by freely rubbing the extremities whilst the patient is in the bath, and in many cases the putting of a hot-water bag to the feet whilst in the bath will give great comfort, and do good by preventing the blood from being altogether driven to the trunk.

The effects of the external use of cold are so decisive that in private practice the practitioner should insist that every obstacle be surmounted. An easy method of using cold is that devised by H. C. Wood. An ordinary cot should be placed at the side of the bed, half opened, and covered with a rubber sheet so arranged that the upper end goes over the head-board, whilst the lower end forms a sort of trough at the foot of the cot, the head of which is slightly elevated. The patient, being wrapped in a sheet, can be readily slid from the bed into the cot, and then by means of a large carriage-sponge soused continuously with cold water, which lies also about the patient and as it accumulates runs off at the bottom of the cot into a tub. R. H. Fitz places the rubber sheet under the patient in the bed, makes a trough of it, and souses with water as required.

Under proper management, instituted early, contra-indications to the use of the cold bath are very rare. Menstruation is not a contra-indication. Pulmonic congestions and pneumonia are largely prevented, and, when they occur, do not forbid the use of cold if the bodily temperature be 103° F. Intestinal hemorrhage and peritonitis are, on the other hand, contra-indications to the use of the bath, as is also extreme exhaustion with feeble heart, such as is seen in persons who have suffered from long-continued high temperature in typhoid: when it is necessary to reduce the temperature in a person with very feeble heart, the latter should be sustained with strophanthus and digitalis, the bath should not be below 80° F., and the extremities should be kept warm as far as possible by hot applications while the subject is in the bath.

Treatment of Symptoms and Accidents.—The medicinal treatment of typhoid fever consists chiefly in meeting indications for the relief of symptoms which may arise from time to time. When in the first days of the attack there is any tendency to excessive furriness of the tongue, nausea, vomiting, or constipation, good results often may be obtained by the cautious administration of calomel (from one-eighth to one-twelfth of a grain) every two to three hours until griping pains or liquid passages are produced. Constipation in the course of the disease is to be met by enemata.

When there is persistent nausea or vomiting in typhoid fever the diet should consist exclusively of two to three parts of milk with one part of lime water, or of milk and carbonic acid water, and, if these fail, of animal broths. In very severe cases no stronger food than wine whey, barley water, or albuminous water (strain white of eggs through muslin and mix with double the amount of water) may be borne, and sometimes it may even be necessary to withdraw for a time all food by the mouth, the patient being sustained by thoroughly peptonized nutrient injections. The drugs which are useful are cocaine (from one-eighth to one-quarter of a grain) ten to twenty minutes before the administration of food; bismuth, with or without minute quantities of calomel according to the individual case, administered when the stomach is empty; and nitrate of silver (one-eighth of a grain) A sinapism or a blister to the pit of the stomach may be of great service.

Unless the diarrhœa amounts to more than three passages a day, it should be rarely interfered with. If the patient fail to have a loose stool every day, especially if there be a tendency to excessive tympanites, sweet oil may be given, or a small injection may be used. For the control of excessive diarrhœa, paregoric, or opium suppositories, may be employed. The mixture of bismuth and carbolic acid (see formula 5) is especially valuable. Rarely vegetable astringents may be used, such as the logwood mixture. (See formula 6.) When the stools are large and very thin, plumbic acetate may be given. Beta-naphtol, with small doses (three grains) of strontium salicylate, sometimes acts favorably.

There is some difference of opinion in the profession as to how far the intestinal ulceration can be affected by remedies. Pepper believes that silver nitrate is useful in this way: it should always be given in pill, with a little extract of opium. The practice originally introduced by George B. Wood of using oil of turpentine in typhoid fever still meets with much favor, and we believe is capable of doing great good. Modern research has shown that oil of turpentine is especially inimical to the typhoid fever bacillus; and, owing to its volatility, it must when freely given fill the whole intestinal tract with its vapor, and influence not only the bacillus but also the process of ulceration. When properly administered the drug is harmless to the patient, and I (H. C. W.) believe it should be given as a matter of routine to every case, unless for some reasons especially contra-indicated, from the middle of the second week until towards the close of the fever, either in capsule or in emulsion, five or ten minims from three to six times in the twenty-four hours. If it disturb the stomach it should be withdrawn. Sometimes during convalescence Peyer's patches are very slow to heal, and keep up a tendency to diarrhœa. Under these circumstances I (H. C. W.) have repeatedly seen the best results obtained by the use of turpentine after the failure of other means. Thymol is strongly recommended by some clinicians in doses of from two to five grains as a substitute for turpentine. Carbolic acid, salol, iodine, and chlorine have all been strongly recommended for the purpose of destroying the typhoid bacillus, but there is no sufficient reason for believing that the course of typhoid fever can be modified by any of these agents. Some of them may act favorably by their local effect upon the alimentary canal. In choosing a remedy the practitioner should always see that the drug selected is incapable of harm. The treatment of typhoid fever at one time in vogue by large doses of hydrochloric or other mineral acid certainly accomplishes nothing so far as the general disease is concerned, and very frequently increases the local irritation.

In all cases of typhoid fever with severe abdominal symptoms and tympany, counter-irritation over the whole surface of the abdomen by means of the spice plaster, or the turpentine stupe, or a cloth wrung out of a tincture of spices, should be practised. The application of the turpentine stupe followed by warm fomentation is perhaps the best method.

When intestinal hemorrhage occurs, opium should be freely used to secure quiet and prevent alarm; absolute rest must be enjoined, and in severe cases the patient should not be disturbed with the bedpan, but allowed to pass the dejecta into a sheet. Ice should be applied over the surface of the abdomen, and the food should be restricted with a severity proportionate to the severity of the hemorrhage. In bad cases albumin water or strong animal broths or essences should alone be given. Styptics are of value if properly employed, but the greatest care should be exercised not to upset the stomach or irritate the intestines with them. According to our own experience, the best styptic is Monsel's salt (not solution), which should be given in double capsule in doses of one-half to one grain, at intervals of from one to four hours. Tannic acid is used by some practitioners, whilst others employ plumbic acetate. Oil of turpentine has some hæmostatic power, and, if the patient have not taken it already, may be used. Oil of erigeron in doses of fifteen to twenty drops every two or three hours may sometimes be advantageously substituted. Extract of ergot (ten grains in capsules) may be of great service: we often alternate it with Monsel's salt in such a way that the patient gets one or the other every two or three hours. In sudden bad cases extract of ergot may be given hypodermically.

Collapse occurring from intestinal hemorrhage must be met with the usual remedies, and transfusion would seem to be indicated in some cases; or the normal saline solution (one-half per cent.) may be injected into the reins or the buttock. (See Cholera.)

Peritonitis in typhoid fever is usually dependent upon perforation, and ends fatally when not localized in the neighborhood of the appendix. Its best treatment is in the use of opium up to continuous mild narcotism and in abstinence from food. When perforation can be diagnosed with fair degree of certainty the question of the performance of laparotomy becomes a very urgent one. Undoubtedly perforation is occasionally recovered from without surgical interference: how often, it is impossible to say accurately, because the diagnosis of perforation can rarely be made with absolute certainty before death. Recovery from peritonitis certainly occurs, as Fitz in 1891 collected twenty-seven recorded cases,—three after operation, seventeen after resolution, and nine after the spontaneous discharge of pus. He further examined the records of ten cases of early operation for perforation with one recovery. According to Osler, the corrected statistics up to 1895, excluding doubtful cases, are seventeen cases with three recoveries, or, taking all the cases, twenty-four laparotomies with six recoveries.

When headache in typhoid fever is severe enough to require treatment, phenacetin or antipyrin may be used very carefully and only in moderate dose; under no circumstances should they be actively pushed, the reliance being upon opium if the pain be otherwise uncontrollable. Ice to the head is often of service; blistering over the back of the neck or to the scalp is to be employed only with the greatest reluctance, especially when there is severe adynamia, since there is always danger of sloughing or other severe local symptom. Nevertheless, when epileptiform convulsions occur, or when the delirium and insomnia are excessive, a blister may be applied to the scalp; it should not be allowed to remain on more than half to three-quarters of the ordinary period, the part being dressed with a poultice if the blister has failed to rise. For the relief of insomnia, opium, trional, or sulphonal may be employed. When there is no pronounced exhaustion, chloral may act most happily, but it must always be given with great caution; ordinarily the best results are obtained by a combination of the narcotics.

In the excessive adynamia of typhoid fever strychnine (one-thirtieth to one-fifteenth of a grain) is a most useful remedy. In severe cases it should be given hypodermically at intervals of four hours, or, better, alternately with cocaine (one-sixth to one-third of a grain) at intervals of six hours (three hours between doses). Digitalis and strophanthus are sometimes useful for sustaining the heart.

In the crisis of typhoid fever, when the failure of vital power shows itself in simple collapse, in a furious delirium, in a high temperature which cannot be controlled except for the moment by the application of cold, or in coma vigil, musk is a very valuable remedy. It should be given in doses of fifteen grains every four to six hours by the rectum in two ounces of starch water, with a little laudanum.

Pulmonic congestion in typhoid fever calls for further stimulation of the circulation, as it is largely the outcome of cardiac and vaso-motor weakness: alcohol, strychnine, cocaine, and digitalis should be given, alternately or in combination. Large doses of extract of ergot (ten grains every two or three hours) may be used for vaso-motor stimulation, whilst oil of turpentine or of eucalyptus and terebene are the best expectorants. Turpentine stupes should be used freely. Ammonium carbonate is given by various practitioners, but its employment in large amounts continuously is of doubtful expediency in a low fever with blood dyscrasia.

TYPHUS FEVER.

DEFINITION.—An acute, contagious, febrile disease, without pathognomonic lesions, characterized by an abrupt onset, great prostration, high fever, profound nervous disturbances, and a macular eruption which usually appears from the third to the fifth day and often becomes petechial.

ETIOLOGY.—Typhus fever is, in all probability, dependent for its existence on some peculiar organism, although such organism has not as yet been isolated. It is endemic in Ireland, Bohemia, the Valley of the Danube, and certain other portions of Europe, and is liable to occur epidemically in any portion of the world. It has been closely connected with famine, overcrowding, and the miseries of extreme poverty; and it has especially abounded in jails, in emigrant ships, and during sieges,—hence the names of *prison fever*, *ship fever*, *camp fever*. It is extremely contagious, the danger increasing enormously when cases are collected in hospitals, under which circumstances the nurses and medical attendants are very prone to suffer. It may be communicated through the wearing apparel, the bedclothing, etc., and persons not suffering from it themselves may become sources of infection. Epidemics may also arise from bales of rags or other similar material which have been gathered in affected districts. It is probable that the poison escapes from the body through all possible avenues, although on this point there is little definite knowledge.

Epidemics are more common and more severe in winter than in summer, probably because of the herding together of people and the lack of ventilation which prevail in cold weather. It is therefore a matter of the gravest importance to provide in every fever hospital wards where patients may be isolated and abundantly supplied with fresh air, whilst the utmost precaution should be taken in the disinfecting of the discharges, of the bed-linen, and of the clothing. (See Typhoid Fever.)

MORBID ANATOMY.—There is no characteristic lesion of typhus fever. According to Murchison, in about two-thirds of the cases the spleen is hyperplastic and softened, and not rarely there are enlargement and softening of the liver. Various secondary lesions of the mucous and serous membranes are common, and almost invariably there is pronounced hyperæmia in the lower lobes of the lung. The blood is dark and coagulates with great difficulty

SYMPTOMATOLOGY.—The period of incubation of typhus fever is usually put down as twelve days, but may be much longer or much shorter. During this stage the symptoms are very slight; the disease in most cases commences suddenly with a chill, followed by an immediate rise of temperature, which may reach 105° or 106° F. on the second day. During the next ten or twelve days there is no remission of the fever, but the evening temperatures are from two to four degrees higher than those of the morning. At the end of the tenth or twelfth day the temperature usually falls, not with the absolute abruptness of a pneumonic crisis, but in the course of two or three days. In fatal cases it is common for death to be preceded by a sudden rise of temperature to a great height, even 108° F.

The general symptoms of typhus fever develop almost as rapidly as the fever; the pulse is full and quick but soft, and only in rare instances dicrotic; from the first it lessens in power more and more as the disease progresses. A constant phenomenon even in the earliest stage is the typhus face, which is characterized by the dark reddish, almost cyanotic tint not only of the face itself, but also of the conjunctiva, and by its heavy, stuporous expression. The tongue, which is whitish and moist, soon becomes dark and assumes a brownish color, corresponding with the ever-increasing sordes about the teeth. A peculiar odor, resembling somewhat that of putrefaction, is given forth from the skin, with the breath, or with the excretions. The violent headache, and the atrocious pains in the back and in the limbs, which mark the onset of the disease, may continue until they are lost in a stupor, which is commonly broken by a low, muttering delirium.

The mental state varies greatly in different cases, or sometimes from day to day in single cases. A wild raging mania may break forth, or an hallucinatory delirium with a never-ending rapid succession of visions, with extreme agitation and emotional excitement, may closely simulate delirium tremens and give rise to attempts at escape, to assaults upon care-takers, who are mistaken for tormenting demons, and even to suicide as a means of escape from haunting melancholia. Rarely the delirium takes the form of a sustained mental effort; the hours will be spent in wild harangue to an imaginary audience upon a religious or other topic.

Usually on the third or fourth day, although sometimes delayed until the eighth, the eruption appears upon the front of the chest and abdomen and rapidly spreads, so that in two or three days it covers the whole body. At first glance it suggests the rash of measles, but when closely studied it will be found to consist of rose-colored spots, which at first disappear upon pressure but soon become petechial, and of a fine, irregular, dusky-red mottling, which looks as if it were beneath the surface of the skin and were seen through a semi-opaque medium. As the disease progresses the rash becomes more and more distinctly hemorrhagic, until it takes the form of small, irregular, petechial patches.

During the second week of the disease there are extreme prostration, a rapid, feeble pulse, subsultus tendinum, carphologia, and, it may be, a tendency to sloughing of the buttocks, heels, and other parts exposed to pressure. Almost invariably bronchial irritation and pulmonic congestion are present in the beginning of a typhus fever, and not rarely they increase until they become a serious element of danger.

Pronounced abdominal symptoms are rare in typhus fever, and when they do occur are to be looked upon rather as accidental than characteristic. The anorexia is complete, but there is generally no active disgust for food. There is no meteorism, and no abdominal tenderness. In some epidemics diarrhœa has been present, but ordinarily the bowels are constipated, the stools being normal in color and consistency. If perchance they are liquid they are usually dark-greenish, never being of the ochre-yellow of the typhoid stool. The spleen may or may not be enlarged, and deep upward pressure will sometimes reveal hepatic tenderness. The urine is scanty and may in somewhat exceptional cases be albuminous, but nephritis is very rare. There is the usual febrile increase of urea and uric acid, with lessening of the chlorides.

Typhus fever is as a disease much more uniform and self-consistent in its course than is typhoid fever, varying chiefly in intensity. It may be so slight that the diagnosis is uncertain; it may be so malignant that the patient dies in profound exhaustion as early as the second or third day, covered with petechiæ from blood-destruction.

The convalescence from typhus fever is usually rapid and free from complication, though it may be interrupted by septic purulent inflammations, such as parotitis and abscesses, or very rarely by paralysis due to neuritis.

DIAGNOSIS.—The abrupt onset, the rapid development of the fever, the peculiarities of the eruption, the absence of abdominal symptoms, the presence of the peculiar odor of the disease, so clearly separate the disease from typhoid fever that in the ordinary case there can be no difficulty in the differentiation. More difficult of separation would seem to be cerebro-spinal meningitis, especially in view of the fact that in some cases of typhus there is early in the disease a wide-spread and very pronounced hyperæsthesia. Under these circumstances it may be necessary for a time to reserve opinion until retraction of the head or other local evidence of basal brain inflammation is distinct. Malignant small-pox may resemble for a time a foudroyant typhus, but is usually early associated with some papular eruption and with hemorrhage from the various mucous membranes. From measles typhus fever is to be separated by the greater severity of the constitutional disturbance, by the absence of conjunctival irritation, and by the fact that the eruption is darker in color, not crescentic, and appears first on the body (not on the face).

PROGNOSIS.—The mortality of typhus fever varies in different epidemics. In nearly eighteen thousand cases treated in the London Fever Hospital during twenty-three years the deaths averaged one in 6.34, but in certain epidemics the mortality has risen as high as thirty or even more per cent. The disease is said to be more fatal in men than in women, and its gravity is greatly increased by the existence of alcoholism, any constitutional feebleness, or previous disease.

The age of the patient is a very important factor in the prognosis. Under five years of age the mortality in the London Fever Hospital has been 67 per hundred, decreasing rapidly, so that between ten and fifteen years it was 10.3 per hundred, and then increasing between fifteen and twenty years to 45 ; from twenty to forty years, 35.3 ; from forty to fifty years, 43.5 ; from fifty to sixty years, 53.9 ; above sixty years, 67.

The prognosis is grave in proportion to the evident severity of the attack ; early extreme adynamia, with muscular tremblings and carphologia, represents a very dangerous condition. Spasmodic, irregular respiration, a pronounced myosis, great violence of cerebral disturbance, paralysis of the sphincters, a very profuse and very dark eruption, coma vigil, early and complete failure of the first sound of the heart,—each and all of these are of most serious import.

TREATMENT.—There is no specific treatment of typhus fever. The management of the case should be upon the same principles as those which have been already discussed in the article on Typhoid Fever. It is the duty of the physician to see in detail that by most careful nursing the patient's strength is saved as much as possible and bed-sores prevented. Alcohol is better borne and should be given in larger quantities in an ordinary case of typhus than in one of typhoid. The bowels should be kept freely open. Food should be given in the form especially of concentrated broths and carefully prepared hashes, eggs, and milk, in as large quantities as can be digested, whilst the various symptoms must be met as they arise. Depressing remedies are under all circumstances contra-indicated.

RELAPSING FEVER.

DEFINITION.—A contagious febrile disease, produced by the presence in the blood of the spirillum (*Spirochæte*) of Obermeier, and characterized by a succession of febrile paroxysms and remissions, each of about six days' duration and recurring from two to four times.

ETIOLOGY.—The immediate cause of relapsing fever appears to be the spirillum which was discovered in 1873 by Obermeier. The contagium is capable of producing the disorder without any predisposing causes. Indeed, it seems doubtful whether much importance can be attached to filth, famine, and overcrowding. The contagium may be communicated directly from person to person or may be carried in various fomites. It seems to be rather less acute and enduring than the contagia of typhus and scarlet fever. Neither age, nor sex, nor race, nor season has distinct etiological influence. In India relapsing fever seems to be almost endemic. Violent epidemics of it are known to have occurred in Europe in the early part of the eighteenth century, and in 1844 the contagium seems to have been imported into North America by emigrants. The latest visit appears to have been in 1869.

The micro-organisms of relapsing fever are extremely delicate, actively motile, spiral fibres, whose length is from three to seven times the diameter of the red blood-corpuscles. They have been detected in the blood only during the paroxysms of fever, but small, glistening bodies which are believed to be their spores have been found during the remissions.

MORBID ANATOMY.—Hyperplasia of the bone-marrow and of the spleen, with cloudy swelling in the liver, kidneys, and heart, and granular degeneration, with the presence of infarcts in various organs, are lesions which have been noted after death from relapsing fever, but which cannot be considered in any way characteristic.

SYMPTOMATOLOGY.—The period of incubation usually is from five to eight days, but in some cases has appeared to be twenty-four hours. The attack begins abruptly, with chill, general aching pains, often with vomiting and vertigo, and sometimes with convulsions. The bodily temperature rises very rapidly, and may reach 106° F. during the twenty-four hours. It is accompanied by a rapid, full, bounding pulse (110 to 120), by free sweating, violent cephalalgia, insomnia, great muscular pains, hyperæsthesia, marked tenderness of the nerve-trunks, and in some cases delirium, especially in alcoholics. There are usually loss of appetite, often vomiting, and not very rarely diarrhœa, though constipation is the rule.

The fever is in typical cases steadily maintained until the fifth or sixth day, when there is an abrupt defervescence, accompanied by a profuse sweating or sometimes diarrhœa. This crisis is sometimes deferred for ten or even more days, and sometimes develops as early as the third day. In persons of feeble constitution subnormal temperature and collapse are not rare.

The convalescence is immediate, the patient getting up at once. On the fourteenth day of the disease there is generally a return of the chill, with abrupt fever and the other phenomena of the paroxysm. This second paroxysm is, as a rule, shorter than the first, and by a series of recurrences the patient may be left profoundly exhausted. Among the symptoms which are often seen in the disease may be mentioned jaundice, epistaxis, hypostatic congestion of the lungs, nephritis, acute enlargement of the spleen sometimes ending in abscess, and very rarely hemorrhage from the mucous membranes. During convalescence ophthalmia, post-febrile paralysis, rheumatoid arthritis, and purulent otitis media have been noted.

DIAGNOSIS.—The diagnosis of a typical case of relapsing fever can scarcely go wrong, but in the beginning of an epidemic and in the early stages of the disease it may be impossible to separate the disorder from an anomalous typhoid, save by detecting the organism in the blood.

PROGNOSIS.—Usually relapsing fever has a death-rate of not over four per cent., but in certain epidemics, and especially when the epidemic has occurred among a people enfeebled by famine and chronic disease, the mortality has been very large, rising as high even as fifty per cent. Prolongation of the pyrexia, symptoms of intense exhaustion, active delirium, convulsions, and the development of any severe complication, are all evidences of danger.

TREATMENT.—There is no known way of aborting a paroxysm of relapsing fever or of preventing its recurrence. Quinine is entirely without influence. The treatment is restricted to careful nursing and feeding and the meeting of symptoms as they arise. Isolation of the patient and rigid disinfection of the clothing are essential to prevent the spread of the disease. The subject should be absolutely confined in bed, not only during the paroxysm, but during the intermission, and be fed freely with milk, broths, and other light food. For the pains in the back, opium, hypodermically or by the mouth, should be used *pro re nata.* Salicylates of all kinds are injurious, and are to be absolutely avoided. The bromides are also too depressing, but chloral, trional, and sulphonal may be carefully used when there is great wakefulness. Alcoholic and other stimulants are to be freely given when there is much prostration. The hyperthermia is much better met by the use of cold baths than by the administration of antipyrin or other remedies of its class. No especial attention should be paid to jaundice when it arises, but gastro-intestinal irritation must be carefully treated. After the crisis active treatment of the collapse with strychnine, cocaine, atropine, digitalis, alcohol, and other stimulants is often essential.

ACUTE INFECTIOUS JAUNDICE. INFECTIOUS FEBRILE ICTERUS. BILIOUS TYPHOID.

DEFINITION.—An infectious febrile disease, characterized by fever, jaundice, splenic enlargement, and, frequently, nephritis.

Under the name of *typhus biliosus* Griesinger observed and described in Cairo, in 1850, an infectious febrile disease, which closely resembles—indeed, is probably identical with—that to which attention was especially directed in 1886 by Weil, and which has been designated as *Weil's disease.*

ETIOLOGY.—This disease occurs especially in summer, and in localities where there is faulty drainage. Thus, in the famous Ulm epidemic it was ascribed to swimming in foul water. It usually, but not always, occurs in local epidemics, and especially attacks young males. In 1892 Neelsen discovered in the bodies of persons dead of the disease a peculiar bacillus; whilst H. Jäger in the same year described as coming from the same source Bacillus proteus fluorescens, and Bosc has found Staphylococcus aureus in fluid taken from the liver by aspiration during life. The etiological value of these organisms is doubtful.

MORBID ANATOMY.—The lesions are those characteristic of infection. A granular, in part fatty, degeneration of the cells of the liver and kidneys, and circumscribed collections of leukocytes, occur in these organs.

SYMPTOMATOLOGY.—The disease is announced by a sudden chill, followed by a rapid elevation of temperature to 104° or 105° F. It remains thus elevated until between the fifth and eighth days, when it falls by steps, the normal temperature being reached between the tenth and twelfth days. In about one-fourth of the cases a recurrence of the fever takes place within the subsequent week, milder than the original attack and lasting five or six days. Headache and dizziness are of early occurrence, and restlessness, delirium, stupor, and prostration follow. Severe pains are complained of in the nape of the neck, the back, and the calves. There are loss of appetite, nausea, perhaps vomiting. Diarrhœa or constipation may be present. Mild jaundice occurs early, and lasts perhaps a fortnight. At the end of the first week roseola, erythema, and herpes are frequent. There may be epistaxis, sore throat, or bronchitis. The pulse varies from 100 to 110 in the early part of the disease, but falls with the occurrence of jaundice, and may become subnormal. The fæces are pale, sometimes colorless. The urine contains albumin, red and white blood-corpuscles, and hyaline and epithelial casts. With the occurrence of the jaundice the liver becomes enlarged and tender and the spleen enlarged.

In severe cases violent hemorrhage may occur from the various mucous membranes, with wide-spread ecchymoses. Herpes, erythema, parotitis, various catarrhs, pneumonia, and peripheral neuritis may develop as complications or sequelæ, and the convalescence is usually so slow that two or three months are required for the restoration of health.

DIAGNOSIS.—The physical and rational signs are indicative of an acute infectious process, in which jaundice is conspicuous and the range of temperature is characteristic. The enlargement and tenderness of the liver and spleen, and especially the occurrence of the disease in small epidemics, are characteristic.

PROGNOSIS.—The mortality-rate has varied in different epidemics or groups of cases from ten to thirty per cent. Death may occur early or late in the disease.

TREATMENT.—The treatment of infectious icterus is that of any general infection,—*i.e.*, symptomatic and supporting.

CEREBRO-SPINAL MENINGITIS. SPOTTED FEVER.

DEFINITION.—A febrile disorder, occurring in wide-spread or local epidemics, characterized pathologically by a cerebro-spinal meningitis and a pronounced tendency to the destruction of the blood, and clinically by a very variable course, during which the most marked symptoms are those of meningitis and hæmic disorganization.

ETIOLOGY.—In its etiology cerebro-spinal meningitis is among the most mysterious of diseases. Its origin cannot be traced to accumulations of filth or other ordinary causes of febrile disease. Most of its outbreaks have occurred over periods of from ten to fifteen years, during which time both Europe and America have been affected without it being possible to detect the travelling of the disease along watercourses or lines of commercial intercourse. The first known period was from 1805 to 1816 ; the second, from 1837 to 1850 ; the next, from 1856 to 1864 ; since which time, however, there have been scattered epidemics of the disease, which, indeed, may be considered to be endemic in the larger cities of the Northern United States. The disease has appeared simultaneously in distant regions without traceable connections, is more abundant in winter than in spring, and cannot in any way be attributed to known peculiarities of the soil or other local causes. It seems, however, to be favored by crowding of individuals, so that it is especially prone to appear amid the misery and poverty of large tenement-houses, and has been very severe among soldiers in garrison towns. It attacks both sexes alike, children more frequently than adults. It is not contagious, the attendants of the sick are very rarely affected, and there is no evidence that the disease passes directly or indirectly from man to man. It is not known to be carried by fomites. Long-continued, excessive labors, whether mental or bodily, seem to predispose to the attacks. Micrococcus lanceolatus, and also the pneumococcus, have been found in the meninges of persons dead of the disease, but reliable bacteriological studies of the epidemic disease are still wanting.

MORBID ANATOMY.—In the apoplectiform cases ending fatally within twenty-four hours there may be little or no visible alteration of the meninges, but the brain is swollen, its convolutions are flattened, and the furrows are obliterated. The characteristic appearances are to be seen towards the second half of the first week and later. They consist essentially in the manifestations of an acute leptomeningitis, serous, fibrinoserous, or purulent. The dura mater is tense, its free surface usually unaltered. The outer surface of the pia mater is also usually normal. The meshes, however, are infiltrated with a more or less opaque yellow, serous, fibrinous, or purulent exudation, which often varies in character in different parts of the brain of the same case. The inflammatory exudation is generally most abundant at the base of the brain and over the convexities. In the former region it fills the space between the optic chiasm and the pons, and is abundant over the cerebral peduncles and on the upper surface of the cerebellum. It may follow the sheaths of the auditory and optic nerves. Over the convexities the exudation forms opaque yellow parallel lines accompanying the injected veins as they overlie the furrows and spreading thence over the cerebral convolutions. Minute hemorrhages may be present in the injected pia mater. Similar appearances are to be seen in the pia mater of the spinal cord, either throughout or in limited portions. The infiltration is usually more extreme on the posterior surface of the cord and in the most dependent portions, especially in the lumbar region.

The brain is pale, the convolutions are flattened, and the ventricles are distended with an opaque fluid from which yellow, viscid clots settle at the lowermost parts, usually the posterior cornua of the lateral ventricles. The ependyma is swollen, soft, perhaps ecchymosed. The choroid plexuses are injected. The inflammatory infiltration of the pia mater is extended along the perivascular spaces into the cerebral cortex, and minute foci of hemorrhagic or suppurative encephalitis may be seen, perhaps with the unaided eye.

If the patient dies during the later stages of the disease the pia mater is thickened and opaque in patches and spots and adherent in places to the brain. It is somewhat discolored from the presence of blood-pigment. The convolutions of the brain may be atrophied and the meshes of the pia mater œdematous.

There are splenic hyperplasia and granular degeneration of the heart, liver, and kidneys. Bronchial catarrh is frequent, and atelectasis and hypostatic and lobular pneumonias are associated. The lymph-follicles of the intestine are swollen. The appearances characteristic of arthritis, endocarditis, pleurisy, nephritis, and enteritis are present when these affections complicate the course of the disease.

SYMPTOMATOLOGY.—The course and symptoms of cerebro-spinal meningitis vary so greatly as almost to baffle concise description. The number of varieties made by clinical writers is very great, but all such varieties everywhere shade into one another; and for the purpose of description it is probably wisest to recognize only the ordinary type, the malignant type, and anomalous forms.

Ordinary Type.—The premonitory symptoms, such as vertigo, prostration, feverishness, or chilliness, may be altogether wanting, or may last from one to twenty-four hours. In young children convulsions may usher in the attack; in adults the first distinct phenomenon is ordinarily a chill, slight or severe, followed at once by excruciating headache, vomiting, vertigo, and a rise of temperature to 101° or 102° F. The pulse is slow, full, and strong; the face is livid, or perhaps pale, with an expression of great anxiety. As the hours go by, the headache increases in severity and becomes associated with a violent backache, as well as with a contraction of the neck-muscles and marked pain when the head is forcibly flexed. The motor disturbances grow more and more decided until the muscles of the back are rigidly contracted, as in tetanus. Trismus is not uncommon; tremors of the muscles may be present; or more frequently tonic or clonic spasms invade the extremities. The muscles of the eye and face do not escape, so that strabismus and facial spasms are frequent. Delirium may follow immediately upon the chill, and is almost invariably an early symptom. It may be wandering, but is often furious and maniacal. Sometimes from the first it is wanting, and ordinarily it gives place in a few days to an increasing stupor.

The special senses are affected; severe tinnitus aurium may be an early symptom; in the advanced stages of the disease deafness is almost universal. Double vision is an ordinary result of strabismus; choked disk and failure of eyesight frequently mark the excessive pressure at the base of the brain. In the last stages sometimes the pulse becomes slow and irregular from pneumogastric irritation, whilst Cheyne-Stokes breathing may result from the disturbance of the respiratory centre.

The symptoms thus far enumerated are in great part a direct outcome of the meningitis, and are not so characteristic of the disease as are the skin eruptions. Herpes, especially herpes labialis, is very frequent and persistent, but the pathogenic eruption is that of petechiæ which cover the whole surface of the body, beginning as small, bright, deep rose or purplish spots or occurring in patches of various sizes, and in severe cases coalescing into great blotches over large territories. This eruption may develop as early as the third day, or it may be put off as late as the eighth. It is a striking feature in some epidemics, whilst in others it is almost altogether absent. Taking all the cases together, it probably occurs in about one-third. In some epidemics it has been replaced or accompanied by urticaria, scarlatinoid or rubeoloid rashes, and even pemphigoid bullæ.

The abdominal symptoms in cerebro-spinal meningitis are not pronounced. Vomiting is usually present in the beginning, and may occur at any stage of the disease. Ordinarily there is constipation. Enlargement of the spleen is sometimes demonstrable.

The average temperature of cerebro-spinal meningitis is distinctly lower than that of most of the serious continued fevers; it may range, even in severe cases, below 100° F.; rarely does it reach above 103° F. It may, however, especially in fatal cases, mount up even to 107° F. Exacerbations of pain are often accompanied by rise of temperature. The differences between the morning and the evening temperature may be very great, but may be wanting. Sometimes the maximum temperature occurs in the morning. The only characteristic features of the temperature curve are its great irregularity and its failure to follow any definite course.

Malignant Form.—There are two chief varieties of malignant epidemic meningitis. In the apoplectic or cerebral type the symptoms are violent headache, rapidly developed delirium with or without retraction of the head, great vital depression, moderate elevation of temperature, and a feeble pulse, which may be slow or rapid. Death in coma may occur within six or eight hours. In the second type there is vital depression, with moderate or high temperature, and the almost immediate appearance of ecchymoses on various parts of the body, rapidly spreading and involving the whole surface in dark purple spots, with clearness of intellect, and death within twenty-four hours. There are cases in which the symptoms of these two typical varieties are intermingled; and every grade of case between the most malignant and the ordinary form occurs.

Anomalous Forms.—Among the anomalous forms which have been described is that which is sometimes known as the intermittent type, in which the fever is remittent or intermittent, with paroxysms which recur daily or every second day. A second variety is that which may be termed the neuralgic or rheumatoid, in which the pains in the legs and arms are extremely violent, accompanied by great hyperæsthesia, and it may be excessive pain on movement, and even by pronounced redness and swelling of the joints. It is probable that in these cases there is a peripheral neuritis, although this has not been demonstrated. There are two abortive forms,—one in which the symptoms are throughout very mild ("walking cases"), and one in which the onset may be furious, with very threatening symptoms and high temperature, which, however, subside after a few hours, or at the most in three or four days, and leave no abiding ill effects.

Another form of the disease is that which has been especially commented upon by Heubner as the *chronic*, in which the course is protracted over many months, with remissions, intermissions, and recurrences of the fever, and with very varying symptoms. In most of such cases there are pronounced evidences of basal meningitis, with great loss of strength and wasting. The symptoms are chiefly maintained by the local disease of the brain and its membrane, and not rarely there is an occlusive meningitis (see page 490) with its concurrent hydrocephalus.

The course of epidemic meningitis is entirely irregular from day to day, and also in its duration. In fatal cases death usually happens from the fourth to the seventh day, but it may occur almost immediately or be postponed beyond a month. Largely owing to the local changes, even in favorable cases, convalescence may be prolonged almost indefinitely, and is usually accompanied by gradually subsiding symptoms of meningitis, not rarely interrupted by relapses. Permanent loss of hearing is very common, producing in young children deaf-mutism. Partial or complete amaurosis is not rare; weakness or loss of memory, general impairment of intelligence, and even chronic hydrocephalus, various local paralyses, disorders of speech, and epileptic attacks, are among the sequelæ of the disease.

DIAGNOSIS.—The diagnosis of cerebro-spinal meningitis is, during an epidemic, ordinarily not difficult. In sporadic cases it is essential to determine the non-existence of tubercle, otitis media, syphilis, or other cause for an existing meningitis which has been recognized. The presence of herpes or of any non-syphilitic skin eruption is usually in favor of the epidemic disease. The symptoms may so resemble those of typhoid fever, or of rheumatism, or of pneumonia with meningeal symptoms, as to require great care on the part of the practitioner to avoid mistake. The shifting irregularity of the symptoms, the peculiarities of the temperature range, the failure of conformity of the febrile and other manifestations to the type of the pneumonia or the typhoid or whatever other disease may be simulated, should excite suspicion; whilst the existence of distinct stiffness in the neck under such circumstances must be looked upon as decisive. A septic meningitis is to be made out by finding the point of infection. The type or form of a cerebro-spinal meningitis may shift during the attack, and pneumonia, or at least pulmonic congestion, may in the beginning be very misleading. We have seen a case diagnosed first as pneumonia and then as rheumatism by excellent practitioners, who ought to have been put upon their guard by the shifting, uncertain character of the symptoms.

PROGNOSIS.—The prognosis is unfavorable in direct proportion to the severity of the symptoms; but should always be guarded, as sometimes apparently mild cases suddenly take on an unfavorable course, whilst recovery may occur out of a condition of apparent hopelessness. The mortality varies in different epidemics from twenty to seventy-five per cent., probably averaging about thirty per cent. Petechiæ, although a serious symptom, are not as unfavorable as are marked evidences of meningeal inflammation.

TREATMENT.—Cerebro-spinal meningitis should be treated in accordance with the general principles that govern the management of infectious fevers. The nursing should be most careful, and the saving of the patient's strength most rigorous; whilst the diet should be simple, nutritious, and up to the digestive powers of the individual patient, milk, animal broths, eggs, oysters, and farinaceous foods being given *pro re nata*.

At one time venesection was largely practised in robust cases, and it has been strongly advised by various physicians. It seems to us, however, distinctly contra-indicated, and we have never seen a case in which its use was justifiable. The local abstraction of blood by means of leeches or cups to the temples or the back of the neck is affirmed by Stillé and others in many cases greatly to mitigate the pain and distinctly to relieve the local disease. Even local blood-letting should be, however, practised with caution, and in our experience its free use is rarely justifiable. Blisters have been largely used and strongly commended. It is plain that the most that can be accomplished by them is relief of the meningeal and cerebral congestion, and that there is danger, if they be used too severely, of producing violent local inflammations, which may, as the dyscrasia of the disease becomes more and more developed, take on a very serious form. Vesication should, therefore, always be superficial, and in order to be effective must cover a large surface. It is to be entirely avoided when the symptoms of breaking down of the blood are pronounced. The best site for the blister is from the nape of the neck upward over the occiput. The continuous application of cold by means of ice-bags to the head and upper spine is of great importance, and probably has as much effect upon the local disease as has local blood-letting or counter-irritation.

In the older epidemics American physicians were accustomed to produce violent diaphoresis for many hours by the use of external warmth, aided by hot infusions of aromatic herbs and other drugs, and many writers affirm that in this way the disease can be aborted. This does not seem in accordance with our present knowledge of febrile disorders, and the practice has been generally abandoned.

As the disease progresses, the symptoms should be met as they arise. When there is high temperature, cold may be employed as in typhoid fever. There is much testimony as to the especial value of opium, for which an extraordinary tolerance usually exists, and which should be, at least in part, preferably given hypodermically in such doses as to maintain a very mild narcotism. Von Ziemssen affirms that morphine is the indispensable medicine in the treatment of the disease. Quinine has also been largely used, but is probably more powerful for evil than for good. Calabar bean, potassium bromide, chloral, ergot, and various medicines acting upon the nerve-centres have been strongly commended, and as strongly condemned. None of them have any definite effect upon the disease, but some of them may aid from time to time in the mitigation of harmful symptoms. Thus, chloral may sometimes be useful in the quieting of excessive spasm or in the obtaining of sleep if insomnia exist. Antipyrin and remedies of its class, if employed at all, should be used simply as quietants, not for the reduction of temperature. In the rheumatoid cases there is a great temptation to administer salicylic acid, but in our experience with sporadic cases it has not been useful ; it does more harm than good. Alcohol is to be employed in proportion to the existence of exhaustion ; laxatives to overcome constipation ; mild diuretics if the renal secretion fail.

INFLUENZA. GRIPPE.

DEFINITION.—A contagious febrile disease, especially characterized by the enormous extent of its epidemics and by the occurrence of local catarrhal symptoms.

ETIOLOGY.—Infectious influenza, although it had almost been forgotten until it broke out in 1889–90, was very fatal during the Middle Ages, having, it is said, in 1580 killed nine thousand persons in Rome, and in 1780 attacked fifty thousand people in St. Petersburg in a single night. It is affirmed to be endemic in the neighborhood of St. Petersburg, and in September, 1889, it began rapidly to advance from the old centres in Russia, reaching Paris by the 26th of November, and thence spreading over the whole civilized world. We believe it to be contagious. It travels especially along lines of water or railroad connections, and has been noted both in Paris and in Philadelphia to form foci in the great retail mercantile establishments, from which it soon is scattered over the whole community. At the same time the disease apparently travels independently of contagion, as ships at sea have entered belts of it, almost the whole crew being stricken down in a night.

The nature of its cause has not been demonstrated, although in 1892 Canon and Pfeiffer discovered simultaneously in the blood and in the bronchial discharges of patients suffering from influenza a very minute bacillus, occurring singly or in chains, which they believe to be the germ of the disease. Neither age nor sex seems to have much influence as a predisposing cause. It is affirmed that the disease spreads to animals ; but this is certainly contrary to what has happened in this country.

MORBID ANATOMY.—In influenza death almost invariably results from local complications, so that the only changes found at the autopsies are those of the complications.

SYMPTOMATOLOGY.—Infectious influenza is a very polymorphic disease ; the varieties depend chiefly rather upon the local manifestations than upon the constitutional symptoms. The onset is usually sudden ; in some cases the attack is ushered in with a distinct chill, in which there may be pronounced delirium or other severe nervous symptoms. Almost invariably there are violent pains in the back, limbs, and extremities, a peculiarly intense weakness, whose severity is out of proportion to the other symptoms, and fever. From this point the cases diverge : five distinct varieties of the disease are met with. In the typhoid form there is a continuing fever, with extreme languor, wide-spread muscular pains, and severe nervous symptoms, such as stupor or delirium, and the rapid development of the dry tongue and general manifestations of a typhoid state; even in these cases, however, there is usually some catarrh or other local complication. The cardiac form of the disease may be considered to be a modification of the typhoid; in it there is an alarming cardiac failure, with very rapid and often broken, feeble, it may be intermittent, pulse, and sometimes with cardiac distress, although the subjective sensations of heart-failure are usually less severe than are the objective symptoms. The third variety of influenza is the more ordinary one, in which there is wide-spread severe respiratory catarrh, involving the whole mucous membrane of the lungs, and having a great tendency to pass into capillary bronchitis or catarrhal pneumonia: coryza, hoarseness or suppression of voice, violent cough, and the ordinary physical symptoms of pulmonary disease may overshadow the constitutional affection. In the gastro-intestinal form, in addition to the constitutional symptoms there is pronounced abdominal disturbance; this usually takes the form of very severe serous diarrhœa, accompanied by very large and frequent watery passages, without vomiting and without pain; in some cases, however, there is much vomiting, and in others pronounced abdominal pain. In the so-called rheumatoid type the pains are exceedingly violent and persistent, and seem to be localized in the muscles and muscular attachments, neither the joints nor the nerve-trunks showing tenderness to pressure or change to the eye.

Although typical cases of each of these varieties are abundant during an epidemic, very commonly the symptoms are so intermingled and changeable that it may be impossible to decide in which category to place the individual case, or the same case may on different days belong in different subdivisions.

The intensity of grippe varies, from a condition in which it is uncertain whether the subject should or should not be considered sick, to the most alarming state. In the mildest cases there is only a slight evening rise of temperature; in the average case the fever ranges from about 101° F. in the morning to 102° F. in the evening; in the severest cases the maximum may be 105° or 106° F. The temperature curve is apt to be entirely irregular, with depressions and elevations, sometimes to be accounted for by the local complications, often, however, without apparent cause. The average duration of the disease is from three to ten days; when there are severe local complications the course may be long protracted, and sometimes, even if there be no settled local lesions, the disease holds on for several weeks. In some cases there is a sudden ending with a sort of crisis; more commonly the patient passes into convalescence so gradually that it is impossible to say when the disease should be considered to have ended. The convalescence from even the mildest influenza is apt to be exceedingly slow; the pains and local disturbances are often very obstinate, but much more enduring are the weakness and inability not only for physical but also for mental exertion, which may last for months.

COMPLICATIONS.—The great danger in influenza is from the occurrence of complications. Of these the most frequent and most fatal are the pulmonary. Extensive catarrhal pneumonia, with symptoms of great oppression, pale cyanotic countenance, violent cough, and high fever, is common. The sputum is apt to be copious and dense, but the characteristic rusty sputum of true fibrinous pneumonia is exceptional. The pneumonia diplococcus and streptococcus can usually be found. Pleurisy with large serous or more rarely purulent effusion is not rare. Pericarditis occasionally develops, and, in very bad cases, pneumonia, pericarditis, and pleuritis may coexist. Among the rarer complications may be mentioned purulent inflammation of the middle ear and of the conjunctiva, or even of the eye itself. Furuncles, localized abscesses, peripheral neuritis, and various skin eruptions, such as herpes, urticaria, roseola, etc., are occasionally developed. Confusional insanity of melancholic or maniacal type may follow the attack.

DIAGNOSIS.—Although the symptoms of a mild influenza cannot be distinguished from those of a simple cold, during the prevalence of an epidemic of the disease the diagnosis of influenza is the safer, and is justifiable in all cases resembling the typical disease. The chief symptomatic difference between an infectious influenza and a climatic catarrh is that in the influenza the malaise and disturbances of temperature precede the local disease and are entirely out of proportion to its extent.

PROGNOSIS.—The prognosis in influenza, so far as ultimate recovery is concerned, is good, provided the subject be not debilitated and proper care be taken to avoid complications. When such complications arise the prognosis becomes grave in direct proportion to the gravity of the local disease.

TREATMENT.—In treating a case of influenza it must be remembered that four-fifths of the deaths from the disease occur from exposure, and that the slightest influenza is a serious disease, requiring confinement to bed and careful nursing. In the majority of cases the attack can be greatly and immediately ameliorated by free sweating, which is best produced by a mixture of aconite, antipyrin, and pilocarpine. (See formula 7.) A dessertspoonful of this may be given, the patient put in a bath of 105° F. for ten minutes, taken out, and given a strong, hot toddy with a teaspoonful of the mixture, the latter to be repeated in half an hour if sweating does not come on. For those individuals in whom morphine does not produce after-depression one-eighth to one-quarter of a grain of the alkaloid may be added to the mixture. The sweating may be induced in various other ways: hot toddy and Dover's powder, aided by the hot bath, will usually suffice, and is preferable when there is pronounced cardiac weakness. In the ordinary case of influenza all that is required after the sweating is the use of moderate doses of quinine, five to ten grains a day, and the administration of cocaine and strychnine in tonic doses. The food should be nutritious and simple, and given at intervals of not more than four hours. Small quantities of alcoholic drinks given with food are often very beneficial. The greatest care must be exercised in allowing a patient to go out when convalescence begins, as the liability to catarrh may continue for an almost indefinite period.

In gastro-intestinal influenza the food should be at once reduced to boiled milk slightly thickened with flour, animal broths with or without egg, toast, and tea. Opium should be used freely; bismuth with carbolic acid, or aromatic sulphuric acid diarrhœa mixture (formula 6), or a chalk mixture (formula 8), may be used. Chlorodyne or a mixture of chloroform, camphor, and an aromatic oil is sometimes very serviceable. (See formula 9.) Slow, long-acting counter-irritation over the abdomen by means of weakened mustard or spice plasters should also be employed.

The pulmonic catarrhs are to be treated as they would be if arising from other causes, but the type of the disease is always distinctly adynamic. Tartar emetic should never be given, but if secretion be not free the potassium citrate mixture with apomorphine may be used; after this ammonium chloride sometimes acts well; but usually, after free secretion has been established, the best results are obtained with oil of eucalyptus or with terebene. In obstinate catarrhs, guaiacol, compound tincture of benzoin, or syrup of garlic may be essayed. Free, persistent, and extensive counter-irritation is always valuable. High temperature rarely needs treatment, because it is not long sustained. If, however, 104° F. is reached, sponging or the tepid bath may be employed, and if these fail of effect, cold baths may be given. Phenacetin and antipyrin are sometimes serviceable as antipyretics, and also as quieting pain and other nervous disturbances, but under no circumstances should they be given in large dose,—not over fifteen grains in twenty-four hours. When there is heart-failure, alcohol (in moderation), strychnine, cocaine, tincture of strophanthus, and tincture of digitalis are to be employed. Sometimes it is necessary to give very large doses, and when there is a tendency to vomiting or the symptoms are alarmingly acute, cocaine and strychnine may be used hypodermically. We have also seen life apparently saved by the hypodermic use of tincture of digitalis (ten to twenty minims).

DENGUE. BREAK-BONE FEVER.

Definition.—An epidemic, contagious fever of subtropical countries, characterized by violent muscular and articular pains and a polymorphous rash.

Etiology.—Epidemics of dengue have been noted in subtropical Asia, Europe, and America. J. W. McLaughlin states that he has found a peculiar micrococcus in the blood. The disease is immediately contagious. An extraordinary feature is the fact that usually four-fifths of the whole population exposed take the disease.

MORBID ANATOMY.—Death from dengue being almost unknown, there is no knowledge of lesions produced by the disease.

SYMPTOMATOLOGY.—After a period of incubation varying from some hours to five days, dengue commences abruptly with very severe aching pains, headache, and a more or less pronounced chill, followed by fever, which usually reaches its maximum during the first twenty-four hours. In severe cases there are rapid pulse, general adynamia, and even nocturnal delirium. Loss of appetite is universal; mucous or bilious vomiting is very common. Very frequently there appears almost at once an erythematous rash, which may invade the mucous membrane, producing redness and swelling of the conjunctiva, of the internal nares, and of the throat. Both large and small joints are affected, and often become swollen and red by the third or fourth day. In from forty-eight to sixty hours a rapid defervescence occurs, often accompanied with critical phenomena, such as colliquative sweat, diarrhœa, and epistaxis. At this time, in a large proportion of the cases, develops the so-called secondary or terminal rash of the disease, which is characterized by its polymorphism. It may be papular and circumscribed, or papular and diffused; or it may be vesicular or pustular. More frequently, however, it is an exanthem which resembles that of measles or that of scarlet fever, or is like an urticaria. Several forms of the eruption may exist in the same case. Enlargement of the lymphatic glands is not uncommon. A secondary fever may follow the eruption and gradually subside.

Abortion frequently results in pregnant women, but death from the disease is almost unknown, except in very young infants, who sometimes die in coma and convulsions. Convalescence may be very slow and protracted, with long continuance of the muscular and articular pains, and also of the glandular swellings.

Desquamation follows the secondary eruption, and may be complete in six days, but may be protracted to from ten to fifteen days. As no known agent is capable of distinctly modifying the course of dengue, the treatment must be purely symptomatic. If phenacetin and antipyrin fail to control the pains, opium should be used.

THE PLAGUE.

DEFINITION.—An extremely contagious, usually epidemic disease, especially characterized by an eruption of boils and the formation of buboes.

In the Middle Ages the plague was of frequent occurrence in widespread disastrous epidemics. At present it seems to be confined to certain districts in Asia and Africa. In 1881 there was a very severe epidemic in Mesopotamia, and in 1894 one in Bagdad; whilst China still suffers.

Kitasato states that during the Hong-Kong epidemic in 1894 he found in the blood, glands, and viscera of the patient a short bacillus having rounded ends, whose pure cultures produced, when inoculated into animals, a series of symptoms like those of the plague in human beings. This bacillus he believes to be the cause of the disease, and to be capable of invading the human body through the respiratory and digestive tracts, and also through wounds.

An attack of the plague begins suddenly, with intense headache, vertigo, dilatation of the pupils, great anxiety and general depression, ending, in from a few hours to some days, in a violent chill, followed by high fever, with excessive prostration, vomiting, rapid pulse, accelerated breathing, complete anorexia, severe abdominal pain, and not rarely diarrhœa. Great nervous disturbances, delirium, coma, bronchial catarrh, serious hemorrhages, excessive adynamia, and violent depression of the heart, may, in this stage, end in death. If the patient survive, after two or three days of fever buboes will appear and proceed rapidly to suppuration and ulceration; whilst, not universally, but very generally, boil-like pustules will break out upon the extremities, perhaps ending in local gangrene. In favorable cases from the eighth to the twelfth day a slow convalescence sets in. The mortality of the ordinary forms of the plague is about ninety per cent. In malignant cases death may take place during the first day. The only characteristic lesions are the bubonic tumors, which always have their origin in lymphatic glands.

The treatment of the plague must be purely symptomatic. It is doubtful whether the course of the disease can be in any way modified for the better by human agency.

DIPHTHERIA.

Definition.—A highly contagious disease, characterized by fever, usually by a pseudo-membranous inflammation of the pharynx, and often by symptoms of a toxæmia due to the presence of a specific bacillus whose growth produces the poison which is absorbed.

Although epidemics, endemics, and sporadic cases of severe sore throat of a probably diphtherial nature are mentioned in early medical writings, it was Bretonneau, in 1821, who first included under the term *diphthérite* the disease now known as diphtheritis or diphtheria, and applied this term in virtue of the presence of a false membrane, *διφθέρα*.

Etiology.—Diphtheria occurs in all countries, at all seasons, but particularly during the colder months of the year. Its extension is favored by the crowding of people in limited quarters, and especially by the presence of large numbers of children in schools. McCollom has recently emphasized the fact that the number of cases is much greater when the schools are in session than during the summer vacation. Predisposing causes are to be found in exposure to faulty hygienic surroundings, especially filth, dampness, and poor ventilation. McCollom, however, states that imperfect drainage and insalubrious conditions are not important in increasing the frequency of diphtheria, this disease having been found more prevalent in localities in which there was no fault to be found with hygienic conditions than in sections where the reverse was the case. Children, especially the young, are more prone to the disease than adults. Sucklings are rarely affected. Persons debilitated from whatever cause, and those liable to recurring attacks of sore throat, are especially susceptible.

The immediate cause is universally admitted to be the bacillus discovered by Klebs in 1883 and obtained in pure cultures in 1884 by Loeffler, who demonstrated its pathogenic importance. This bacillus, the Klebs-Loeffler or diphtheria bacillus, is a slender rod, usually slightly bent in the middle, its extremities club-shaped and tending to become more deeply stained than the other parts. The bacillus inclines to form groups of two to five lying parallel. It is usually from 2 to 3 μ long and from 0.5 to 0.8 μ broad. It is thus nearly as long and twice as broad as a tubercle bacillus. It thrives in milk and grows readily upon the mixture of blood serum and bouillon recommended by Loeffler, colonies being formed in the incubator in the course of twelve hours before any considerable growth of associated bacteria has taken place. When kept in darkness in a moist state it lives for months. Its vitality may be preserved for a number of weeks in a dried state, as in fragments of diphtherial membrane, but it is destroyed by exposure for half an hour to a temperature of 140° F. This resistance of the bacillus to atmospheric influences satisfactorily explains the occurrence of sporadic cases of diphtheria and their occasional origin in rooms in which careful efforts at disinfection have been made. Great variations in its virulence exist. Bacilli resembling the diphtheria bacillus in every respect except in being non-virulent are sometimes found on normal mucous membranes. These have been called the pseudo-diphtheria bacillus, but recent investigations favor the view that this bacillus is an enfeebled, non-virulent diphtheria bacillus, whose vitality and virulence can be restored under suitable conditions. The diphtheria bacillus is present upon the inflamed mucous membrane in all cases of diphtheria, and may be found months after convalescence from the attack, although usually disappearing in the course of two or three weeks. We are informed from the bacteriological laboratory of the Harvard Medical School that of nine hundred and nine cases of diphtheria examined during four successive months there were one hundred and sixteen in which diphtheria bacilli remained at the end of two weeks. Of these the bacilli were present in sixty-three between two and three weeks, in thirty between three and four weeks, in eleven between four and five weeks, in seven between five and six weeks, in four between six and seven weeks, and in one between seven and eight weeks. The bacilli may be found upon the conjunctival and genital mucous membranes and upon the wounded surfaces of persons suffering from diphtheria. They also may be found upon the cutaneous wounds of persons who have not been exposed to this disease, and, in rare cases, upon the mucous membrane of the throat of healthy persons. In the dissemination of diphtheria the important element is the transfer of the bacillus from one person to another. Persons harboring the diphtheria bacillus may be free from the disease, but may transfer the virulent or non-virulent bacillus which may produce the disease in a second individual.

The diphtheria bacillus is at times to be found among the domesticated animals, especially in dogs, cats, cows, and fowl. In cats and dogs the associated symptoms are loss of appetite, cough, and emaciation. The possibility of the transfer of this disease from such animals and from the use of infected milk, as reported by Klein, is to be recognized.

The diphtheria bacillus is usually stained by Loeffler's solution of methylene-blue,—thirty parts of a saturated alcoholic solution of methylene-blue in one hundred parts of a solution of caustic potash in water (1 to 10,000). After staining for from three to five minutes, wash in water. Hunt's method is favored in the Harvard bacteriological laboratory in those cases in which the bacilli present are of a doubtful character, or in which there are many cocci and but few bacilli. This method decolorizes other bacteria than the diphtheria bacillus and deeply stains the club-shaped ends. The prepared cover-glass is stained for thirty seconds in a saturated solution of methylene-blue. It is then washed in water, dried, and placed for a few seconds in a ten per cent. solution of tannic acid. After being again washed and dried, the cover-glass is placed for a few seconds in a saturated aqueous solution of methylene-orange. After being finally washed and dried, the specimen is to be mounted. Gram's method is to be employed for the study of diphtheria bacilli in the tissues.

MORBID ANATOMY.—The sore throat in diphtheria may be of a catarrhal, a pseudo-membranous, or a gangrenous character. The catarrhal inflammation alone may exist, and the pseudo-membranous and gangrenous inflammations are usually associated with the catarrhal variety. The presence of the diphtheria bacillus is the only characteristic by means of which the diphtherial nature of the process is to be absolutely determined. In the *catarrhal* inflammation the mucous membrane of the tonsils, uvula, soft palate, and pharynx is swollen and of a dark-red color. Its surface is at times covered with a mucous layer, which is sometimes opaque from the presence of abundant leukocytes. The tonsillar crypts may contain opaque white or yellow material not projecting above the surface, and consisting of cells, granules, and bacteria. The appearances above described are not to be distinguished from those occurring in non-diphtherial varieties of sore throat and in lacunar or follicular forms of tonsillitis, except by the bacteriological examination.

The *pseudo-membranous* sore throat of diphtheria affects the regions above described, but extension to the upper surface of the soft palate, to the nostrils, pharynx, and larynx, even to the trachea and bronchi, occurs. In this variety the bacillus destroys the superficial epithelium and promotes exudation from the blood-vessels, in consequence of which false membranes are formed. These first appear as spots or patches of a gray or grayish-white color, oftenest on the tonsils, and thence spreading by continuity and contiguity eventually form a membrane which may cover the tonsils, soft palate, and uvula, as well as extend into the adjoining parts. The older the false membrane the more likely is it to become discolored by blood, medicines, or food, and then it may assume a yellow, green, or brown color. A distinction is drawn by Virchow between a *fibrinous* and a *diphtheritic* inflammation of the mucous membrane in diphtheria. The former is rather membranous than pseudomembranous, and is more often found in the larynx, trachea, bronchi, and nose than in the pharynx. It may occur in the air-passages in diphtheria either alone or in association with the other anatomical varieties of inflammation. It may be found alone in the nose in membranous rhinitis, in which affection the diphtheria bacillus is often present. This membrane is easily separated from the underlying mucous membrane, and is made up largely of clotted fibrin and cells. The diphtheritic false membrane of diphtheria more rarely extends to the larynx or the nose, and is intimately attached to and forms a part of the inflamed mucous membrane, attempts at removal resulting in the tearing of superficial portions of the mucous membrane. In severe but not fatal cases healing is attended with ulceration and scars. Microscopical examination shows that the diphtheritic false membrane also is composed of a net-work of clotted fibrin, but it is intimately connected with the mucous membrane, in which there is a fibrino-cellular infiltration, and its superficial portions are necrotic.

In the gangrenous condition following diphtheritic inflammation, the *putrid sore throat* of earlier writers, the necrotic tissue becomes infected with other bacteria, and putrefaction results. Hemorrhages are frequent in gangrenous diphtheria, and sloughing of the tonsils and of the palate may occur.

The pharyngeal inflammation may extend into the Eustachian tubes, while its continuance is frequent into the larynx, trachea, and bronchi. Above the vocal cords the false membrane is intimately adherent to the mucous membrane, but below these cords it is generally but loosely attached, and often lies upon the inflamed mucous membrane. In the trachea it is apt to form a hollow cylindrical cast of this tube. The same is true of the larger bronchi, while its extension into the smaller bronchi is generally in the form of solid cylinders. The lungs are usually distended, injected, and contain numerous patches of lobular atelectasis and nodules of broncho-pneumonia. If stenosis of the larynx occurs, emphysema of the lung, either intra- or extra-alveolar, is likely to be found.

The lymphatic glands beneath and behind the lower jaw are enlarged, soft, and on section of a reddish-gray color. Pericardial and pleural ecchymoses are frequent. The heart is of an opaque gray or grayish-yellow color, and the muscular fibres may show extensive fatty degeneration. Acute endocarditis sometimes, though rarely, occurs. The spleen is enlarged, its consistency diminished, and on section the pulp is increased and of a reddish-gray color. The kidneys are enlarged, the capsule readily detached, the surface at times speckled with extravasated blood. On section the cortex is swollen, the region of the convoluted tubes opaque. On microscopic examination the epithelium may be found necrotic or in a state of fatty degeneration, Bowman's capsules thickened, and the nuclei in the glomeruli increased. Hyaline casts are to be found in the tubes. The liver shows the appearances characteristic of a parenchymatous degeneration. Diphtheritic patches are sometimes to be found upon the lips, tongue, cheeks, œsophagus, and stomach. The solitary follicles and Peyer's patches in the small intestine are swollen, and the mesenteric lymph-glands enlarged and injected. The brain may contain spots of red softening, probably due to embolism, and the lesions characteristic of a neuritis have been found in the peripheral nerves. Among the rarer complications are descending retropharyngeal abscesses involving the tissues around the œsophagus and in the anterior mediastinum, abscesses of the lung, suppurative pleurisy, pericarditis, and arthritis. Diphtheritic inflammation of the conjunctiva, vulva, and vagina, and of wounded surfaces, especially in case of tracheotomy, are sometimes found.

Frosch and others, most recently Wright and Stokes, have sought for the diphtheria bacillus in other parts of the body than the inflamed throat. It has been found extensively, but only in small numbers, it being necessary to use comparatively large portions of the parenchyma and blood upon the culture media to secure a growth. It has been in this way obtained from the lungs, liver, spleen, brain, and bronchial and cervical lymph-glands, and from the blood from the heart. The toxin of the diphtheria bacillus is considered to be especially destructive to cell-life, and the necrosis of cells in remote parts of the body, as well as of those in the inflamed pharyngeal mucous membrane, is regarded as the effect of this toxin. The combined effect of the diphtheria bacillus and of associated cocci is especially dangerous. Disturbances due to the diphtheria bacillus are instituted in the place of its growth, while those from other organisms, especially the streptococcus and the staphylococcus, result from their invasion of the interior of the body by means of the lymphatics and the blood-vessels or air-passages.

In the false membranes of diphtheria streptococci may also be present in combination with the diphtheria bacillus. Especial importance is to be attached to the presence of the former in explanation of some of the severe complications of diphtheria, and in accounting for the unfavorable results from the use of antitoxin. This agent, it is asserted, though capable of neutralizing diphtherial toxins, is not known to oppose the action of pyogenic cocci.

SYMPTOMATOLOGY.—The symptoms of diphtheria may arise in the course of two days or more after exposure. The earliest symptoms are usually fever and pain in swallowing. The fever may be preceded by a chill or chilliness. The temperature rises from 102° to 104° F., and the dysphagia may be slight or considerable. The higher the fever the more probable the occurrence of headache, backache, loss of appetite, and weakness. The examination of the throat may show no other change than the redness and swelling of inflamed tonsils and pharyngitis. The especial feature suggestive of diphtheria is the formation of a false membrane. This appears first as grayish-white spots or patches, often formed in the course of a few hours and rapidly increasing in size. It is to be remembered that diphtherial patches may be present on the upper surface of the soft palate, or in the pharyngeal pouches, hence invisible without a mirror, and the case be one of diphtheria. As the disease progresses, the spots and patches coalesce, and extend to the soft palate and the uvula, and in persons with large tonsils, especially children, the voice becomes thick, the lymph-glands behind the jaw moderately swollen and sensitive, especially on the side corresponding to that showing the more advanced inflammatory changes, and the urine is likely to contain a small or large trace of albumin. The attack may be mild or severe, a change from one variety to the other often taking place unexpectedly. In mild diphtheria there is but little general disturbance. The appetite may remain good, prostration slight, the fever quickly disappear, the membranes become thinner and smaller, thus fading away, and in less than a week the patient be well.

The severe cases of diphtheria are characterized by conspicuous septic symptoms. The severity of the attack may be evident from the first, or the graver symptoms may develop in a few days in apparently mild cases. The temperature may be high, 104° F., at the outset, or, as graphically shown by Heubner, rise on the third or fourth day. It may, however, be only moderately elevated, or even subnormal. The pulse is rapid and weak, corresponding to the range of temperature. The patient may be delirious, but usually is in a condition of indifference or is drowsy. There is no appetite. Vomiting and diarrhœa are frequent. The breathing is slow, perhaps rapid and noisy, and the voice is hoarse. A thin red or yellow acrid discharge flows from the nostrils, producing sores or crusts upon the lips. The mouth is usually open, the tongue dry and fissured. The false membrane is found throughout the pharynx either diffused or in patches, and forms a thick opaque yellow crust resembling wash-leather. In the gangrenous cases the pseudo-membranous patches become green or brown, are moist and shreddy, and of a very offensive odor. Hemorrhages are frequent and sometimes considerable from the mouth and nose, and may also occur in the skin. The glandular swellings behind and under the jaw become greatly increased, and the resulting deformity is enhanced by œdema of the surrounding fibrous tissue. There is marked albuminuria, and hyaline casts are present.

Severe cases of diphtheria progress on the one hand with suffocative symptoms, on the other with those of septicæmia. Mild cases of diphtheria may be attended with suffocative symptoms, and a fibrinous laryngitis may be the sole significant lesion in diphtheria, diphtherial croup, or laryngeal diphtheria, the Klebs-Loeffler bacillus being found in the pharynx. *Croupous* symptoms may develop suddenly or gradually. The respiration becomes more noisy and labored. A frequent dry barking cough is present. Paroxysms of dyspnœa may occur and the face become purple, relief being brought about by the expulsion of pieces of fibrinous membrane from the larynx or trachea. If relief is not obtained, the patient becomes anxious, the skin pale and moist, the extremities cool, the pulse weak, and death occurs sometimes from acute suffocation, sometimes from prolonged asphyxia. The latter condition prevails in those cases in which the fibrinous inflammation extends into the bronchi. When the symptoms of septicæmia are prominent, the prostration of the patient is extreme, and swallowing is difficult, largely due to paralysis of the soft palate, perhaps necessitating the use of the stomach-tube. The pulse is weak and irregular, and the heart-sounds are faintly heard. Death may occur suddenly and unexpectedly from cardiac paralysis even during apparent convalescence.

COMPLICATIONS.—The complications occurring in diphtheria are both early and late, and are especially met with in the severe cases. The early complications consist in extension of the pharyngeal inflammation to the middle ear and of the fibrinous inflammation to the air-passages, and the production of broncho-pneumonic nodules by the inhalation of food and of bits of membrane. Abscesses of the lung and pleurisy may occur. Retropharyngeal, periœsophageal, and mediastinal abscesses may result from infection from the throat. Serous or purulent inflammation of the joints is at times found. Such lesions are usually indicated by an elevation of temperature in addition to localizing symptoms, as pain or swelling. Most important of the late complications is paralysis, the result of a peripheral neuritis, which generally occurs about the third week after the acute symptoms of the disease. The soft palate is oftenest affected, then the muscles of the eye, especially those of accommodation, and less frequently the muscles of the larynx, trunk, and extremities, and the diaphragm. The muscular paralysis may occur in cases of diphtheria so mild as not to excite suspicion of this disease. The affection of the palate is indicated by a nasal voice and the escape of liquid from the nose during attempts to swallow. Strabismus or weakened eyesight gives evidence of the ocular paralysis. Aphonia, or a hoarse or whispering voice, indicates the affection of the vocal cords, while inability to sit upright or to support the head indicates paralysis of the muscles of the trunk, and an ataxic gait and absent patellar reflexes give evidence of the affection of the nerves of the legs. Adolf Baginsky calls especial attention to the importance of recognizing paralysis of the diaphragm, which is indicated by dyspnœa, a sunken abdomen, and suffocative attacks. The sensitive nerves also may be affected in consequence of a neuritis. Severe cerebral symptoms, as spasms, hemiplegia, and coma, may be due to diphtheria, and when occurring are to be regarded as the result of a focal encephalitis of embolic origin. The nephritis present as a late complication of diphtheria is represented by the persistence of the albuminuria and casts, but is usually insufficient to produce uræmic symptoms or dropsy, speedy recovery being the rule.

A rare manifestation of diphtheria is membranous or fibrinous rhinitis. It is characterized by the presence of an opaque white membrane upon the mucous membrane of the nostrils, as a rule associated with but little symptomatic disturbance except anæmia and debility, but often persisting for weeks or months. Abbott and others have found virulent diphtheria bacilli present in the membrane, and some, if not all, of such cases are to be regarded as nasal diphtheria, dangerous to others, if not harmful to the patient.

Diagnosis.—The diagnosis of diphtheria ultimately depends upon the discovery of the Klebs-Loeffler bacillus in a case of sore throat. Its presence should be suspected until disproved in cases of apparently simple catarrhal tonsillitis or pharyngitis during the occurrence of epidemics of diphtheria, and especially after known exposure to a case of diphtheria. According to Heubner, it is impossible in certain cases of diphtheria to make a positive diagnosis in the first two or three days without a bacteriological examination. All cases of membranous sore throat, whether appearing as lacunar tonsillitis, fibrinous or diphtheritic tonsillitis, or pharyngitis, are to be regarded as diphtheria until the bacteriological examination has denied the presence of the specific bacillus. Cases of fibrinous laryngitis, so-called croup, are usually of diphtherial origin even in the absence of pharyngeal symptoms, and should be regarded as laryngeal diphtheria unless directly attributable to the inhalation or application of irritants or unless bacteriological examination has shown the absence of the diphtheria bacillus. Cases of membranous rhinitis are to be regarded as nasal diphtheria unless the absence of the Klebs-Loeffler bacillus has been demonstrated. A diphtheritic conjunctivitis and a diphtheritic inflammation of wounds should lead to a search for the diphtheria bacilli: their presence in such cases would demand the isolation of the patient, that others might not incur the risk of infection. The appearance of a false membrane is insufficient for the absolute diagnosis of diphtheria. A pseudo-membranous inflammation of the throat, including the tonsils, soft palate, and uvula, may occur in scarlet fever, measles, typhoid fever, and in other infectious diseases. The membrane may present no physical characteristics by means of which it is to be distinguished from that occurring in diphtheria. The bacteriological examination in such cases shows the presence of streptococci, perhaps of staphylococci and other bacteria, but no diphtheria bacillus. To such a condition the term *pseudo-diphtheria* or *diphtheroid* has been applied. Morse examined four hundred cases of inflammation of the throat occurring in diphtheria and scarlet fever and found the Klebs-Loeffler bacillus present in sixty per cent. of the cases. Welch states that in his experience in not more than five per cent. of the cases in which the clinical diagnosis of diphtheria was well established were the Klebs-Loeffler bacilli lacking. Of five hundred and fifty-eight cases examined by Heubner in which the presence of bacilli was suspected in virtue of the stage and course of the disease and the localization of the exudation, in only seven were they not found.

The diphtheritic pharyngitis in scarlet fever most closely resembles that occurring in diphtheria. The fever is higher and more continuous. The degree of swelling is greater, the extension to the Eustachian tube is more constant, and the characteristic eruption of this disease soon makes its appearance. The possibility that the diphtheria bacillus may be present in the scarlatinal sore throat may be considered, but Booker examined bacteriologically the secretions from the throat in twenty-three cases of scarlet fever and found streptococci in all, the diphtheria bacillus being absent. Park found that most cases of false membrane limited to the tonsils of adults were not diphtheria, that most cases of acute pharyngitis with little or no membranous exudation were not diphtheria, and that the majority of uncomplicated cases of membranous laryngitis were diphtheria. In young children the tonsillitis accompanied with exudation limited to the crypts or extending beyond them, whether there was much or little false membrane, might or might not be due to diphtheria. He also found in the milder cases of membranous pharyngitis as a characteristic of diphtheria irregular patches of adherent false membrane on the tonsils or margins of the faucial pillars.

For the recognition of the Klebs-Loeffler bacillus special training in bacteriological methods is obviously necessary. The importance of making cultures especially from doubtful or suspicious cases of sore throat is such that laboratories for this purpose are rapidly becoming established at accessible points in various parts of the country. The larger cities and towns offer gratis the necessary facilities. Test-tubes containing the culture medium, carefully packed in metal boxes which carry the necessary directions, are to be had at various centres of distribution.

PROGNOSIS.—The mortality from diphtheria varies within wide limits during successive years and in different localities in the same year. The statistics of such mortality are only of relative value, owing to the difficulties inherent in the satisfactory classification of the cases under consideration. In a recent communication by Welch on the treatment of diphtheria by antitoxin, it is stated that the mortality at the surgical clinic in Berlin during ten years varied between 58.5 and 43.2 per cent. In the London hospitals the variation in certain years was between 29.3 and 40.7 per cent. Of seven thousand one hundred and sixty-six patients recently tabulated by Welch as having been treated with antitoxin, the mortality was 17.3 per cent. Mason states that in Boston the mortality from diphtheria from January to May, 1895, during the use of antitoxin, was 14 per cent., against 31 per cent. in the previous year. According to Heubner, in 1894 the mortality in thirteen hundred and thirty-two patients with diphtheria in the Berlin hospitals was 38.8 per cent., while of fifteen hundred and thirty-four cases treated with antitoxin the mortality was 19 per cent. Baginsky between March 15, 1894, and March 15, 1895, treated five hundred and five children with antitoxin, with a mortality of 15.6 per cent. In August and September antitoxin was not used, and one hundred and twenty-six children were treated, with a mortality of 48.4 per cent.

The prognosis in any case of diphtheria is always doubtful, since the mildest cases may become severe and the severe cases may improve. The prognosis is especially grave among children and in proportion to their youth; sucklings, however, are rarely affected. The prognosis is more severe among patients debilitated by previous disease or faulty hygienic surroundings. The outlook in the individual case is worse as the disease becomes septic or gangrenous, since a mixed infection then exists. Convalescence from the mild cases of diphtheria usually occurs towards the end of a week, while in septic cases the disease may continue for a period of two or three weeks. Gangrenous diphtheria is usually fatal in the course of a week. The range of temperature is less indicative of the degree of toxæmia than are the extreme prostration, swelling of the lymphatic glands, offensive discharge from the mouth and nostrils, and abundant albuminuria. The prognosis becomes grave when there is extension of the inflammatory process to the larynx and lungs, or when the action of the heart becomes weak and irregular. It is to be remembered that patients, especially children, may suddenly die from cardiac paralysis, generally due to extensive fatty degeneration of the heart, even weeks after apparent convalescence. Nephritis is rarely sufficient to serve as a cause of death, the symptoms of this complication ordinarily disappearing in the course of a few or several weeks after recovery from the acute symptoms of the disease. Diphtheritic paralyses are usually recovered from unless respiration and circulation are conspicuously affected, and especially when there is paralysis of the diaphragm.

TREATMENT.—In the treatment of diphtheria it is desirable that the patient be thoroughly isolated in a room from which have been removed carpets, unnecessary furniture, and other articles capable of acting as fomites. The utmost care should be exercised to see that the urine, the fæces, and especially all discharges from the mouth and nose are at once thoroughly disinfected. (See Typhoid Fever.) The room should be well ventilated, and kept at a temperature of about 70° F., with the air thoroughly moistened by means of a steam atomizer, boiling tea-kettle, or other device. The inhalation of balsamic vapor is thought by many to be useful, and we have ourselves apparently gained advantage by heavily impregnating the air about the child with the oil of eucalyptus, diffused by means of the steam atomizer or by simple boiling. No one should enter the room except the nurses and the medical attendants, who should take the greatest care to avoid personal infection from the discharges and also the infection of others by carrying the poison upon their clothing. Thus, the doctor should put on a linen duster, apron, or other similar garment whenever he comes to the patient. Deaths have occurred among doctors and nurses from lodgement of a piece of infected mucus in the eye or upon some abrasion during the local treatment.

The tendency of the disease is usually towards exhaustion. The child should be kept as quiet as possible, and during early convalescence should not be too much encouraged to play with toys. Whenever there are signs of cardiac failure the horizontal position should be rigidly enforced. The strength should be sustained by feeding at intervals of from three to five hours with nutritious, easily digested food, in as large quantities as can be borne. Owing to the difficulty of swallowing, milk, eggs, soups thickened by stirring in them ordinary mashed potatoes, tapioca, sago, or other starchy materials, milk toast, and other liquid or semi-liquid foods, must be chiefly relied upon; but sweetbreads, birds, and similar food should be used on occasion. The tendency not only to exhaustion, but also to depression, is always so great that alcoholic stimulants should usually be exhibited from the beginning of an attack. At first they should be given with the food and in moderate doses; in the latter stage of the disease they should be exhibited with the greatest freedom both with the food and at other times.

The objects of the local treatment of diphtheria are the controlling of the inflammatory action of the throat, the cleaning out of débris and material capable by its putrefaction of producing poisonous substances, the removal of the membrane, and the destruction of the diphtherial organism. Experience has shown that the only way of controlling the inflammatory action of the throat is to destroy the cause of that action, —namely, the bacillus: so that at present the attempt to subdue inflammation is reduced to the use of ice, or more rarely of hot applications, which, at least, often serve to relieve discomfort. The ice should be continuously applied, by means of especially prepared india-rubber bags, Leiter's tubes, or other receptacles, to the swollen glands and tonsillary region, whilst the patient should be encouraged in the free use of cracked ice, for which often may be substituted with advantage frozen milk, ice-cream, frozen beef-essence, frozen chicken jelly, or other frozen foods.

For the cleaning out of the débris of the throat or nose salt water may be used, but certain solutions are preferable. Among these the most important is the official solution of dioxide of hydrogen, which may be applied (from twenty-five to fifty per cent.) every three or four hours to the pharynx by means of the mop, or may be used diluted with four or five times its bulk of water by the atomizer. It is capable of irritating and even causing ulceration of the mucous membrane, but in our experience we have never seen it do harm. If it seem too strong for any individual throat, it should be further diluted. When it causes irritation, atomization with a saturated solution of boric acid with thymol (gr. ii to f℥i) often soothes the throat.

In nasal diphtheria it is especially important that the passages be thoroughly cleansed, and, if possible, disinfected, at short intervals. Lime water, simple solutions of salt, the one per cent. solution of carbolic acid, saturated solution of boric acid, and diluted Loeffler's solution have been variously recommended by authorities. We have used ten per cent. solution of sodium sulphite with satisfaction. In adults or children the nostrils may be cleaned out by throwing the spray of an atomizing syringe into the posterior nares through the throat. Usually the better way is to introduce the nozzle of an atomizing syringe horizontally into the external nostril and to give the injection with such freedom and force that, if it be possible, it shall work its passage through the other nostril.

Two methods of destroying the membrane are conceivable. One is by astringent substances which shall cause such coagulation and shrinkage as to bring about removal; the other is by solvents. Early in the attack some practitioners use a very strong solution of silver nitrate. Various preparations of iron have a tendency to shrivel the mucous membrane, and are also germicidal. They have been much used. Perchloride of iron has been especially employed in various forms. We have often used Monsel's solution as equally efficient and less irritant than the chloride. As solvents of the false membrane, animal ferments, such as pepsin and trypsin, and vegetable ferments, such as papain, have been very highly commended from time to time. They are entirely safe, produce little or no irritation, and are therefore harmless, but their effectiveness is a matter of great doubt. Lime water has been much used; but probably the most powerful available solvent of false membrane is lactic acid. Lennox Browne highly recommends the application of the pure acid to the throat by means of a dense swab of absorbent cotton with sufficient firmness to detach the membrane at its edge. The usefulness of both astringents and solvents is much limited by the fact that complete destruction of the false membrane (were it possible) would not affect the bacillus in the underlying mucous membrane.

Various germicides have been used, in the hope of destroying the bacillus. In the first stages of the disease the small spots of membrane may be carefully touched with a concentrated carbolic acid; later, glycerin containing from three to five per cent. of carbolic acid may be used upon large, diffused surfaces. *Loeffler's solution*, composed of ten grammes of menthol diluted in thirty-six cubic centimetres of toluol and added to four cubic centimetres of liquor ferri sesquichlorati (Br. Ph.) and sixty cubic centimetres of absolute alcohol, has been much used abroad, applied in full strength by the swab or diluted by atomization. Corrosive sublimate, one part to one thousand, is employed by some practitioners. The biniodide of mercury is preferred by others because it does not precipitate serum albumin, and is, therefore, more apt to penetrate the membrane: it is also less apt to undergo decomposition. Mercurial preparations should be applied by means of an atomizer of some sort, and a known quantity of the mercurial should be thrown into the throat, so as to avoid any possibility of giving too much of it, especially when calomel or corrosive sublimate is being given for its general effect.

The result of our own experience in diphtheria has led us to believe that the thorough cleansing of the throat at short intervals with dioxide of hydrogen is a very valuable part of the treatment, and that, though such use may be followed by a more pronouncedly germicidal application, the latter is of doubtful value, especially if mercurials are being employed internally. If calomel be administered in a dry powder, or corrosive sublimate in a not too dilute solution, it diffuses over the throat before entering the stomach, so that whatever local action it is possible to get from the mercurial is obtained. This also applies to the iron preparations.

There is no known specific drug in diphtheria. Potassium chlorate has no control over the disease, and in no way supports or increases the resistive power of the system. It is largely eliminated by the saliva, and when taken internally exerts a continuous influence upon the diseased parts, but is not sufficiently bactericidal to be of value. When given in large doses it very greatly increases the danger to life by its action upon the kidneys, which is parallel with that of the disease. If it be used it should therefore be given in dry powder in very small doses. We do not think that tincture of chloride of iron can be considered as having any influence upon the bacillus of the disease or upon the products which it forms in the blood; it would seem, however, to be indicated by the rapid destruction of the red blood-corpuscles, which is a part of the diphtherial process; and its influence upon the kidneys is beneficial. Its use is also sanctioned by a wide-spread belief in the profession that it is of value. If used at all, it should be given with glycerin and water at short intervals in such doses as the stomach will easily bear. During the last few years the mercurials have been very largely employed in the treatment of diphtheria. Some practitioners prefer the corrosive chloride; others vaunt the value of calomel and soda rubbed together; others prefer simple calomel. It probably makes very little difference which one of these preparations is selected. We have been accustomed to use calomel, which, for the reason already stated, should be given in dry powder. In the beginning of an attack from one-quarter to one-half grain may be administered every two hours until free purgation is produced, and when the constitutional depression is not great the very free administration of the drug late in the disorder seems, at times, to aid in loosening the false membrane. If corrosive sublimate be preferred, one seventy-second of a grain may be given every two hours at two years of age; at six years of age one-fortieth of a grain; at ten years of age one-thirtieth of a grain. Many practitioners, however, employ much larger doses than this; Jacobi recommends half a grain of the bichloride during the day for an infant one year old.

Pilocarpine has been employed to a considerable extent for the purpose of causing free secretion in the mouth and throat and thereby loosening the membrane. Its action for good is, however, very uncertain, and it may greatly increase the danger by provoking so much secretion into the bronchial tubes as to interfere with respiration.

As stimulants in diphtheria, alcohol, strychnine, digitalis, and strophanthus are often of great value.

Sometimes in diphtheria dangerous nasal hemorrhage occurs. It can often be controlled by pushing up the nostril a small roll of absorbent cotton saturated with a fifty per cent. dilution of the official solution of dioxide of hydrogen, or by astringent injections, but may necessitate plugging.

When the temperature reaches 102° F. in diphtheria the patient should be well sponged with cold water. If this fail to reduce the fever the cold pack or bath should be employed. The temperature of the bath should be started at about 90° F. and reduced as low as 70° F. if necessary. It is important, however, that the treatment be no more severe than is absolutely required for the reduction of the temperature, and when the thermometer in the rectum or in the mouth indicates 100.5° F. the patient should be taken out of the bath. In feeble cases it may be wise to apply the hot-water bag to the feet during the bath.

When in a case of diphtheria cyanosis and restless insomnia are combined with evident labored breathing and marked retraction of the lower ribs and at the supraclavicular spaces and the suprasternal notch, either intubation or tracheotomy becomes necessary. According to the statistics of Henry R. Wharton, intubation gives better results than tracheotomy in children under two years of age; after this the results are about the same. (For the method of performing these operations the reader is referred to works upon surgery.)

Feeding by means of a tube may be necessary during the acute stage in consequence of the dysphagia resulting from pharyngeal pain and obstruction. A soft rubber catheter passed through the nose may then be found more convenient than the stomach-tube introduced through the mouth. In the stage of paralysis the stomach-tube may be required to prevent regurgitation of food through the mouth or nostrils.

Antitoxin Treatment.—In 1890 Behring and Kitasato published their first article upon the use of the blood serum of artificially immunized animals in the treatment of diphtheria. After the third publication in 1892 the subject attracted wide-spread attention, and became a matter of clinical investigation by Roux and others.

It has been demonstrated that in the lower animals diphtheria can be cured with antitoxin. How the antitoxin acts still remains uncertain, but the probabilities favor the theory that it influences the living body in such a way as to render the cells tolerant of the toxin. Nevertheless, the chemical theory that the antitoxin directly neutralizes the toxin still has advocates. The antitoxin has no direct bactericidal effect, although it arrests the spread of the local inflammation and the growth of the bacillus, probably by preventing the tissues from being so poisoned by the toxin that they are unable to resist the bacillus. It is proved that it requires a definite quantity of the antitoxin to neutralize the effects of a definite quantity of toxin.

In using antitoxin in human medicine it is, of course, impossible to know how much toxin is present in the individual patient. The dose of the antitoxin is, therefore, always uncertain and empirical: the older the patient, the longer the duration and the greater the intensity of the disease, the larger the dose required. Certain untoward effects may follow its use; rarely a local abscess is formed, but diffused erythema, rheumatoid swelling of the joints, general urticaria, and albuminuria have been noticed in a number of cases,—effects sufficiently serious to make it wise to repeat the small or moderate dose of antitoxin, if necessary, rather than in the beginning to give an overwhelming amount.

Anti-diphtheric serum may be of various strengths, but the unit of dose generally received is that inaugurated by Behring: this unit is one cubic centimetre of the so-called normal serum, which is of such strength that one cubic centimetre will overcome ten times the minimum dose of diphtheric poison fatal to a guinea-pig. The ordinary dose of the serum, which should be injected into the buttock or flank, is sixty antitoxin units. If by the next day there has been no marked improvement, one hundred units may be given. In very severe cases, or when the patient is not seen until late in the disorder, from one hundred to one hundred and forty units may be administered at the first dose. In successful cases the effects of the serum are apparent within a few hours in the subsidence of the fever, the slowing of the pulse, and the reduction in the severity of the local symptoms. Inside of twenty-four hours the membrane should begin to disappear.

Although the exact power of the antitoxin treatment can hardly be considered to be determined, yet certainly its value has been so far proved that it should be used in every case of diphtheria with as much positiveness and determination as quinine would be employed in malaria. In the statistics collected by Welch, embracing many thousands of cases, the mortality was reduced by the use of antitoxin about half. In the Paris hospitals, from 1880 to 1889, the yearly average of deaths from diphtheria was 1840. In 1890 there were 1668 deaths; in 1891, 1361; in 1892, 1403; in 1893, 1266; in 1894, 1009; and in 1895, 435. The total death-rate thus fell after the introduction of the serum treatment to about one-fourth of what it had been for many years, and to one-third of the average for the previous five years. Nevertheless, it is certain that the serum treatment frequently fails. Some of the reasons for such failure are, however, obvious. In the first place, antitoxin cannot remedy damage already done to organic cells and tissues, so that an injection late in the disease, though it may put an end to the diphtherial process, may not prevent death. In the second place, in most serious cases there are two infections, a primary one,—that with the diphtherial organism,—and a secondary one,—that with various streptococci and other pathogenic germs which follow upon the diphtheria. An antitoxin treatment may put an end to the first infection, but death may result from the secondary infection, over which the antitoxin has no direct influence.

It is evident that the earlier the treatment with antitoxin the greater the chances of recovery. Very few fatal cases are on record in which death has occurred when antitoxin was properly administered during the positively determined first day of the disease. In eight hundred and fourteen cases reported collected by Welch, in which treatment was begun before the third day, only a very little over five per cent. ended in death. Indeed, it would seem from these statistics that when the serum treatment is begun on the third or fourth day the mortality is thirty-six per cent. greater than in cases treated on the first or second day, and three and a quarter times less than in cases treated after the fourth day. In our opinion, the practitioner should at once begin the antitoxin treatment whenever the clinical features of the case warrant the diagnosis of diphtheria, without waiting for the confirmation of this diagnosis by bacteriological methods.

There is no reason for believing that the antitoxin has any direct sedative influence upon the heart or irritative influence upon the kidneys, and certainly by arresting the diphtherial process it has great tendency to prevent complications and secondary effects. In laryngeal diphtheria with stenosis, requiring operation, there is sufficient accumulated experience to show that the serum is a very valuable agent in preventing the progressive development of the false membrane in the small tubes, and that in many cases in which intubation would be otherwise insufficient the antitoxin treatment does away with the necessity of tracheotomy.

The value of antitoxin as an immunizing agent has not been clinically determined, although guinea-pigs may be rendered completely immune. One attack of diphtheria does not protect from a second, and it therefore seems incredible that a permanent immunity can be obtained in any artificial way. The exact immunizing dose has not been agreed upon: Behring considers it to be two hundred antitoxin normals, but Rosenthal affirms that half this quantity is sufficient when the exposure is simply that of coming into the neighborhood of the infection. Behring directs that the dose be repeated in eight weeks. Whilst there is so much doubt concerning immunization, it seems to us that the better plan is to watch exposed cases and inject the serum when the first symptoms of the disease appear.

WHOOPING-COUGH.

Definition.—A contagious disease, especially attacking children, characterized by violent paroxysms of coughing, with spasm of the glottis, and respiratory catarrh.

Etiology.—Whooping-cough is endemic in most large cities, but occurs especially in epidemics, in which it probably spreads by contagion from child to child. The exact nature of the poison is not entirely determined. Letzerich asserts that he was able to produce the disease in animals by insertion of the sputum into the trachea, and believes the germ to be a micrococcus. Deichler affirms that it is an amœboid protozoön. According to Afanassiew, it is a short bacillus (*bacillus tussis convulsivæ*), pure cultures of which, when applied locally, cause in the lower animals respiratory catarrh. The fact that occasional cases have been reported in which the disease was congenital would indicate that the poison, whatever its nature may be, is capable of causing the affection by inoculation, and that therefore whooping-cough is not necessarily primarily a disease of the respiratory tract, due to the local presence of an organism; although it is probable that in whooping-cough, as in diphtheria, the germ usually first finds a local lodgement. Subjects who are especially liable to pulmonary catarrhs are certainly very susceptible to the whooping-cough poison, but of all predisposing causes youth seems to be the most active. Whilst the disease is rare in infants under six months, more than half the cases occur in children under four years of age, and attacks are common up to six, but rare after ten, and exceedingly rare in adults, although sometimes occurring in old age. Recurrences are occasional. The wide-spread belief that the disease is more frequent in girls than in boys is of doubtful correctness.

Morbid Anatomy.—The characteristic change in whooping-cough consists of a catarrhal inflammation of the respiratory mucous membrane, which, according to the studies of Meyer-Hüni and Von Herff, is most severe in the nose, larynx, and trachea, although it may extend into the small tubes. The so-called *cough region*, which is supplied by the sensitive filaments of the superior laryngeal nerve,—namely, the posterior wall of the interarytenoid region,—seems in most cases to be the chief focus of disease. The most frequent secondary lesions are capillary bronchitis and pneumonia. Enlargement of the tracheal and bronchial glands is so common that the theory has been supported by able clinical pathologists that the disease is essentially a bronchial adenopathy. This certainly is not correct, and it is probable that the frequency of these glandular enlargements at post-mortems depends upon the excessive fatality of whooping-cough in strumous children. Although albuminuria is frequent, severe nephritis is very rare.

SYMPTOMATOLOGY.—The period of incubation in whooping-cough is usually from three to four days, but it may be as short as forty-eight hours, or may extend beyond the week. In accordance with general custom, three stages of the disease may be recognized, if it be understood that these stages are artificial divisions and pass insensibly one into the other. The first or catarrhal stage usually comes on insidiously. In the beginning the symptoms are those of an ordinary cold, but in a short time a distinct tendency to nocturnal exacerbations and to paroxysmal coughing may be noticed. Suspicion also should be aroused by the fact that the cough is much more severe than would seem to be called for by the very slight physical signs presented, and by the absence of distinct disorder of voice. Fever is often wanting, and when present consists chiefly in a rise of temperature towards evening. If, however, a pulmonary catarrh develop, the fever may be severe. The stage lasts in most cases about two weeks. Sometimes it may be shortened to three days or even less. More frequently it is prolonged even up to six weeks. As a rule, the younger the child the shorter the catarrhal stage.

As the second stage is reached the coughing becomes more purely paroxysmal, and finally is attended with the distinctive "whoop." In a violent paroxysm, the child, moved by a warning sensation, ceases its play, runs to its care-taker or catches hold of some object for support, and is immediately seized with a series of short, rapidly repeated, explosive, expiratory coughs, without any respiration between them, and with an increasing turgidity and cyanosis of the face, which may continue until the whole countenance is dark and swollen, with prominent eyeballs, protruding mouth, watery eyes, and seemingly imminent suffocation. Then the spasmodically closed glottis partially relaxes, and a deep inspiration occurs, accompanied by a loud crowing or whooping sound. This may be followed by a return of the cough, with the whoop at the end of it. In very severe cases subconjunctival, nasal, or even tracheal hemorrhage may occur, and involuntary urination or defecation occasionally happens. Very frequently the paroxysm is cut short by violent vomiting, attended with a free expulsion of ropy mucus from the respiratory tract, and when severe it may be followed by complete exhaustion. The paroxysms vary in frequency as they do in intensity. There may be six or there may be eighty in the twenty-four hours. They are always much more severe and frequent at night, and are liable to be excited by singing, shouting, or any act which irritates the larynx. The patient's general condition is usually good between the paroxysms, unless the latter are so severe or attended with so much vomiting as to interfere with the taking of food or with sleeping at night. There is generally fever in proportion to the severity of the attack. The urine is sometimes saccharine, and is very frequently albuminous. Any râles which can be detected in the chest are due to complicating catarrh.

The duration of the second stage is generally about four weeks, but varies from two to seven or even more weeks. The terminal stage may be considered to begin when the symptoms show signs of distinct amelioration. Its duration is extremely variable; it may last a single week or as much as eight weeks: the average is about a month. The symptoms are those of the second stage, with a progressive amelioration, the paroxysms and other attending phenomena becoming not only day by day less severe, but also more infrequent. Long after the whoop has disappeared, and when the cough is scarcely even paroxysmal, the recurrence of pulmonary catarrh from exposure or other cause may bring back typical paroxysms.

Almost invariably there may be found during the acute stage of whooping-cough a superficial grayish-yellow ulceration of the frænum of the tongue, which probably is the result of the mechanical irritation of the part by the lower incisor teeth during the violent efforts of coughing.

Complications.—The most important and fatal complications of whooping-cough are inflammations of the respiratory tract. Wide-spread broncho-pneumonia is common, and even in the most favorable cases runs a very slow and dangerous course. Atelectasis is not infrequent in young, weakly, or rachitic children. Emphysema is often developed, but very rarely remains after the disease passes by. A paroxysm may end in a convulsion; the convulsion may be purely functional, but it may be due to a rupture of a meningeal or other cerebral vessel, and be followed by hemiplegia, aphasia, or other evidences of focal organic brain disease. In such cases epilepsy, spastic paralysis, aphasia, imbecility, blindness, or similar loss of function may be the result of a permanent brain degeneration. Rachitic or tubercular tendencies are much intensified by whooping-cough.

Diagnosis.—The nature of those cases of whooping-cough in which the disease is so slight as never to go beyond the catarrhal stage can only be inferred from the occurrence of the attack during an epidemic of the disease. Before the development of the whoop the nocturnal and paroxysmal character of the cough should awaken strong suspicion. A single whoop usually settles the diagnosis; but it must be remembered that a severe complicating pulmonary catarrh may prevent the occurrence of the whoop, and also that a whooping-cough may insensibly, so far as the symptoms are concerned, pass into a tuberculosis of the lungs.

Prognosis.—Pertussis is usually regarded as a disease of comparatively little importance, but in feeble children it is a dangerous affection, to be treated with great care. According to T. M. Dolan, in London it causes one-fourth of the deaths among children. In any individual case the prognosis depends chiefly upon the original condition of the child and the care given during the illness.

PROPHYLAXIS.—Isolation and disinfection are as important and powerful in suppressing the contagion of whooping-cough as of all other diseases of the class, but, probably because some cases are improved by being taken into the air, the disease is continually met with not only in public places but in public vehicles. The contagion may not only be communicated directly, but may be carried in fomites.

TREATMENT.—The hygienic treatment of whooping-cough is of the utmost importance. The chief peril to life lies in the probability of pulmonary inflammations; but experience has abundantly demonstrated that the confinement of children in even well-ventilated apartments has a distinct tendency to aggravate the symptoms, so that very great judgment is often required in the obtaining of out-door air without exposure. In summer the child should be out in the air the whole day when the weather is fine; in winter out-door exercise should be confined to dry, still days on which the temperature is not too low. Winds are even more dangerous than damp. In some cases the best results are to be obtained by the use of large apartments with very free ventilation. The food should be easily digested, palatable, and nutritious. The whole tendency of the disease is towards exhaustion. The vomiting may make it difficult for the child to obtain sufficient nourishment, so that frequent feeding is often judicious. It is essential that at night the child wear warm underclothing, at least on the body and arms, in addition to the night-wrapper. As in many cases the patient is not frequently seen by the physician, the immediate care-taker should note the temperature at least twice—better, three times—a day. Any increase of fever should be viewed as a danger signal, it being almost always an early indication of developing pulmonic catarrh. In advanced whooping-cough the greatest benefit is sometimes obtained by change of air, especially to sea air.

The general medicinal treatment is naturally divided into that which is directed against the nervous elements of the disease, that which has to do with the catarrh, and that which has to do with the general support of the system. Mild cases may progress satisfactorily without medication, but usually not only the demands of patients for medicine but the frequency of the paroxysms and the catarrhal irritation of the mucous membrane may be sensibly modified by the administration of emulsion of asafetida in very large doses at short intervals. Almost all drugs which act upon the nervous system have been given in pertussis; tincture of belladonna is, on the whole, the most generally useful, but must be given in sufficient doses to cause slight dryness of the mouth or dilatation of the pupil in order to get its full effect. It also acts better when given by atomization, so as to have its local benumbing effect upon the larynx. Antipyrin and phenacetin are very valuable drugs for checking the frequency and severity of the paroxysms; they are usually well borne, especially phenacetin, and may be given in ascending doses until some relief is obtained or some disagreeable symptom is caused. Acetanilid is probably as efficacious, but more dangerous. The bromides are sometimes of service: the best of them is ammonium bromide; it may be frequently given with great advantage at the same time as antipyrin and belladonna, but it is usually much better not to give the drugs in a single prescription, so that the dose of one can be altered without affecting that of the others. Bromoform has been very highly recommended, but we have had no experience with it, and late clinical reports are not favorable: from two to four drops of it may be given to a child three years of age three or four times a day. Chloral is a very useful remedy, whose administration should be reserved for the late evening, to get its hypnotic as well as its anti-convulsive effect; frequently it may be advantageously combined with opium.

Great benefit is often derived from local treatment. Belladonna has already been spoken of, and spraying the larynx with a one per cent. solution of cocaine may in some very severe cases be serviceable. If there be coryza, the nostrils should be kept clear by washing them out with warm salt and water, or with the official peroxide of hydrogen diluted with ten times its bulk of warm, slightly saline water. Saturating the air of the room with steam from an atomizer or with water from slaking lime often does good. Sometimes benefit is derived from using in the steam atomizer a two per cent. solution of carbolic acid or of thymol.

During convalescence the system should be built up as much as possible by the use of cod-liver oil, tonics, nutritious food, and change of scene.

MUMPS. EPIDEMIC PAROTITIS.

DEFINITION.—A contagious, febrile disease, characterized by inflammation of the parotid gland.

ETIOLOGY.—Although mumps usually occurs in the form of an epidemic, and especially prevails in the spring and autumn, it certainly spreads by direct contagion, and probably also by means of fomites. The nature of the virus is unknown. Nursing infants are extremely insusceptible, and adults are not very often attacked, whilst late childhood and early adolescence are the ages most susceptible. Males are said to be affected more frequently than females. One attack of the disease gives immunity, provided both glands have been affected. Cases certainly occur in which the mumps attacks only one parotid, and in which years afterwards a second attack is confined to the previously unaffected gland. It would look, therefore, as though the immunity were a local affair.

MORBID ANATOMY.—The changes in the glands consist of hyperæmia, serous infiltration of the acini, and a catarrhal inflammation of the ducts.

Resolution is usually complete, though permanent enlargement of the glands may occur.

SYMPTOMATOLOGY.—The period of incubation is from one to three weeks. The first symptoms are swelling and pain just below the ear on one side, with a slight fever (100° F.) and some malaise. The swelling rapidly increases, passes forward, backward, and downward, and may involve the submaxillary gland. In one or two days the opposite side usually follows. There is much pain on the attempt to open the mouth, so that chewing is greatly interfered with; whilst deglutition and even speech become difficult. In favorable cases the symptoms subside in from seven to ten days, with a rapid convalescence.

Mumps, though usually a trivial affection, may be a severe one, especially in scorbutic, tubercular, or otherwise broken-down subjects. In some cases the prodromes are pronounced, and during the attack there are high fever, vomiting, rapid pulse, delirium, and great prostration. Partial loss of hearing is not rare, and sometimes there is much earache. Such symptoms usually subside with the attack, but are liable to leave permanent impressions.

A frequent and curious complication is an involvement of the sexual glands. Occasionally in girls the mammæ or the ovaries become the seat of the irritation and are swollen and painful. In males who have already passed through puberty orchitis is very frequent. The left testicle is said to be most frequently attacked, but either or both glands may be affected. There is marked swelling, with, at times, effusion in the tunica vaginalis, and sometimes a subsequent atrophy, which very rarely affects more than one testicle. Vulvo-vaginitis or urethritis sometimes occurs.

DIAGNOSIS.—The only difficulty of diagnosis in mumps consists in distinguishing between a parotid and a lymphatic swelling. Probably the best test is the existence of a point of intense tenderness high up in the angle of the jaw immediately behind the auditory meatus.

PROGNOSIS.—It is doubtful whether death ever occurs from mumps in a previously healthy subject unless after exposure and consequent secondary complication; but it is stated that there are epidemics in which meningeal symptoms and even death are frequent.

TREATMENT.—A laxative, confinement of the patient to bed or to a warm room, and a light, liquid diet are usually all that is necessary. If there be distinct fever, aconite and antipyrin fever mixture may be given. Hot local applications are generally grateful and may be used freely, but when there is high inflammatory action cold compresses or iced poultices may be preferable, and even leeching may be practised. Rubbing the glands with a belladonna-mercurial ointment (equal parts) is efficacious when resolution is slow. In typhoid cases appropriate support and stimulants should be given. If orchitis occur, absolute rest in bed should be enforced, the scrotum well supported, the belladonna-mercurial ointment used, and, when the tenderness has subsided, strapping employed.

ERYSIPELAS.

Definition.—A contagious febrile disease, due to the presence of a streptococcus which produces at the point of inoculation a peculiar spreading inflammation, usually accompanied with much serous exudation.

Etiology.—The contagion of erysipelas consists of the streptococcus originally described by Fehleisen under the name of *Streptococcus erysipelatis*, but now generally believed to be identical with *S. pyogenes.* As this organism has been found repeatedly in phlegmonous suppuration, in ulcerative endocarditis, in puerperal endometritis, and even in angina with false membrane, it is clear that these diseases are closely related to erysipelas. The organism occurs in chains, is non-motile, and is not known to form spores. By inoculation of its cultures Fehleisen produced typical erysipelas in man. There has been much discussion as to the relation of so-called idiopathic erysipelas to surgical erysipelas, or erysipelas of wounds. We believe that in medical erysipelas the organism finds some crack, excoriation, or abrasion in which it effects a lodgement, and that all erysipelas is the result of an inoculation at the position of the first outbreak.

In the development of erysipelas predisposing causes are of great importance. The disease is rare before puberty, and still less frequent in the very old. Certain individuals and even certain families are exceedingly susceptible to the poison ; chronic alcoholism, Bright's disease, excessive poverty with its attending hardships, and other influences which lower human vitality, are predisposing causes. Recently delivered women are particularly prone to the disease. The contagion is usually not very virulent, but it can be conveyed by a third person, and may lurk in the furniture or on the walls of an apartment. Under special circumstances not understood the poison of erysipelas becomes endowed with great virulence and reproductive power, resulting in epidemics which are especially prevalent in the spring months.

Occasionally erysipelas recurs at comparatively short intervals. In such cases it has been rendered extremely probable by the researches of Leroy de Lille that the micro-organisms remain in the body in a latent state.

Morbid Anatomy.—Erysipelas is essentially an infectious lymphangitis, either superficial or deep-seated, and the anatomical alterations are both local and general. In superficial erysipelas the local changes are essentially of a microscopical character, since the redness and swelling observed during life rapidly disappear after death. A furfuraceous desquamation of the epidermis, blisters, pustules, scabs, or sloughs may be present. On microscopical examination of hardened, stained specimens of the reddened skin, the superficial lymphatics and their radicles, the juice-canals, are found to be filled with the erysipelas-coccus. The blood-vessels are distended with blood, and the surrounding tissue is infiltrated with leukocytes.

In deep-seated or phlegmonous erysipelas the subcutaneous tissue is infiltrated with a fibrino-serous and cellular exudation in addition to the presence of pyogenic cocci. There results a yellowish, gelatinous or brawny appearance of the tissue, the color of which may be more or less opaque. Necrosis of the inflamed tissue is likely to occur, and the skin may be undermined by gangrenous abscesses which tend to break through the surface.

General changes indicative of the infectious nature of the process are the swelling of the spleen and the granular degeneration of the heart, liver, and kidneys. Chains of bacteria and bacterial emboli may be found in the capillaries of various organs. Inflammation of the serous membranes, especially of the endocardium, sometimes occurs.

SYMPTOMATOLOGY.—The incubation period is very various, but has been assigned as from three to seven days. The local outbreak may or may not be preceded by malaise, headache, and vague prodromes, but is usually ushered in with an initial chill, which is sometimes very severe, and is followed by a rapid rise of temperature. The dermatitis most frequently appears near the nasal angle of the eye, or in the immediate neighborhood of the nostrils, or in the crease of the cheek which runs from the nose to the chin. It may, however, first develop in the ear or in an acne pustule or local lesion anywhere about the head. Shortly after or even before the erysipelatous swelling, enlargement of the submaxillary lymphatic gland can be detected. The color of the inflamed skin varies from a rose color to a deep red, is for a moment effaced by momentary pressure of the finger, and is accompanied by an elevation of temperature of from one to three degrees above that of the healthy surface. The erysipelatous plaque is slightly elevated, and is often separated from the sound tissues by a sharp ridge, which if not visible can be perceived by the finger. The surface is smooth and shining, and may become bullous or vesicular or even pustular within from twenty-four to forty-eight hours. In the hemorrhagic form of the disorder which occurs in very old or cachectic subjects, the exudate in the vesicles is bloody and scattered ecchymoses occur, the whole ending, it may be, in gangrene. There is sometimes a burning pain, more commonly an annoying sense of tension. As the disease rapidly spreads, the face becomes enormously swollen, the eyelids closed, the ears enlarged to twice their normal size, and the features obliterated.

The fever in erysipelas is probably always proportionate to the severity of the intoxication, but certainly not to the amount of local lesion. It usually reaches 103° to 104° F., with very moderate morning remissions (less than a degree), or sometimes with great oscillations, especially in severe cases. Often the morning and evening temperatures are a little lower than on the preceding day, but there may be steady maintenance or even ascent of temperature until death. In typical cases the local lesions cease to advance from the fifth to the tenth day, at which time there occurs a rapid fall of the general temperature, amounting, it may be, to five degrees in thirty-six or even twenty-four hours. Constitutional disturbance and adynamia are usually not very pronounced, but in the old and debilitated, and especially in alcoholics, the typhoid state, with dry glazed tongue, rapid pulse, delirium, and stupor, may develop.

The subsidence of the local swelling is usually rapid, and may be attended by a furfuraceous or sometimes membranous desquamation. When the scalp has been invaded the hair often falls out, to return, however, in full vigor. Cases have been reported in which white hair has been replaced by black, but the contrary is much more common. Convalescence is usually rapid, but is sometimes interrupted by cutaneous and other local abscesses.

Of the varieties of superficial erysipelas made by systematic writers, the only one necessary to mention is the so-called *E. migrans*, in which large portions of the body and even of the extremities become inflamed.

In some cases erysipelas passes into the deep connective tissues of the body, producing the so-called *phlegmonous erysipelas*, which is distinguished from the superficial erysipelas by the greater swelling and darker color of the part and by the greater intensity of the pain. The part is hard and tense; after a time it becomes soft and boggy, an evidence that suppuration has occurred. Moist gangrene of the skin may follow this, with discharge of pus and of shreds or even masses of areolar tissue. This form of erysipelas is usually considered a surgical rather than a medical disorder, because it especially occurs in connection with wounds which enable the poison to get into the deeper fascia of the part.

Erysipelas of the mucous membranes may arise during an attack of external erysipelas or may be primary. When the disease attacks the nasal mucous membrane it produces a coryza, with burning pain, headache, and fever. Erysipelatous stomatitis is rarely primary; it may be accompanied by large pseudo-membranous patches and by pronounced stomatitis. Erysipelatous angina differs from catarrhal angina in its being excessive, painful, and accompanied by little tumefaction of the mucous membrane, but by great swelling of the lymphatic glands and high fever. In bad cases phlyctænulæ appear and may end in gangrene; retropharyngeal abscesses are not rare. When primitive and near the submaxillary glands it is one form of the so-called *angina of Ludwig* (*angina Ludovici*), and is apt to be accompanied not only by severe local symptoms, such as excessive swelling of the submaxillary glands and œdema of the glottis, but also by general infection and nephritis. If the larynx be involved, serious dyspnœa results.

As has been especially pointed out by Cornil, erysipelatous pneumonia is distinguished from true pneumonia clinically by the insidiousness of the attack and the rapid spread of the inflammation over wide lung territories; histologically by the total absence of fibrin from the exudate. Erysipelatous gastritis and enteritis may exist as primitive diseases, but as such are extremely rare. The genito-urinary mucous membrane is very rarely attacked except as the result of distinct local infection by the hands of the accoucheur or the gynæcologist. Even in puerperal women with facial erysipelas rigorous antiseptic precaution will almost invariably prevent the local infection. The vulva should be kept well covered with an antiseptic dressing. Erysipelatous or streptococcus meningitis, neuritis, pleuritis, arthritis, pericarditis, myocarditis, and endarteritis have been proved to occur, but are less frequent than endocarditis. Albuminuria is commonly present in ordinary erysipelas, and of all visceral complications nephritis is the most common.

DIAGNOSIS.—The diagnosis of external erysipelas requires no discussion. The recognition of the nature of internal erysipelatous inflammations without history of exposure to infection is extremely difficult, and often impossible. The rapid wide-spread development of the inflammation and the serous character of the exudate are the only characteristic points.

PROGNOSIS.—Death is extremely rare in so-called idiopathic erysipelas, unless in very old or feeble subjects. *E. migrans* is a very grave form, and is apt to be very prolonged. In puerperal or pregnant women the prognosis is good so long as inoculation of the genito-urinary tract is prevented.

TREATMENT.—Erysipelas being a distinctly contagious disease, isolation and personal disinfection are demanded. The contagion is, however, so little active that ordinarily in private houses no great degree of caution is essential. In hospitals or where there are wounded or lying-in subjects the utmost care should be taken. The obstetrician should refuse all cases of the disease when actively employed in his profession. The disease being in its beginning a purely local infection, the most natural treatment would seem to be local antisepsis. Unfortunately, however, cases are rarely seen until such a wide-spread area is affected that local treatment avails but little. At one time silver nitrate and iodine were much used locally: in our opinion their capabilities are much greater for harm than for good. Injections of solution of carbolic acid (two per cent.) or of corrosive sublimate (1 to 10,000) just beyond the edge of the spreading inflamed area are stated by some practitioners to act most happily: we have not used them sufficiently to warrant us in giving a personal opinion. Ichthyol is much used; the skin should be thoroughly washed with corrosive sublimate solution (1 to 1000), thickly smeared with a mixture of equal parts of ichthyol and vaseline, and covered with antiseptic cotton or gauze. The old application of lead water and laudanum simply tended to subdue inflammation; but its use in our hands has survived more modern methods. The cold-water dressing dates back to Hippocrates, and perhaps is as good as any other local treatment.

The constitutional treatment should consist of nutritious light diet, with stimulants when there is feebleness or adynamia. Tincture of ferric chloride should be given in doses of ten to fifteen minims every three to four hours, well diluted ; whilst quinine and strychnine may be administered in doses proportionate to the weakness. Constipation, diarrhœa, restlessness, insomnia, and other untoward symptoms should be met as they arise ; but no depressing remedies should be used : large doses even of the bromides are too depressing.

In phlegmonous erysipelas the pus and other products of the disease must be evacuated early and thoroughly. For the details of the treatment the reader is referred to works upon surgery.

Streptococcus antitoxin serum has been used in the treatment of erysipelas by Marmorek, Gromakowsky, and a few other observers, with alleged good results. Marmorek injected of the serum prepared in the Pasteur Institute of Paris ten cubic centimetres, or in very bad cases twenty cubic centimetres, followed in twenty-four hours by ten cubic centimetres. In one case one hundred and twenty cubic centimetres were given in ten days.

SEPTICÆMIA.

Definition.—A condition due to the absorption into the blood of the products of decomposition, or of the organisms which produce such changes.

The definition just given of septicæmia covers two conditions : one (*sapræmia*, *septic toxæmia*, or *septic intoxication*) in which no organisms are found in the blood ; and one in which micrococci or other septic organisms are in the blood (*septic infection*).

In many cases of disease the immediate cause of death is a septic intoxication superadded to the poisoning of the blood by the products of the primary bacterium. Thus, in scarlet fever, in small-pox, in diphtheria, in phthisis, and in other acute and chronic disorders, streptococci and other organisms flourish in the soil which has been prepared for them by the labors of the original pathogenic organism, and greatly aid in bringing about a fatal result. It is probable that in all cases of septicæmia there are a primary lodgement and local development of the organism which produces the disease ; but certainly there have been cases in which the symptoms were indistinguishable from those of a septicæmia, yet in which no local deposit of the pathogenic organism was found at the autopsy, though carefully looked for by competent observers.

That there are two forms of septicæmia, sapræmia and septic infection, has been abundantly demonstrated by experiments upon the lower animals, and these forms must exist in man, though this has been denied by some authorities. A little reflection shows that the term "septicæmia," which arose at a time when the pathology of the disease was not even suspected, is a very general one, covering a large number of blood-poisonings, each of which would in a strictly scientifically detailed nomenclature have its own name, such as streptococcæmia, staphlyococcæmia, etc.

Morbid Anatomy.—Usually at the seat of the local lesion there is a decided septic inflammation of the minute lymphatics, accompanied by an œdematous condition of the adjacent tissues, swelling of the lymphatic glands, and a host of organisms. The changes throughout the body are not very pronounced, consisting mainly of cloudy swelling of the fibres of the heart and of the secreting cells of the liver and of the kidneys and of the epithelial and other layers of the gastro-intestinal mucous membrane, enlargement of the spleen and liver, and an alteration of the blood, which is remarkably fluid, dark-colored, with little tendency to coagulate, and contains often an abundance of organisms. Endocarditis is sometimes present.

Symptomatology.—A pure sapræmia is a rather rare affection, except after labor, when the decomposition of retained blood-clots or of portions of the placenta or of the membranes may give rise to the rapid production of poisons whose absorption is favored by the large exposed surface of the uterus and the vagina. Under such circumstances, with or without a chill, there may be a sudden rise of temperature, accompanied by free sweating, with nervous disturbance, delirium, and diarrhœa, and, if the symptoms are not checked, a tendency to collapse. In ordinary septicæmia the symptoms usually develop rapidly, but not with absolute abruptness. The disease may or may not be ushered in by a slight chill, but headache, malaise, great depression and anxiety, loss of appetite, perhaps nausea, occur, and are accompanied by a fever in which the temperature usually ranges from 100° to 102° F. As the symptoms increase, a peculiar apathy comes on, and may develop into a sort of stupor, out of which the patient can easily be aroused for the time being, so as to answer questions intelligently. The gastric disturbances usually increase, the tongue becomes drier and more coated, the febrile temperature rises. Delirium, rapid feeble pulse, great failure of strength, hurried irregular respiration, subsultus tendinum, involuntary discharges, and death as early as the fourth day, or in protracted cases much later, results.

Symptoms similar to those which have just been described may develop without apparent local lesions: to such cases the name *cryptogenic septicæmia* has been given by Leube. Unless, however, in any individual case some local focus can be discovered, or unless micro-organisms can be found in the blood, the true nature of the attack must remain in doubt. Some of these cases are probably the outcomes of ptomaine poisonings produced by fermentative changes in the alimentary canal, and hence may be looked upon as instances of sapræmia; indeed, of such nature have been considered the many cases of temporary wretchedness, with or without febrile reaction, which are sometimes known as biliousness, and are cured by a free purgation which empties the alimentary canal.

The symptoms of a septicæmia vary somewhat with the nature of the invading organism. A streptococcus septicæmia is usually very rapid in its course and attended with excessive prostration. A staphylococcus septicæmia is commonly slow of development.

DIAGNOSIS.—The constitutional symptoms of a septicæmia are similar to those of most infectious fevers, so that the positive diagnosis of a septicæmia must rest upon the discovery of the local infection or of the organisms in the blood. When neither of these can be made out, a positive diagnosis is not possible. Suspicion should be aroused by the presence of the constitutional disturbance without local organic disease and without the peculiar symptoms which characterize the infectious fevers.

PROGNOSIS.—The prognosis of septicæmia varies with the nature of the invading organism and the situation of its local lesions. A streptococcus infection is usually much more serious than an infection by a staphylococcus. A local colony in the throat is more dangerous than one in the finger. Moreover, the same organism varies greatly in its virulence: the more rapid the local growth and the development of the symptoms, the greater the virulence of the organism and the more serious the prognosis. If it be not possible to destroy the primary focus of infection, the prognosis becomes very serious.

TREATMENT.—The only effective treatment of a septicæmia is surgical, consisting in the thorough opening and disinfection of the wound or part in which is the local lodgement of the organism. Careful nursing, absolute rest, alcohol in doses proportionate to the depression, moderate doses of quinine, the sustaining of the heart and respiration by digitalis, strychnine, strophanthus, cocaine, and other stimulants, and the treatment of various symptoms as they arise, constitute all that can be done by the physician. High temperature is to be met by the use of external cold rather than by the administration of antipyretics.

PYÆMIA.

DEFINITION.—A condition produced by the entrance of pyogenic organisms into the circulation, clinically characterized by frequent chills and an extremely irregular, intermittent fever, with sweating, and anatomically by multiple metastatic abscesses and various local inflammations.

Probably in all cases of so-called pyæmia there is an absorption not only of pyogenic bacteria, but also of septic material produced by decomposition of the pus: hence the terms *septico-pyæmia* and *pyo-septicæmia*, used by some authorities. It is evident that septicæmia and pyæmia are closely allied and often coexistent conditions.

Etiology.—The number of organisms which may under various circumstances cause the formation of pus is very large. The so-called "essential pus-organisms" are the staphylococcus aureus and staphylococcus epidermidis albus, the streptococcus pyogenes, the bacillus pyogenes fœtidus, and the bacillus pyocyaneus; but other staphylococci and streptococci, as well as the micrococcus lanceolatus, the gonococcus, the bacillus coli communis, the bacillus proteus, the bacillus typhi abdominalis, the bacillus aerogenes capsulatus, the pneumococcus, and other low forms of life, are capable of inducing suppuration. The organism may find access to the general system through the lymphatics from a local lesion, which may be undiscoverable (cryptogenic), but probably more commonly enters by producing an infectious inflammation of the wall of a vein, which leads to a coagulation upon the surface of the inner lining, in which the bacteria grow and cause a puriform softening of the thrombus, portions of which become detached and are carried along the course of the circulation as infectious emboli.

Morbid Anatomy.—The anatomical changes characteristic of pyæmia as distinguished from septicæmia are those connected with the formation of pus in various parts of the body. Hence suppurative thrombo-phlebitis, lymphangitis, arthritis, periostitis, osteomyelitis, inflammation of serous membranes, and embolic abscesses are to be found. The lesions indicative of septicæmia as differentiated from pyæmia are the acute splenic hyperplasia, and the granular degeneration of the heart, liver, and kidneys. In septico-pyæmia the focal lesions are associated with the degenerative changes in the parenchymatous organs.

Symptomatology.—An acute pyæmia is usually ushered in by a chill, which may be very light or very severe. If an immediate inspection of the wound be made, it will usually be found that the inflammation has increased, or that a change has taken place in the character of the pus. Even during the chill the temperature begins to rise, and in the course of from one to a few hours reaches 103° to 105° F., after which it drops to normal with a profuse sweat. Usually the next day a second paroxysm occurs, and a condition is entered upon in which there is a continuous slight pyrexia, attended by great malaise, weakness, loss of appetite, nausea and vomiting, emaciation, and recurrent severe febrile paroxysms. In these paroxysms the temperature rises from two to five or even six degrees with extreme rapidity and falls almost as abruptly, the defervescence being accompanied with profuse sweating. Symptoms of the typhoid state now come on; the tongue becomes coated, and the pulse rapid and feeble; but the intellect usually remains clear until the last stages of the disease, when delirium and coma appear. As the case progresses, erythematous, roseolous, or even pustular rashes may develop.

Local symptoms appear early in a case of acute pyæmia. They are the result of the metastatic abscesses, and their nature is, of course, dependent upon the site of the local changes. When the secondary abscesses are formed in external parts, as the parotid gland or the testicle or joints, they can scarcely be overlooked; but when they occur in internal organs the symptoms they produce may be very obscure. Cough and expectoration, with dyspnœa, dulness upon percussion, and pleuritic rub or pneumonic râles, may reveal local changes in the lungs; but pulmonic metastatic abscesses may exist without any distinct disturbance of the lung function. Empyema is not extremely rare. Jaundice may be due to a metastasis into the liver or to a duodenal catarrh, but may result from the rapid destruction of the red blood-corpuscles, and is, therefore, diagnostic only when associated with hepatic tenderness or increased area of liver dulness. Metastatic infarcts and abscesses may develop in the spleen and give rise to enlargement and tenderness, though they are somewhat rare. Endocarditis is liable to occur, and may be accompanied by distinct physical signs, but in some cases, even though there be a great alteration of the valves, muffling and indistinctness of the heart-sounds are the only changes from the norm. Septic conjunctivitis may occur, or panophthalmitis may end in the destruction of the eye. Even when there is no pronounced inflammation of the organ, white necrotic spots may appear in the retina, with or without hemorrhage, and are very characteristic. Suppurative periostitis or acute osteomyelitis is especially prone to develop when the invading organism is the staphylococcus aureus, constituting what was at one time known as *bone typhoid*. Septic nephritis, with infarcts and abscesses, may be accompanied by the characteristic urinary changes of acute Bright's disease, but may exist with a normal urine. Wide-spread inflammations of the joints are especially apt to occur in the chronic forms of pyæmia and septicæmia, giving rise to various so-called rheumatisms. As examples may be mentioned the gonorrhœal and scarlatinal rheumatisms.

Acute pyæmia runs a rapid course, but the cases grade into the chronic form, which may last for months. Chronic pyæmia is characterized by irregular fever, with excessive diurnal ranges of temperature (97° to 103° F.), slight occasional chills, great loss of strength, emaciation notwithstanding a good appetite, frequent diarrhœa, progressive anæmia, excessive sweating, great feebleness of voice, and death either in an exacerbation or from exhaustion. The intellect commonly remains clear almost to the last.

DIAGNOSIS.—The first stage in the diagnosis of a pyæmia is the recognition of the local disease which produces it. It should be remembered that an osteomyelitis, a gonorrhœa, a prostatic abscess, a pyelitis, a tubercular ulcer in the lungs or elsewhere, in brief, any local disease which produces suppuration, may be the source of pyæmia. The disease-processes which most closely resemble an acute or a subacute pyæmia are typhoid fever, acute miliary tuberculosis, and malarial diseases. The irregular intermittent fever which accompanies chronic gall-stones or other hepatic disease is in all probability a form of septicæmia: it is to be recognized by the persistent local symptoms of gall-stones or hepatic diseases. The nature of the malarial disease is to be determined by the therapeutic test: an intermittent fever which is not arrested by massive doses of quinine is not malarial.

The diagnostic characteristic points in the constitutional disturbances of pyæmia are the irregularities of the paroxysms, the free sweating which accompanies them, and the great swing of the temperature. Septicæmia may produce similar symptoms; but in pyæmia the constitutional manifestation is soon followed by evidences of multiple local lesions, and it is the conjunction of constitutional disturbance with the local lesions that makes the diagnosis positive.

PROGNOSIS.—The distinction between septicæmia and pyæmia is largely an artificial one: an abscess may produce a septicæmia with no organisms, with few organisms, or with many organisms in the blood; further, the same organism may, under varying conditions, be either septogenic or pyogenic. Restriction of the name pyæmia to those cases in which metastatic abscesses form has the practical advantage of isolating the almost necessarily fatal cases from those which are simply dangerous. We have seen hundreds of cases of acute pyæmia (chiefly during our civil war), with but one recovery in a clearly defined case; septicæmia, on the other hand, is frequently recovered from. Chronic pyæmia is usually fatal, but may pass into a condition of partial restoration to health.

TREATMENT.—The most essential point in the treatment of pyæmia is the disinfection of the local source of infection. Unless reinfection can be prevented, the case is, of course, hopeless. Alcoholic and other stimulants should be used; quinine should be given in sustaining and not antiperiodic doses; strychnine, cocaine, digitalis, strophanthus, and the whole list of stimulant drugs and stimulant foods should be used freely as demanded by the symptoms. Opium may be employed for the relief of pain; chloral, trional, and sulphonal, if necessary, to produce sleep; external cold for the reduction of high temperature; and bismuth, astringents, and opiates for the arrest of diarrhœa; but no known measures have any pronounced influence upon the course of the disease.

TETANUS.

DEFINITION.—A febrile disorder, produced by the presence of a peculiar bacillus, and characterized by violent tetanic (spinal) spasms, continually recurring in response to the slightest peripheral irritation.

ETIOLOGY.—The cause of tetanus is a peculiar bacillus, which was first found by Nicolaier in garden-earth, and was afterwards isolated by Rosenbach from a wound of a man dead of the disease. It is especially characterized by an enlargement of one end, due to the presence of a bright spore, which is so resistant to morbific agents that it may be isolated by heating materials containing it to 80° Centigrade for one hour, at the end of which time all bacilli and all other spores except those of tetanus are killed. The tetanus bacillus is also most tenacious of life in the presence of ordinary disinfectants, not being killed in ten hours by a five per cent. carbolic acid solution, or in three hours by a one to one-thousandth solution of mercuric chloride. It develops in almost all media at ordinary temperatures, provided there be no oxygen. When inoculated either from natural or from artificial cultures into men and most domestic animals it rapidly grows, but does not enter the lymph or blood, remaining, therefore, a local infection. Even for such local growth it seems to be necessary for the local bacillus to be accompanied by a poison, since when introduced into an animal without the poison which it produces it is rapidly destroyed. Certain chemical agents, notably lactic acid, are, however, capable of replacing the natural poison. This poison which produces the symptoms of tetanus is a toxalbumin of such extraordinary virulence that its minimum fatal dose is about one two-hundredth that of strychnine, being, it is affirmed, 0.23 milligramme. That it is very rapidly produced after inoculation is shown by the fact that Kitasato found that excision of the point of inoculation in the mouse fails to save the animal unless practised within an hour after the inoculation. More or less time is required for the production of the toxin by the local growth of the bacillus, so that the period of incubation of tetanus in man is usually between five and ten days, but may be prolonged to three weeks. The stage of incubation may, however, be wanting. Thus, Robinson has reported the appearance of tetanus half an hour after the reception of a wound of the finger; whilst, according to Jaccoud, death from tetanus has occurred fifteen minutes after the reception of an injury. In such cases there has been direct inoculation with an already produced tetanus toxalbumin. Nicolaier caused a severe tetanus in himself by pricking his hand with a needle moistened with a toxin free from bacilli.

Tetanus toxin has been prepared from cultures and from the blood and urine of tetanized animals. Further, a solid antitoxin has been obtained from the blood-serum of horses, goats, and dogs poisoned by the gradual introduction of the toxin.

The peculiarities of the organism, and the fact that it abounds in certain soils and localities, explains the facts that punctured wounds, first of the feet, next of the hands, are especially apt to cause the disease, that men are more frequently attacked than women, and that those who work about stables are especially prone to suffer.

The occasional occurrence of local epidemics of tetanus in man, the fact that certain districts are notorious for the frequency of the disease, and the belief of veterinarians that certain stables are local centres of tetanus, are also in accord with our present knowledge of the nature of the poison. It is evident that under unknown favoring conditions there is an excessive local development of the organism.

In the peculiarities of the tetanic bacillus is to be found the explanation of certain etiological peculiarities of the disease. Thus, the negro race is alleged to be extraordinarily susceptible to tetanus, because in some of the West India Islands more than half of all the negro children born are known to die of tetanus. The evident explanation of this is to be found in the squalor and filth of the negro hovels, which cause them to be locally infected with the tetanus organism, so that the ulcers of the umbilical cords or the trifling abrasions of the new-born become the seats of inoculation. The same explanation attaches to the occurrence of puerperal tetanus among the negro mothers. It is probable that heat favors the growth of the bacillus; certain it is that the disease is more frequent in hot climates. Thus, whilst, according to Rosenthal, there are in Vienna 2.39 cases of tetanus for one thousand of sick, and in Guy's Hospital 1.13 for one thousand, in Bombay the proportion is said to be 7.3 for one thousand. Both in India and in New Orleans (Louisiana) fatal tetanus has followed upon the hypodermic injection of quinine sulphate.

Much light has been thrown upon the origin of the so-called idiopathic tetanus by Carbone and Perrero, who very recently found in a case the tetanus bacilli in the inflamed bronchi mixed up with pneumonic diplococci, and discovered that this form of the tetanus bacillus, so far from being strongly anaerobic, flourishes best in the atmospheric air.

MORBID ANATOMY.—Death may occur from tetanus without any demonstrable alteration of the spinal cord or in the nerves. In some cases, however, there has been an inflammation of the spinal centres; but these and other lesions which have been found by competent observers in the bodies of those dead of the disease are not essential phenomena, though they may have been the outcome of the excessive irritation of the spinal motor centres by the poison.

SYMPTOMATOLOGY.—Tetanus may develop abruptly, but usually the characteristic muscular contractions appear after some hours of prodromes, such as chilliness, stiffness of the neck, and malaise. Occasionally the spasmodic contractions begin in the part which has been wounded, but ordinarily they first appear in the muscles of the jaws, from which they pass to the muscles of the neck, the back, and the whole body. Violent trismus is, therefore, commonly the first pronounced symptom of the disease, and is apt to be accompanied with disorder of the movements of the tongue and the muscles of the larynx, and, therefore, with disorder of the speech.

As the disease progresses, tetanic contractions of the facial muscles produce a peculiar immobility of the face, with wrinkling of the forehead, drawing up of the corners of the mouth, exposure of the firmly closed teeth, and wide-open staring eyes,—the "sardonic grin." The great deepening and stretching of the lines of the face produce, also, an extraordinary appearance of age. Owing to the greater strength of the muscles of the back, the head is usually drawn somewhat backward, whilst the spinal column is so bent that the chest and upper abdomen are thrust forward and the body rests upon the head and shoulders and the buttocks (opisthotonos). The abdominal muscles are rigidly contracted, flattening out the belly; whilst the lower limbs are immovable in extension. The arms usually can still be controlled by the patient. In rare cases, by the irregular muscular contractions the body is thrown into emprosthotonos or even into pleurosthotonos. Painful erections of the penis sometimes occur, but are rare.

In some cases the tetanic rigidity is continually maintained. Severe clonic contractions often occur, involving the whole muscular system, and producing violent shocks, intense opisthotonos, and thrusting forth of the tongue, which may be badly bitten. The respiration may be so interfered with that there is a pronounced sense of suffocation, and a cyanosis, which may go on until unconsciousness and even death result. In very mild cases these crises may not be pronounced; in the more severe cases they are liable to be produced by the slightest peripheral irritation, such as may be caused by a jar of the bed, a draught of air, or even a loud sound or a bright light. They are often worse at night than in the day; hence there is usually pronounced insomnia; but when sleep is obtained the spasm ceases, to recur upon awaking. The muscular contractions, especially in the clonic movements, are accompanied by muscular pains, which may be very violent, and sometimes also by a hyperæsthesia, though the tactile and thermic senses have been noted in individual cases to be distinctly diminished. The intelligence is not affected. Local or even wide-spread paralyses have been recorded, but are uncommon.

In the onset of the disease the temperature is usually normal or slightly elevated, but later the fever becomes very pronounced, and in fatal cases, just before death, the temperature often rapidly rises, reaching, it may be, 110° F., and continuing to go up for some time after death. The pulse often remains normal for a long time, but sooner or later becomes very rapid (even 180 per minute), small, and somewhat irregular. The urine in occasional cases contains albumin or sugar. Various systematic writers state that there is no increase of the excretion of urea; but our present evidence is too contradictory to warrant any positive conclusion. Senator, who failed to find increase of the urea, found lessening of the creatin and creatinin. Hupert discovered a distinct increase in urea elimination.

There is no definite course to tetanus: it may end in a few days, usually fatally, or may drag on for weeks. When recovery takes place, it is usually by a gradual subsidence of the symptoms. In relation to its course, tetanus may for the purpose of its discussion be divided into the foudroyant form, in which death occurs in a few hours, it may be even before the muscular contractions become general; the acute or ordinary form; and the chronic variety, in which the symptoms are mild and stretch over many days.

Under the name of *cerebral tetanus* has been described a condition which originates from a wound of the head and is characterized by paralysis of the facial muscles on the same side as the wound, with trismus and difficulty of swallowing. How far this is the same disease as ordinary tetanus remains somewhat doubtful.

Tetanus neonatorum, or tetanus of the new-born, usually appears about the fifth or sixth day after birth, with prodromic restlessness, failure of appetite, and general illness, followed by trismus, facial contraction, difficulty in deglutition, and tonic muscular contractions, which spread through the whole body as in ordinary tetanus, and terminate after two or three days either in fatal asphyxia or in collapse.

In the so-called *idiopathic tetanus*, which originates without a wound, the symptoms are usually not nearly so severe as in the traumatic affection, and the course is almost invariably prolonged.

PROGNOSIS.—There is an extraordinary difference in the statements of authorities concerning the mortality-rate of adult tetanus. Thus, Reichter in battle-field cases gives the mortality-rate as eighty per cent.; in civil cases the mortality is, according to Marcosignori, twenty-five per cent.; to Albertoni, twenty-one per cent.; to Gowers, ninety per cent.; to Dean, eighty per cent.; to Sormannie, forty-four per cent.; to Osler, eighty per cent. In infants the prognosis is practically fatal; in older children the disease is less dangerous than in adults; in puerperal tetanus recovery is almost unknown. In attempting to decide the chances of individual cases, it should be remembered that the shorter the time that has elapsed between the injury and the coming on of the symptoms the worse the prognosis. Thus, J. T. Whittaker gives the mortality as ninety-six and six-tenths per cent. when the interval has been less than ten days, and eighty-four per cent. for all cases. Further, the result is almost invariably proportionate to the early severity of the symptoms, and the greater part of the deaths take place in the first week: so that if this period be survived the chances of recovery are at least fifty per cent. After survival for two weeks the danger is comparatively slight. From the best obtainable statistics it would appear that in cerebral tetanus the mortality-rate is in acute cases over ninety per cent.; in chronic cases about twenty-five per cent. In idiopathic tetanus more than fifty per cent. recover. The favorable indications are late onset, general lack of intensity of the convulsions and their confinement to the muscles of the jaw and neck, absence of fever, and a tendency to a general slow course. When the disease develops early and the symptoms are from the beginning severe, there is almost no hope.

DIAGNOSIS.—The characteristic symptoms of tetanus are the early occurrence of trismus, the steadiness and universality of the tetanic contractions, the presence of fever, and the length of the course. It may be closely simulated by hysteria, in which case, however, there will be almost invariably a history of previous existence of hysterical temperament and emotional disturbance, and a more or less partial character of the convulsions. In strychnine poisoning the course is more acute than is ordinarily seen in tetanus. Trismus, if it occur at all, comes on as a late symptom, and the relaxation between the convulsive crises is more pronounced than in the ordinary disease. In certain stages of strychnine poisoning the diagnosis without history may be almost impossible. The toxin of tetanus is eliminated by the kidney, so that if in a doubtful case injection of the urine should produce immediate tetanic convulsions in a rabbit the diagnosis would be clear. A negative result, however, does not disprove the existence of tetanus.

TREATMENT.—In order to prevent the development of tetanus, punctured wounds, especially in the extremities, should always be freely opened and thoroughly disinfected; and even after the occurrence of tetanoid symptoms free excision of the wound or of its scar and cauterization should be practised, so as to break up, if possible, the local bacillus colony and prevent a further formation of the toxin. It is essential that the patient be put on a wide bed in a darkened, very quiet room, and kept absolutely free from all disturbances. A slight draught may produce a fatal convulsion. Bromide of potassium should be given in very large doses in diluted solution. Half an ounce of it should be exhibited at a dose as soon as the case is seen. If the symptoms be severe, drachm doses should be administered at intervals of from three to six hours.

Almost any of the spinal depressants may be useful in tetanus. Chloral is of special value because of its tendency to produce sleep. It may in very acute cases be used continually day and night, but in chronic cases it is better to employ it at night in order to obtain sleep. Under these circumstances it is best administered in combination with opium. Twenty to thirty grains of chloral, with one-fourth of a grain of morphine, may be given at a time, and repeated, if necessary, in half a dose. The combination of chloral with hyoscine hydrobromate is sometimes very serviceable. Calabar bean extract has been much used, with not very gratifying results. On account of its varying strength, it should at first be given in small doses, one-sixth of a grain, which may be increased. Eserine in the dose of one-fiftieth of a grain, at intervals of from four to six hours, increased if necessary, is preferable to the extract. Opium, given continuously in small doses up to mild narcotism, is often very serviceable. Chloroform and the nitrites are of the greatest service, but are fugitive in their action, and therefore should be especially used in the crises of convulsions. Nitroglycerin (Spiritus glonoini, *U. S.*), being more persistent in its influence, is for a steady effect preferable to the other nitrites, but even it should be given at intervals of not less than an hour (dose, one to two drops). Cannabis indica is largely used in India and much relied upon, given to the point of intoxication.

In administering these various remedies it must be remembered that none of them are specific, and that the sufferer from tetanus may go on rapidly to death although the convulsions are for the time being set aside. It is plain, therefore, that, whilst very large doses are necessary, it is often better to subdue the convulsion than to overcome it completely; for such complete overcoming may demand the use of doses so large as to be distinctly depressant to the heart or the general system. Further, in chronic cases it is essential to vary from day to day the spinal depressant, so as to prevent the system from becoming accustomed to any one remedy, and to put aside the danger of any accumulation of the drug. In many cases digitalis may be added to prevent heart-weakness.

As there is a toxic adynamia, alcohol is indicated. Moreover, alcohol in large doses is a spinal depressant, so that for a double purpose it should be freely employed. Owing to the impossibility of chewing, and the common difficulty of swallowing, concentrated liquid food should be administered in as large quantities as the patient can digest. If the trismus be very severe, the patient should be fed by a tube through the nose, or sometimes a tooth may be extracted so as to allow the passage of a tube into the mouth. If there be obstinate constipation, mild laxatives should be given. Injections are liable to produce reflex convulsions.

In the laboratory artificial tetanus produced in various animals can be certainly controlled by early treatment with antitoxin, but in practical human medicine the affair is entirely different, because there can be no estimate of the amount of toxin in the body, and especially because treatment cannot be commenced until the toxin has permeated the whole system.

Howlett found in sixty-eight cases of all forms of tetanus treated with antitoxin a mortality of thirty-six per cent.; in an elaborate analysis of fifty-four cases Kanthack has shown that in the more acute cases, with an incubation period of less than eight days, the mortality has been eighty-five and seven-tenths per cent., but in the chronic cases only five and seven-tenths per cent. Kanthack gives strong reasons for believing that many fatal cases have not been reported, and seems to us to be fully justified in his conclusion that in acute tetanus the antitoxin is practically powerless. On the other hand, it appears probable that in chronic cases the remedy has distinct value. In regard to the amount used, Kanthack shows that as the manufacture of antitoxin has improved, the dose has decreased steadily from over fifteen grammes to nine-tenths of a gramme.

The first dose of the antitoxin serum of Behring and Roux is put down as from twenty to thirty cubic centimetres; the after-doses, given at intervals of from five to ten hours, are from fifteen to twenty cubic centimetres. Antitoxin prepared by Tizzoni and Cattani, near Bologna, is furnished in a dry state in small flasks, which must be kept sealed until the time of use, when their contents are dissolved in distilled water recently boiled and allowed to cool, one part by weight in ten parts by weight of water. The injection is to be practised in the thigh with a syringe which has been rendered aseptic by heat, not by chemical disinfectants. In a mild case in the adult half of the contents of the vial is to be injected as the initial dose, and the remainder is to be divided into four doses, to be used at intervals the following day, according to the necessities of the case. If the case be seen late, or if the incubation period has been very short, or the symptoms are severe, the first injection should contain the whole contents of the vial, and a second vial be used the next day. With children the doses are to be about one-half. In very severe cases larger doses than those above mentioned may be given. The use of the antitoxin should not interfere in any way with the other treatment of the disease.

Overshadowing the use of antitoxin is the practical difficulty of procuring a pure article. We have seen increase of fever and other symptoms produced by the use of a standard antitoxin, and other similar cases have been reported. It is probable that the antitoxins used in these cases contained toxin. As one ten-thousandth of a grain of toxin might seriously injure a patient, the danger of trusting to any manufacture of tetanus antitoxin is obvious.

MALARIAL DISEASES.

Definition.—Affections which are produced by the presence in the human body of a peculiar hæmatozoon.

Etiology.—Malarial diseases are not contagious, and do not pass from man to man ; they are the outcome of a poison which is produced outside of the body. For the production of this poison there are required a proper soil, an abundant moisture, and a sufficient heat. As these conditions are wide-spread, malarial districts are found from the subpolar regions to the equator. As a rule, the extent of the infected districts and the virulence of the poison which emanates from them steadily increase with the heat of the climate, so that the most deadly malarial countries are tropical or subtropical. The character of the soil necessary for the production of malaria is not thoroughly understood ; it is certain that an alluvial soil is an especially fit habitation, and it would seem that the suitability of the soil usually increases with the amount of vegetable organic matter in it. There are, however, especially in the colder malarial countries, great differences in neighboring localities, which cannot be accounted for with our present knowledge. It is probable that there are organic or inorganic constituents of certain soils which inhibit the growth of the malarial organisms and therefore render healthful a certain swamp in an infected district. The amount of moisture in a soil has immense influence : if a tract be covered all the time with even a very shallow depth of water, it is almost innocuous ; if it be alternately exposed and covered with the changes of the tide, it may be very dangerous; but the most deadly of all localities are those in which, without there being water upon the surface, the ground-water reaches close to the top of an alluvial soil containing much organic matter. It was such a soil that in the famous Walcheren campaign in 1809 put twenty-seven thousand out of forty thousand English soldiers into the hospital. As a rule, the deltas of rivers and the country around great fresh-water lakes are abundant producers of malaria. Thus, in Europe the valleys of the Po, the Tiber, the Danube, and the rivers of the Black Sea are the chief sources; whilst in America the valleys of the Delaware, the Chesapeake, and the rivers in general of the southern United States, with certain portions of the shores of the great lakes, are the most dangerous localities. The damming of rivers and the drainage of marshes are powerful factors for increasing or decreasing the production of malaria. Cultivation of the soil in some way seems to lessen its productive power, so far as malaria is concerned. As an instance of the changes wrought by artificial means may be mentioned the Schuylkill River, which in the beginning of the present century was so slightly malarious that on its banks were the habitations of the wealth of Philadelphia; the river was dammed, and the banks became uninhabitable; then paddle-wheel steamers were put upon the dam, and, either as a result or as a coincidence, there was almost complete disappearance of the malaria.

Changes take place, however, in the production of malaria which are not easily accounted for. It is clear that there has been a great decrease of malaria in New England and in the Middle United States, whereas it is asserted that about the ports of the Gulf States the disease is, on the whole, increasing. Can this be by the importation of fresh, extremely virile germs from the tropical islands and mainland? It is affirmed that malaria has disappeared from Lake Ontario; and in the Northwestern States it is almost unknown. How far it is capable of extending northward is somewhat uncertain, but the St. Lawrence region in America and the wide marshes of St. Petersburg are unpolluted.

Age has little or no influence upon the susceptibility to the malarial poison, and instead of an attack affording protection against the disease it renders the subject much more liable. Nor is there, so far as observation goes, any hereditary insusceptibility; the white races, at least, do not become accustomed to the disease, but, in fact, degenerate in the face of a persistent overwhelming malarial poison. On the other hand, the negro races, and, it is affirmed, to a less degree also the Arabs, enjoy almost an immunity. As a certain degree and persistence of heat are necessary for the development of the malarial germ, the late summer and the early fall are the seasons of greatest danger. In certain seasons the relations of temperature and moisture are such as greatly to stimulate the growth of the malarial germ; but, independently of such open climatic influences, there are in some years violent malarial epidemics.

It is plain that ordinarily in thickly populated cities the conditions are not favorable for the development of the germ, hence malaria is a disease of the country rather than of the town; but it is not true that complete protection is afforded even in the most thickly populated city. Heavy fogs and the moist air of night favor the rising from the ground and the dispersion of the malarial poison. Moreover, owing probably to mechanical reasons, high elevation above the earth affords protection, and the obstruction of a high wall or a dense wood may be sufficient to alter distinctly the malarial relations of a certain place. High winds may carry the germs to a considerable distance.

In 1879 M. Laveran, a French army surgeon, announced the discovery of a hæmatozoon, the germ of malaria. This discovery was confirmed in 1882 by Richard, in 1884 by Marchiafava and Celli, and since by numerous observers in Europe, Asia, and America. The malarial organism is usually believed to belong among the sporozoa. Its life-history outside of the body is unknown. So far, all attempts to grow the parasite have entirely failed. Nor as yet have we knowledge as to how the germ enters the blood. The experiments of Gerhardt, of Marchiafava and Celli, of Geraldi, and of others, have shown that intravenous or even hypodermic injection of blood may transfer the disease from man to man; but all attempts to pass the hæmatozoa through the alimentary canal into the system have so far failed. Inside of the body the hæmatozoa have a regular cycle of existence within the red blood-corpuscles; at first they consist of small hyaline amœboid bodies which develop within the red blood-corpuscles; these bodies as they grow in size become replete with minute dark pigment-granules that have been formed out of the hæmoglobin. After a time, when the red blood-corpuscles have been destroyed, the parasites divide into a number of small round or ovoid transparent bodies, each of which breaking away attacks a new corpuscle and enters a new cycle of life. As was first pointed out by Golgi, the parasites of a regularly intermittent fever exist in the blood in great groups composed of individual elements at about the same stage of development: in consequence of this, many thousands of parasites undergo sporulation at or near the same time, which sporulation is always followed by the malarial paroxysm: so that it would appear as if the febrile reaction were caused by some poison produced in the process of development of the parasite. Whether the malarial fever shall take the tertian, the quartan, or the quotidian form probably depends upon the rate of development of the hæmatozoa, although there seems to be still some doubt whether there are hæmatozoa which pass through the whole cycle in twenty-four hours and produce the quotidian paroxysms. There appear also to be differences in the organisms corresponding with clinical differences in the malarial disease.

The parasite of tertian fever grows from a minute hyaline body into a full-grown organism about the size of a red blood-corpuscle, which develops by segmentation into fifteen or twenty sporules in about forty-eight hours; the smaller parasite of the quartan fever requires seventy-two hours for its life-cycle, and produces usually from five to ten sporules, which are commonly arranged about a central mass of pigment so as to form a rosette. There may be in the blood two or more groups of organisms; if there be two groups of tertian or three groups of quartan organisms, each group passing through its own successive cycles independently, a series of quotidian paroxysms will result.

The parasite of remittent malarial fever varies from the other malarial organisms in being considerably smaller, and in giving rise to large, refractive, crescentic, ovoid, and round bodies, with central clumps of coarse pigment-granules, whose significance has not been determined. From these bodies, as well as from the tertian and quartan parasites, long, active flagella may develop, so that the hæmatozoa move freely among the blood-corpuscles.

It is evident that the anæmia and the pigmentation of paludism are the direct results of the destruction of the red blood-corpuscles by the parasites. Further, severe cerebral symptoms may be produced by the filling up of brain capillaries by the organism, which have also been detected in great quantities in the capillaries of the gastro-intestinal mucous membrane in malarial cases that have suffered from choleriform diarrhœa.

In bad cases of malarial fever there is probably produced at each sporulation almost a sufficiency of parasites to destroy all the red blood-corpuscles of the body. Moreover, it is well known that cases of malarial fever may get well spontaneously. It is, therefore, evident that there must be some provision for the destruction of the parasites in the blood. There are at present two theories: in accordance with one, the blood-serum kills the simple hyaline forms before they gain entrance into the red corpuscles; in accordance with the other, certain cells act as phagocytes. Although authorities are not agreed, the present evidence indicates that each of these theories is in part correct,—that is, that the parasites are killed by the blood-serum and also by phagocytes.

There appear to be at least three sets of phagocytes: first, leukocytes of the blood; second, the endothelial cells of the arterioles throughout the body; third, certain of the parenchymatous cells, especially the cells of Kupffer in the liver and the pulp-cells of the spleen. A phagocytic cell may contain any or all of the following: 1, red blood-corpuscles, normal, altered, or in fragments; 2, masses of hæmosiderin (probably formed within the cells); 3, malarial parasites in different phases of the developmental cycle, many of them degenerated or going to pieces; 4, malarial pigment, especially the central pigment clumps from segmenting parasites; and 5, white cells, both mononuclear and polynuclear. Again, certain large cells contain other phagocytes with their contents. According to L. F. Barker, leukocytes are especially apt to contain well-preserved parasites, whilst the large phagocytic cells of the spleen—*makrophages*—and liver contain with the parasites large numbers of infected red blood-corpuscles. Golgi has brought forward evidence that there is a periodicity of the phagocytosis corresponding to the developmental cycle of the parasite.

The observation of Dock, that the malarial parasites are arrested in their development soon after the death of a patient, strongly indicates that phagocytosis is not the only method of their destruction in the body.

MORBID ANATOMY.—The anatomical changes occurring in the milder forms of malaria are but little known, since patients rarely die. The fatal cases are usually those of pernicious malaria, in which the conspicuous changes are to be found in the spleen. This organ is enlarged to a greater or less extent, is dark-colored, and of diminished consistency. On section the pulp resembles dregs of paint. The follicles and trabeculæ are indistinct. At times nodules of hemorrhagic infarction are to be found, and rarely rupture of the capsule may occur. The liver and kidneys are swollen and opaque. Pigment-granules from degenerated blood-corpuscles may be found free or enclosed within leukocytes, in the spleen, and in the blood-vessels of the liver, kidneys, intestine, and brain. Foci of necrosis have been found in the liver and kidneys. In patients dying from exposure to chronic malaria the enlarged spleen is dense, its capsule and trabeculæ thickened, and on section the color is either brown or black. The liver is enlarged and increased in density. On section the color is composed of shades of gray or bluish-gray, with a tendency to the formation of dark patches. The pigment is especially abundant in the vicinity of the blood-vessels, and the connective tissue is somewhat increased. A similar grayish slate-color from the presence of particles of black pigment may be present in the kidneys, brain, and bone-marrow, and sometimes in the mucous membrane of the stomach and intestines.

SYMPTOMATOLOGY.—The paroxysms of an intermittent fever may commence suddenly, or may be preceded by malaise, anorexia, or other general prodromes. The typical attack is composed of three stages,—the chill, the fever, and the sweat. The first stage commences with a feeling of cold in the back, which soon radiates into all portions of the body, and is accompanied with horripilations, which may become so violent that the teeth chatter, whilst the body trembles sufficiently to shake the couch upon which the patient lies. The skin is pale, cold, and by contraction of the erector pili muscles thrown into goose-flesh. Vertigo, cephalalgia, ringing in the ears, troubles of vision, dilated pupils, vomiting, abundant urination, and frequent, small pulse are common phenomena. Notwithstanding the coldness of the surface and of the extremities, the bodily temperature begins to rise at the very onset of the attack, so that before the chill is over 104° or 105° F. may be reached, and the surface be extremely hot. Gradually the cold stage passes into that of fever, with brilliant eyes, congested face, strong, perhaps dicrotic, pulse, furious headache, and various nervous disturbances, such as mental confusion, unrest, and even delirium. Usually in from three to four hours, but in some cases not until ten or even more hours, the hitherto dry skin breaks out into a profuse perspiration, which is followed by a rapid fall of the temperature to 98.5° F., and commonly in from two to four more hours the subject has apparently recovered his normal condition. In some cases there can be detected during the stage of fever an enlargement and tenderness of the spleen, which abates during the sweating stage. Maissuriany affirms that an intermittent splenic bruit synchronous with the pulse may sometimes be heard. The urine examined at the end of the paroxysm is often albuminous, and, according to Eichhorst and others, there is an increase of the urea and of the phosphoric acid, and a decrease of the chlorides, during the whole paroxysm. The observation of Brousse, that the urinary toxicity is increased by the paroxysm, was not confirmed in the experiments of Laveran.

The return of the paroxysm of intermittent fever depends upon the type to which the attack conforms. In the quotidian the paroxysm recurs daily; in the tertian, every other day; in the quartan, every third day. Typically, the hour of recurrence should be that of the first attack, but very commonly in an intermittent fever each paroxysm appears two or even more hours before it is due. On the other hand, especially under the influence of not sufficiently large doses of quinine, the intermittent fever often undergoes retardation, so that the paroxysms are one or more hours behind time in their appearance. The malarial paroxysm occurs almost always in the daytime, and when the paroxysm of an accelerating or retarding intermittent comes to the night it is very prone at one leap to jump over the whole period of darkness. The cause of the rarity of nocturnal attacks of intermittent fever is not certain. We have seen, however, in a watchman who habitually slept during the day and worked at night, nocturnal instead of diurnal paroxysms.

Varieties in the intermittent paroxysms are very common. In the majority of cases as seen in the latitude of Philadelphia the chill is very slight or altogether wanting. The sweating stage is also frequently not pronounced, so that the whole paroxysm is reduced to from six to ten or perhaps more hours of a mild fever with headache.

The variations in the intermittent paroxysm may be so great that the typical febrile attack is entirely replaced by a new set of phenomena. Thus, violent urticaria attended with almost delirious excitement may replace the chill, and is said also sometimes to constitute the whole paroxysm. The most common of these irregular intermittents is that which is known as *brow ague*, in which the attack consists of a violent pain centred in the supraorbital foramen, filling the whole side of the head with agony, attended in rather exceptional cases with great depression, and perhaps with vomiting. In some cases the malarial attack may consist of frightful neuralgic pain in the trunk or the extremities. In a somewhat rarer form of irregular malarial fever, of which, however, we have seen a number of cases, the attacks consist of a recurring serous or choleriform diarrhœa, which yields to no treatment except that which is antimalarial. A very rare variety which we have seen once or twice is that in which the whole paroxysm consists of a protracted epileptiform convulsion, which may so closely resemble the convulsion of idiopathic epilepsy as to lead to the diagnosis of that disease. It is asserted that the irregular malarial paroxysm may take the form of recurring attacks of congestion of the lungs, each attended with the physical signs of an incipient pneumonia. Paroxysmal paraplegia and other paralyses have been reported from time to time as of malarial origin.

Remittent Fever. Æstivo-Autumnal Fever.—The *bilious remittent fever*, or simply the *bilious fever*, of the Southern United States differs clinically from ordinary intermittent fever in having the paroxysms so prolonged that they run into one another without any complete intermission. The types of the paroxysms are the same as in ordinary intermittent fever, quotidian, tertian, and quartan, though the quartan type is extremely rare. Next to the quotidian in frequency is probably the double tertian, in which there are two daily paroxysms at different times of day : the first paroxysm occurs in the morning, the second in the evening, of the first day ; the third paroxysm in the morning, the fourth in the evening, of the second day ; and so on. Remittent fever usually comes on with distinct prodromes, such as malaise and epigastric weight and fulness. Often there is a chill, mostly mild in character, followed by a violent febrile reaction, with heavily coated tongue, extreme thirst, nausea, and vomiting. At this time the pulse is usually infrequent in proportion to the fever. In from eight to twenty hours a remission occurs, with mild perspiration and lessening of the symptoms, to be succeeded by a paroxysm of fever like the first. In this way arises a fierce fever with regular remissions and paroxysms. The pulse becomes more rapid, rising, it may be, to 120 a minute, and the skin dries. Epigastric pains and tenderness, with nausea and vomiting, are common symptoms, and are usually associated with constipation, green, black, or yellowish, very offensive stools, scanty urine, and the appearance of a yellowish hue of the skin and conjunctiva, which by the fifth day of the disease has usually deepened into the bronze of a pronounced jaundice. At this time very commonly hepatic enlargement and tenderness can be demonstrated. The nervous symptoms may consist simply of headache, with apathy and a mild somnolence, but stupor and delirium are common phenomena. In favorable cases the symptoms gradually abate, the first evidence of change often being frequent copious dark tar-like discharges from the bowels. In very unfavorable cases the symptoms of vital failure come on early, and with a dusky, livid, purplish, or bronze skin, cold extremities, dark-brown tongue, tympanitic abdomen, and low delirium ending in stupor or coma, the patient passes on to death, which usually occurs between the seventh and the fourteenth day of the disease. Under these circumstances there are often excessive vomiting, not rarely hiccough, and sometimes black or bloody, persistent diarrhœa. In another set of cases the symptoms are said to assume more and more the appearance of a typhoid fever, with the remissions little marked and the stupor, subsultus tendinum, carphologia, and other symptoms of adynamia strongly pronounced.

Pernicious Fever.—*Malignant* or *congestive* malarial fever may suddenly develop out of an ordinary miasmatic fever, or may come on abruptly during apparent good health. Three forms of it, the algid, the comatose, and the hemorrhagic, are described by systematic writers. The divisions are, however, arbitrary.

In the algid malarial fever, as ordinarily seen, there are livid paleness of the face, with an expression of alarm, and often of collapse, great coldness of the extremities and sometimes even of the surface of the body, and an abundance of colliquative sweat. The internal temperature may be below or above normal. There may be oppression, epigastric weight, intense thirst, violent vomiting, and choleriform diarrhœa. The respiration is apt to be hurried, irregular, panting, not rarely sighing, with occasionally each inspiration interrupted in its progress and effected as by a double effort. The small, irregular pulse may be corded or may be feeble ; not rarely it is intermittent, sometimes as high as 170 a minute. Great restlessness and uneasiness, with a complete retention of consciousness, may exist, as in "walking cholera," clearness of mind and the ability to go about the room remaining when the pulse is imperceptible. These symptoms may increase until death takes place quietly in collapse, or may be followed by reaction and fever.

In the comatose variety there is marked cerebral disturbance, with partial or complete unconsciousness and symptoms of collapse, or with high fever ending in death with profound coma.

In cases of algid pernicious fever the dejections may consist of bloody serum or of pure blood, but the typical hemorrhagic form is that in which either hæmaturia or hæmoglobinuria is the most prominent symptom. In some of these cases the general symptoms resemble those of bilious fever, with chill, violent vomiting, intense lumbar rachialgia, high fever, and jaundice. In other cases the febrile paroxysm is wanting, the only symptoms being hæmoglobinuria, lasting from twelve to thirty-six hours, violent perspiration, and collapse. The urine is albuminous ; it contains an abundance of hæmoglobin, and often, although not always, entire blood-corpuscles.

As seen in the neighborhood of Philadelphia, pernicious fever offers a mixture of the symptoms of the algid and comatose forms. The hæmaturic form occurs in the extreme southern United States, and also in the tropical portions of Southern America, Africa, and perhaps Asia. Cases of pernicious fever may differ entirely from any of the types given :

thus, furious epileptiform convulsions, or violent dyspnœa, or a wild delirium, or an overwhelming sweat with collapse, or sudden, frightful, cardialgic pains, ending in syncope and death, may be the outcome of a malignant malarial poisoning.

Malarial Cachexia.—As the result of the continued ingestion of the poison in a malarious district, or of improper treatment, or of some peculiarity in the malarial germ which makes it with great difficulty respond to treatment, there may be developed the condition known as malarial cachexia, in which the chief symptoms are excessive anæmia and enlargement of the spleen, often with pigment deposits, secondary atrophies, or other structural changes, in various internal organs. In the vast majority of cases malarial cachexia is associated with irregularly recurring febrile paroxysms, but it is affirmed that it may exist without distinct paroxysm. It has been shown by Kelch that a single violent malarial paroxysm may cause a diminution of one million of red corpuscles per cubic millimetre; and in malarial cachexia the proportion of red blood-corpuscles sometimes falls to five hundred thousand, instead of five million, in a cubic millimetre of blood. Headache, insomnia, anorexia, dyspepsia, and various secondary symptoms are common, whilst hemorrhages from the mucous membranes and serous exudations into the subdermal cellular tissue, or into the pericardial and pleural or other cavities, are not infrequent. The liver is usually increased in size, but may be atrophic. Among the most important of the secondary diseases are nephritis, subacute or chronic, and chronic perilobular pneumonia. The enlarged spleen is usually firm and smooth to the touch, and may apparently fill up almost the whole abdominal cavity.

DIAGNOSIS.—The diagnosis of an ordinary malarial fever is easy, but in irregular malaria the symptoms may be misleading. If paroxysmal disturbances of any character recur at not very long intervals with show of regularity, malarial disease should be suspected, and an examination of the blood be made, or the effect of quinine be determined. If sufficient doses of quinine fail to influence the paroxysmal disturbance, the probabilities are altogether against such disturbance being of malarial origin. It has been affirmed by various practitioners that there are in the Southern United States malarial fevers of a continued type which are not arrested by quinine; but the nature of such cases is doubtful, and further studies of the autumnal fevers of the South, with careful investigations of the blood, are urgently needed at this time.

The most common forms of paroxysmal fever simulating malaria are those of septicæmia and hepatic disease. In any doubtful case the final diagnosis must rest largely upon the finding or not finding of the malarial organisms. If there be no detectable organisms and no response to quinine, the case should be considered not malarial. In looking for malarial organisms, the most satisfactory results are obtained by direct examination of the fresh blood. A thin cover-glass, freshly cleaned with nitric acid, then with alcohol, and finally with ether, receives a very small drop of blood from the end of the finger or the lobule of the ear, and is placed upon a thoroughly cleaned glass slide. The blood will spread into a thin layer by the weight of the cover-glass, and should at once be examined with the aid of an oil-immersion lens. The parasites may be seen with a dry lens of high power, but satisfactory results are obtained only with the immersion lens.

Staining of the organisms sometimes is advantageous. A small drop of blood is first allowed to spread out between two thoroughly cleaned cover-glasses, and these are then separated by sliding one from the other. After they have thoroughly dried, they are immersed in a mixture of equal parts of absolute alcohol and ether for half an hour. This fixes the blood firmly, and it may then be stained with methylene-blue and counterstained with eosin. Chezinski's mixture, composed of concentrated aqueous solution of methylene-blue diluted with an equal quantity of water and twice the quantity of one-half per cent. solution of eosin in sixty per cent. alcohol, gives excellent results. The cover-glasses, after being fixed, are treated with the staining solution for several minutes, then washed off, dried, and mounted in balsam. The parasites are stained blue, the red corpuscles pink.

PROGNOSIS.—Malarial fever occurring in a previously healthy subject and in the central United States, if at once recognized and properly treated, never ends in death: it is always curable, provided the nature of the disease be recognized and appropriate treatment employed; but it may be essential to remove the patient out of the malarious district in order to prevent perpetual reinfection. In tropical countries malarial diseases, especially if reinforced by continued exposure to the cause, may end fatally.

PROPHYLAXIS.—Although no prophylaxis will afford an absolute protection from the malarial organism, much can be done by those who must expose themselves by obeying the following simple rules: first, avoid going out in the early morning or during the evening or night, especially when the weather is in any degree foggy; second, sleep in the second or third story of the house; third, take from five to ten grains of quinine either directly after breakfast or on going to bed at night.

TREATMENT.—The ordinary paroxysm of intermittent fever requires no treatment, but the evening after its recognition the patient should take a full dose of calomel and podophyllin, and in the morning from fifteen to twenty-five grains of quinine, so administered that the first dose shall be taken from eight to ten and the last dose from four to five hours before the expected recurrence of the paroxysm. The exact amount of quinine given should depend upon the known obstinacy of the malaria of the district. To the adult, in Philadelphia, twenty grains may be given; farther south, thirty grains. The quinine should be repeated the second day in somewhat smaller or larger dose according to the effect of the first administration. The paroxysms having thus been broken, the patient should be put upon Fowler's solution, from three to six drops after meals, and no more quinine given until the seventh day, at which time the malarial paroxysm has a pronounced tendency to recur; to prevent this recurrence, from fifteen to twenty-five grains of the alkaloid should be administered every seventh day for from four to six weeks. The quinine must be given in solution or in encapsulated powders, or in fresh pills of the bisulphate. Old sugar-coated pills of the sulphate are not trustworthy for prompt action.

When the malarial paroxysm takes on an irregular form, brow ague, for example, larger doses of quinine are required to put it aside, so that from twenty-five to thirty-five grains should be given in the intervals and repeated in ascending doses until complete control is obtained.

The treatment of a pernicious malarial paroxysm is a matter of the greatest importance. We have found that amyl nitrite will at once put an end to the chill in an ordinary malarial paroxysm without in any way interfering with the after-development of the fever and sweat. It is, therefore, probable that the drug will prove of service in the algid form of pernicious malaria in bringing about reaction. If the central temperature during a pernicious chill be low, the hot bath should be used. When there is a distinct hyperpyrexia cold affusions may be practised, whilst at the same time external heat and mild sinapisms are freely used on the extremities. In the cases which we have seen there has been a heart-failure, which is to be met by the free hypodermic use of digitalis, strychnine, and cocaine. No time should be lost in producing a profound cinchonism, in the hope that by destroying the forming crop of parasites the length of the paroxysms will be diminished. If the stomach cannot be employed, a well-acidulated (tartaric acid) rectal injection of thirty grains of quinine bisulphate should be given, whilst ten to twenty grains of the bisulphate are given hypodermically. Two hours later, if relief has not come, the rectal injection should be repeated. At least seventy-five grains of quinine should be given within eighteen hours after the first coming on of the paroxysm, and cinchonism should be steadily maintained for a week, to be followed by the free use of Fowler's solution, with iron and other tonics, and the weekly doses of quinine.

The successful treatment of chronic malaria is often one of great difficulty. The common method of continually giving quinine in moderate dose we believe to be bad: the patient is worried by the drug and the organisms are not scathed; in our opinion it is much better to produce at intervals distinct cinchonism. Experience has shown that quinine has much more influence in these cases if given along with drugs which act upon the emunctories; in some cases potassium bitartrate does good, whilst a bitter purgative, such as aloes, given daily for a length of time in such dose as will produce soft stools is often of the utmost service where there are pronounced hepatic congestion and enlargement; mercurials, nitrohydrochloric acid, ammonium chloride, and other appropriate remedies (see Chronic Hepatitis) may be necessary before success can be expected. The arsenical preparations are especially valuable. In obstinate cases they should be given in such doses as to cause the slightest puffiness of the face or albuminosity of the urine, then for a time withdrawn, and again given *pro re nata.* Iron with a simple bitter may be administered as freely as the stomach will bear. Removal from the malarial district is often essential. It is a popular belief, whose correctness we have confirmed by observation, that going into a high mountainous country will bring out a malaria which has remained latent,—an indication that local deposits of the organism are formed in the body. Short but vigorous courses of quinine and of arsenic should therefore be given from time to time to persons who have suffered from malarial cachexia, even if the symptoms are not active. In bad cases of malarial anæmia it may be essential to put the patient to bed, and even sometimes to enforce a modified rest-cure. When there is enlargement of the spleen, iodine ointment may be used externally over the organ, whilst solid extract of ergot is given in full dose, from thirty to fifty grains a day, in capsules.

DYSENTERY.

DEFINITION.—A specific disease, characterized by inflammation of the large intestine, associated with frequent, slimy, hemorrhagic stools and tenesmus.

The term dysentery was originally applied to painful stools in contrast with diarrhœa,—frequent stools. The seat of the former was considered to be in the large intestine, that of the latter in the small intestine. In recent times research has been directed towards ascertaining the immediate cause of the dysentery, and has resulted in the recognition of a variety of exciting causes.

ETIOLOGY.—It has long been known that dysentery is likely to occur in the form either of epidemics or of endemics, and as sporadic cases. Epidemics occur particularly among collections of individuals living in close quarters, as in camps, barracks, or institutions. The disease is especially likely to occur when the surroundings are insanitary, particularly where there is exposure to cold and wet, insufficient or improper food, contaminated drinking-water, and depressing or exhausting mental, moral, or physical influences. Of persons exposed to such influences, those enfeebled by disease or excesses are chiefly liable. Dysentery is more likely to prevail during hot damp weather, as in the summer or the fall. The conditions of its origin have been found to be especially prevalent in malarial regions, and the exciting cause or causes are likely to be found in drinking-water, the injurious effects of which are enhanced by contamination with the dejections of persons already affected. The disease occurs in all parts of the world, and the variety found in tropical regions has received the term *tropical dysentery.* It is probable that most of the cases of dysentery seen in the tropics, whether endemic or sporadic, are due chiefly to the presence of an intestinal parasite, the *amœba dysenteriæ.* The occurrence of amœbæ in dysenteric stools was first announced by Lösch, and Koch found them in the tissues at the base of the intestinal ulcers and in the capillaries of the liver in the vicinity of hepatic abscesses occurring in dysentery. Osler first in America described their presence in dysenteric abscesses, and numerous observers have since confirmed this observation. Amœbæ, however, are to be found in the colon in a variety of diseases, and even in health, and may not present any morphological characteristics by which they are to be distinguished from amœbæ found in dysentery. The latter amœbæ, as shown by Kartulis, are distinguished by being pathogenic in cats.

Epidemics, endemics, and sporadic cases of dysentery arising in the temperate zones are usually attributed to bacteria, and it is also probable that the progress of tropical dysentery is favored or influenced by their presence. There is no agreement as to the existence of specific bacteria in the production of dysentery, although several observers have described the occurrence of unusually minute bacilli differing from those found in other intestinal affections.

MORBID ANATOMY.—The anatomical changes found in dysentery do not differ from those present in inflammation of the large intestine due to other causes than those specifically concerned in the production of this disease. A catarrhal, follicular, diphtheritic, or gangrenous inflammation of the large intestine may occur in a variety of diseases, as cholera, typhoid fever, scarlet fever, and small-pox, in which affections the intestinal inflammation is to be regarded as a complication of the disease in which it occurs. A diphtheritic colitis may result even from mercurial poisoning or be due to uræmia.

The lesions are usually confined to the large intestine, increasing in severity from above downward. The most extreme alterations are more frequently found in the lower portion, especially in the rectum, and in exceptional instances the morbid changes may extend into the lower part of the small intestine. In the same case milder and more severe lesions may coexist. In *catarrhal inflammation* the mucous membrane of the colon and rectum is swollen and injected, whilst punctate hemorrhages are frequent. The surface is besmeared with a viscid slime, in which epithelium, blood, or pus may be present. The submucous tissue is swollen, and often forms irregular thickenings, partly from œdema and partly from cellular infiltration. The solitary follicles are often enlarged through cellular hyperplasia, and are surrounded by a border of injected vessels. If a predominant enlargement of these follicles exists they may serve as the source of abscesses, which discharge into the intestine and cause ulcers. These increase in size and coalesce, and the mucous membrane becomes extensively undermined. To this condition the term *follicular inflammation* is applied.

A *diphtheritic inflammation* is indicated by the presence of bran-like spots or patches, the former often seated over hyperplastic follicles, the latter frequently first seen in the flexures of the large intestine and over the transverse and longitudinal bands which form the dividing lines between the pouches of the colon. The latter localization is explained by Virchow as the result of the mechanical action of fæcal masses upon the affected portions of the intestine. These patches represent superficial necroses of the mucous membrane, tending to spread in all directions, and eventually covering the entire wall of the intestine. They become discolored by intestinal contents, especially by bile and blood-pigment, and assume a dark green or greenish-brown color. These necrotic patches become *gangrenous*, and are then detached as small or large sloughs, sometimes causing profuse hemorrhage and more or less extensive ulceration of the mucous membrane. The submucous tissue becomes swollen, the muscular coat œdematous, and the inflammatory process may extend to the serous coat, the peritoneal surface of which is injected, hemorrhagic, and covered with fibrin. The mesocolon may also become infiltrated with inflammatory exudation, forming a brawny mass in which abscesses may arise, discharging into the intestine or breaking through the peritoneum and producing peritonitis. The mesenteric glands in the vicinity of the inflamed mucous membrane are hyperplastic and injected, and may become necrotic and softened.

Councilman and Lafleur have recently described in detail the alterations which occur in *amœbic dysentery.* These essentially represent an ulceration of the mucous membrane due to the presence of amœbæ in the deeper layers of the mucous membrane. An infiltration of the submucous tissue occurs, resulting in necrosis and softening of the overlying mucous membrane and the production of ulcers with overhanging edges and tending to become confluent. Amœbæ are present in the infiltrated patches and at the base of these ulcers. If purulent inflammation is present it is regarded as the result of a complicating bacterial invasion.

The catarrhal and diphtheritic inflammations of the intestine may cease at an early stage with the production of little or no destruction of the mucous membrane; but with the formation of ulcers, whatever may be their origin, the inflammatory process tends to become *chronic.* More or less extensive destruction of the mucous membrane exists, portions of which may remain as bridges or as projecting masses forming pseudo-polypi. A fibrous thickening of the remaining intestinal wall may result, with the production of induration or stricture, and the outlet of the glands may be obstructed and multiple cysts be present in the atrophied mucous membrane.

Abscess of the liver is a frequent complication of the severer varieties of dysentery, whether of amœbic or bacterial origin, although relatively more frequent in the former. Councilman has shown that the amœbic abscesses are probably due to the direct invasion of the liver by the parasite, which may produce necrosis, softening, and liquefaction of the tissues without associated suppuration. If pus is also present in such amœbic necroses of the liver, it is attributed to the associated presence of pyogenic bacteria. True abscesses of the liver occur in other varieties of dysentery, and are regarded as the result of emboli from softened thrombi in the intestinal wall or from the continuous extension of a mesenteric thrombo-phlebitis from the inflamed intestine into the liver. The hepatic abscesses are often multiple, although tending to become confluent, and frequently perforate the diaphragm, when the pus is discharged through the lung. The further history of such abscesses is described in the section on suppurative hepatitis.

Among the other occasional complications of dysentery are abscesses in remote parts of the body, suppurative inflammation of the serous cavities and joints, and endocarditis. Noteworthy possible complications of chronic dysentery are parenchymatous degeneration of the kidneys, amyloid degeneration of the spleen, liver, and kidneys, and dropsy.

SYMPTOMS.—The characteristic symptoms of dysentery may develop suddenly or be preceded by an interval of several days of disturbed digestion, associated with chilly sensations, slight fever, and mild diarrhœa. Nausea and vomiting may also occur. Tenesmus and the peculiar stools then make their appearance. The former gradually increases in severity, and may be associated with vesical tenesmus, and the pain may extend into the testes. Frequent attempts, sometimes three or four an hour, are made to empty the bowel, often with the passage of but a small amount of liquid and not affording much relief. Abdominal pain and tenderness, especially in the region of the sigmoid flexure and in the course of the colon, are frequent, usually immediately preceding the evacuation of the bowels.

At the outset the stools consist of liquid fæces, but soon they take the form of a slimy fluid in which bits of fæcal matter resembling chopped spinach appear. Such stools are the smaller in quantity the more frequent their evacuation. The dejections then become mixed with blood, resulting in the presence of a red, gelatinous substance without fæcal odor, in which specks or clots of blood may be present. The gelatinous material may be in clumps, like boiled sago, and the abundantly hemorrhagic stools may be watery, resembling beef-juice. With the progress of the inflammation opaque-yellow streaks and clots indicate pus, and the greater their abundance the more probable is the existence of ulcers. The presence of the latter is further made evident by the appearance in the stools of shreds of tissue. In the milder cases in the course of a few or several days the stools become more fæcal and less frequent. The tenesmus diminishes and disappears. The appetite and strength return. In the severer cases the stools may become very offensive, the tongue dry and cracked, the pulse feeble, the temperature moderately elevated, perhaps subnormal, and in the severest cases there may be irregular chills and fever, profuse sweating, inflammation of the joints, and endocarditis. Paralysis sometimes results, due to a peripheral neuritis. In such cases a long period of convalescence exists.

The symptoms of amœbic dysentery closely resemble those of other varieties of this disease. The course, however, is more irregular, with frequent remissions and exacerbations. Abdominal pain and tenesmus are less frequent and severe, and the stools, though of similar gross appearances, are less frequent and more copious. The presence of amœbæ is their especial characteristic. They are to be found, especially in the blood-stained mucus, as motive masses of protoplasm, especially active in alkaline stools, five or six times as large as red blood-corpuscles, and not infrequently containing the latter within their substance. Periods of constipation may alternate with those of diarrhœa. Even in the milder cases the course is protracted over several weeks, and the patient becomes anæmic, weak, and thin.

Any variety of dysentery may assume the chronic form, of which the principal symptom is diarrhœa. The stools are composed largely of liquefied fæces in which gelatinous particles may be present, but without blood and with relatively little slimy material. Tenesmus and abdominal pain are infrequent. Chronic dysentery may extend over a period of years, during which time there may be intervals of comparative comfort and well-being alternating with exacerbations, in which the acute symptoms already mentioned may return. Usually a normal appetite and undisturbed gastric digestion enable the patient to resist the debilitating effects of the disease, although the longer it persists and the more frequent the exacerbations the greater the loss of flesh and strength.

DIAGNOSIS.—Dysentery is to be diagnosticated by the presence of characteristic stools, tenesmus, and pain in the course of the large intestine. Bloody stools from hemorrhoids or rectal polypi occur without tenesmus, and a rectal examination will frequently permit the exclusion of tubercular, syphilitic, or cancerous ulcers of the rectum, a source of rectal pain, if not of tenesmus. Amœbic dysentery is to be recognized by the discovery of motive amœbæ in the stools. Chronic dysentery is to be recognized by the persistence of diarrhœa, the frequent presence in the stools of sago-like grains, and the occasional presence of pus or blood. The discovery of amœbæ in the stools of chronic diarrhœa is evidence of the limitation of the inflammation to the large intestine, as well as of its nature.

PROGNOSIS.—The mortality in dysentery varies in accordance with the circumstances of its development. The milder cases terminate favorably in a fortnight. The severer cases extend over a period of several months, while chronic dysentery may be continued over a period of years. Epidemics in armies may show a mortality of fifty per cent. and upward, while the average fatality in endemics may not exceed ten per cent. In recent years the mortality among English soldiers in India and in Egypt, suffering largely from amœbic dysentery, has ranged from one per cent. to five per cent. The prognosis is more serious in persons enfeebled by age, disease, or intemperance. Unfavorable signs are copious, bloody, and offensive, putrid stools, abscess of the liver, peritonitis, and arthritis. The ultimate prognosis of chronic dysentery is grave from the liability to recurrences, the tendency to eventual emaciation and debility, and the occasional occurrence of amyloid degeneration or intestinal obstruction.

Treatment.—For the purposes of therapeutic discussion dysentery may be divided into the mild epidemic or sporadic form, the disease commonly seen in the Northern United States and similar temperate zones, tropical dysentery, and the typhoid dysentery of camps and other crowded places. The subject may be further divided into the hygienic treatment, the general medical treatment, and the local treatment. Experience seems to show that, whilst general medical treatment varies, the hygienic and the local treatment are the same for all forms of the disease.

Every case, even apparently the simplest, of dysentery should be looked upon as a serious disease, and the patient put at once to bed upon rigid diet. Strict disinfection of the fæcal passages should also be enforced as in typhoid fever.

In mild cases warm milk, rice, milk toast, and barley gruel may be allowed. In severe cases animal broths, including chicken jelly, should make up the whole dietary, or perhaps be supplemented by the careful use of raw eggs or of milk. Whenever milk is used care should be exercised to prevent the formation of large curds in the gastro-intestinal tract. An ounce of lime water should be added to every eight ounces of milk, or the milk should be partially predigested. It should be taken in moderate quantities at short intervals (two to three hours), and drunk slowly. No spice or condiments should be allowed, and even salt must be used with great moderation. As convalescence progresses and the diet is increased, white meat of chicken, sweetbreads, lamb, and other tender, easily digested meats are to be preferred to starchy, saccharine, or vegetable foods. Pulled bread* or thoroughly toasted stale bread should be the first starchy food allowed.

In the mildest cases of ordinary dysentery a single large dose of castor oil with a little opium may be administered with immediate relief. In the more severe cases the choice is between the mercurial and the saline treatment. We prefer to give one-quarter grain of calomel with one grain of ipecacuanha every one, two, or three hours, *pro re nata*, reducing the ipecacuanha if nausea be produced, until large, bilious passages are obtained. In accordance with the saline method, one drachm of Epsom or Glauber's salt may be administered every three hours until free purgation results. Usually relief follows the action of these remedies, aided by local treatment. If it do not, salol (five grains) and bismuth (fifteen grains) may be administered every three hours, the saline or other laxatives being repeated from time to time if the passages tend to return to their original character. In a very obstinate case the ipecacuanha treatment should be tried.

---

* "Pulled bread," a very useful article of diet, is made by cutting an ordinary loaf in half, pulling out of the soft part with the fingers long pieces, two or three inches thick, and rebaking them for fifteen minutes in a very hot oven.

Tropical dysentery is best treated with ipecacuanha given in large dose. Vomiting is almost always produced, but, according to our experience, is to be avoided as much as possible, although many tropical practitioners prefer to begin treatment with a powder of thirty grains of ipecacuanha. Our method is to give one-sixth grain of extract of opium, followed in fifteen minutes with from five to ten grains of ipecacuanha in capsulated pill, this being repeated every two or three hours, according to the urgency of the case, until absolute intolerance by the stomach of the drug is produced, or large, usually blackish, discharges from the bowels take place. These are almost invariably followed by relief.

In the treatment of any form of dysentery, astringents, full doses of opium, and all other remedies of a constipating character are to be absolutely avoided. In camp or adynamic dysentery the ipecacuanha treatment is probably, on the whole, the most successful, but in many cases the adynamia is so extreme that support and stimulants are essential. No more alcohol, however, should be given than is necessary Strychnine is especially valuable. In this, as in other forms of the disease, if the temperature rises above 103° F., cold applications are of service, and even cold baths should be used.

In all forms of acute dysentery the local treatment is exceedingly important. In robust, severely attacked individuals leeching around the anus may sometimes be practised. Counter-irritation over the abdomen is of service, but should be of such mild type (spice plasters, mustard poultices, 1 to 8) that it can be steadily maintained hour after hour. In the height of the disease great relief, and even permanent good, are often effected by ice suppositories (small pieces of ice shaped like an ordinary suppository), inserted for a length of time, one after another, as fast as they melt. These may be supplemented by large injections of ice-cold water given at short intervals, or of hot salt solution. The tenesmus may be largely controlled by opium and belladonna suppositories, but, as these by drying up secretion may be harmful, they should be used as sparingly as possible. In many cases, after the local use of cold, continuing relief can be obtained by suppositories of iodoform, three grains each. Disinfection of the large intestine by means of large injections containing bismuth, salol, or silver nitrate, theoretically should afford brilliant results. Bismuth we have tried with good results; but with salol and the silver salts we have had no experience in the acute disorder. In all cases the parts about the anus should be well washed and greased (vaseline or cold cream) after each passage, to prevent excoriation.

In the treatment of chronic dysentery a heavy woollen abdominal bandage should be worn day and night. The diet should be rigidly restricted to milk, strong animal broths, tender meats (except pork, veal, tame turkey, ducks, and geese), pulled bread, and toast. Substances containing tannic acid, lead preparations, camphor, and other astringent remedies should be absolutely avoided, unless the discharges become so frequent and severe that they must be checked to save the general strength. Even under these circumstances it should be recognized that the astringents increase the local disease. Of these astringents lead is probably the least harmful. Salol and bismuth preparations are often of service if given in very large doses one and a half hours after eating,—*i.e.*, at a time when the food is flowing towards the large intestine, which they must reach to be of service. Silver nitrate given by the mouth is probably of little value, because of its certain early decomposition. The chief reliance for cure must be upon the local treatment by means of large enemata, which should fill the whole colon. The milder local remedies, such as bismuth and resorcin, may in this way be brought in direct contact with the mucous membrane. Most brilliant results have in our hands been obtained by large injections of silver nitrate (from thirty to sixty grains) in two quarts of water. We have never seen any constitutional or severe local symptoms produced by these large doses. The injection usually is expelled in from three to five minutes. In giving it, however, the practitioner should always have at hand a saturated solution of ordinary salt, and if the bowel fail in a few minutes to discharge its contents he should decompose the excess of silver by using its chemical antidote. In giving the injection the patient should lie upon the back, with the legs drawn up and the hips so placed upon a hard pillow as to elevate the pelvis. The rectal tube should then be inserted five to six inches, and by means of the fountain syringe the fluid allowed to flow in without force.

In some cases of chronic dysentery entire change of life is essential; and when there have been wide-spread ulceration and destruction of the mucous membrane through the disease-processes of years, cure is hopeless.

CHOLERA. CHOLERA ASIATICA.

DEFINITION.—A contagious disease, produced by the comma bacillus of Koch, and characterized by violent serous purging, rapidly followed by collapse.

ETIOLOGY.—Asiatic cholera is endemic in India, whence reports of its existence appeared in European literature as early as the sixteenth century, whilst a good description of it was given by Bontius in 1629. It always exists in India; indeed, apparently it is the chief instrumentality in keeping down the surplus population, having, according to Annesley, between 1817 and 1840 destroyed eighteen millions of Hindoos. Cholera first started on its great world-travels in 1817 and 1818, reaching China in 1821, and in 1823 the borders of Europe, where it ceased for the time to progress. In 1830 it invaded the Crimea by the route of the Caspian; in 1831 it reached Hamburg, in 1832 London and Calais, and the same year, through Quebec and New York, it invaded the American continent, where there were local recurrences of the disease as late as 1835 and 1836. In 1844 cholera again set out upon its march by the route of Persia and the Caspian Sea, reaching St. Petersburg, Hamburg, and the French seaports in 1848, and entering the United States the same year through New Orleans, spreading up the valley of the Mississippi as it had in 1842 travelled down that watercourse. In 1851 partial outbreaks occurred in Europe, and in 1854 the disease reappeared in New York and widely travelled through the United States. In 1865 a new cholera wave entered Europe through Arabia, rapidly spreading to the seaports of the Mediterranean, and reaching the United States in 1866. In 1873 a few cases appeared in the United States, and in 1884, and again in 1892 and 1893, European epidemics sent scattered cases to the United States, which, however, by careful sanitation were prevented from becoming the centres of serious outbreaks.

In all these epidemics the route of the disease was along great lines of travel. Not only did the disease-wave follow watercourses, but railroads and caravan routes; over oceans and seas, through valleys, over water-sheds, across deserts, the cholera passed along with its human prey. The yearly pilgrimage of Mohammedan devotees to Mecca still remains a permanent menace to the civilized world.

In 1884 Koch discovered the cause of cholera, an actively motile, flagellate, curved bacillus, the "comma bacillus," which is about half the length of the bacillus of tuberculosis and is considerably thicker, and which under certain conditions forms long, winding, spiral fibres, and hence is a spirillum. According to the observations of Hueppe, frequently two small, spherical bodies form in the spiral threads and continue to increase in number until the whole thread is resolved into minute round cells, cohering by a jelly. These so-called "arthrospores" resist desiccation and other injurious influences much better than does the comma bacillus, and under favorable circumstances develop into the comma bacillus. They appear, therefore, to be a permanent form of the cholera organism, and it is probably largely through their influence that the disease is spread. The cholera organism develops rapidly in sterilized water, in milk, and in various organic solutions, provided these be not acid. It is easily destroyed by various bacteria, by acids, by germicides, and by a temperature of 130° F. It exists in immense quantities in the alvine discharges of cholera patients, and has been detected in drinking-water, milk, and various foods. Dogs and guinea-pigs fed upon this bacillus do not suffer unless they are so fed and medicated as to overcome the excessive natural acidity of the gastro-intestinal juices, when the cholera bacillus is capable of causing in them death preceded by cholera-like symptoms and lesions. The comma bacilli are never found in the blood or general tissues, although they enter the epithelial cells and basement membranes of the intestine. As the comma bacillus exists in the human body only in the primæ viæ, escapes from the human body only with the alvine discharges, and is incapable of producing cholera when injected hypodermically, infection must take place through the mouth. For such infection it is necessary for drinking-water, food, or other medium of transmission to become contaminated, directly or indirectly, with the alvine discharges.

The exact relation of the spores to the genesis of the disease is uncertain. Pettenkofer believes that it is necessary for the cholera poison to undergo further development in an appropriate soil before it can acquire fresh pathogenic potency, basing his belief upon the fact that the disease is very rare on shipboard, and that certain places, such as Munich, situated on rocky soil, enjoy immunity. A porous soil with high ground-water and much organic matter certainly does seem to favor the development and diffusion of the cholera bacillus. Whether under these circumstances the spores form and multiply in the soil is not known.

MORBID ANATOMY.—The appearances vary somewhat as death occurs early or late in the disease. In the former case rigor mortis is unusually pronounced and prolonged. Granular degeneration of the heart, liver, and kidneys is present. The pleuræ, pericardium, and peritoneum are besmeared with a viscid fluid, and sometimes show hemorrhagic patches. The spleen is but little altered. The principal changes are to be found in the intestine and in the liver. The former contains more or less rice-water material, and the mucous membrane, especially of the small intestine, is injected and swollen, and of a velvety appearance from enlargement of the villi. The solitary follicles and Peyer's patches are enlarged, and the latter may present a sieve-like appearance from maceration and exfoliation of the superficial epithelium. The mesenteric glands also may be swollen, injected, and soft. The kidneys are pale, and the epithelium of the convoluted tubules is necrotic and disintegrated. The mucous membrane of the uterus is often injected; its cavity contains more or less bloody material. If death occurs at a later stage of the disease, rigor mortis is less extreme. Hypostatic œdema and injection of the lungs are frequent. The appearances of the intestine are like those above described, but less extreme. There is fatty degeneration of the epithelium of the convoluted tubules of the kidney.

SYMPTOMATOLOGY.—The incubation period of cholera appears to vary from a few hours to as many days. The first symptom is a diarrhœa, which is usually not accompanied by pain, but with much borborygmus and a constantly increasing serosity of the stools. With this there are usually general malaise and loss of appetite, but no febrile reaction. In some cases there is epigastric and even severe abdominal pain. After from a few hours to six days the second period of the disease may be considered to be entered upon. In this the dejections are extremely frequent and profuse, free from fæcal odor, and almost colorless, having floating through them epithelial débris, the whole resembling rice-water. During this period there are also increasing vomiting of rice-water liquid, excessive thirst, violent abdominal pain and cramp, and finally agonizing cramps in the extremities. The pulse is small and frequent, the bodily temperature lowered, the surface cold. This period may last from one to two days.

In favorable cases the stools become less frequent and smaller, the cramps disappear, and gradually the patient recovers. In unfavorable cases the subject passes into the so-called algid period ; the passages may be less abundant and less frequent, or may even cease, but extreme headache, suppressed or whispering voice, intense anxiety, vomiting, cramps in the extremities, great fall in the temperature of the surface, and especially of the feet and hands, a progressive failure of oxygenation of the blood so that the skin becomes more and more cyanosed until the nails are black, a disappearing pulse, and suppression of the renal secretion,—these make up a series of symptoms which end in complete collapse, sometimes associated with coma, more frequently with a peculiar condition of consciousness in which there is entire indifference as to the result. Although the surface of the body and of the extremities is uniformly cold, the rectal temperature varies ; it may be normal, slightly elevated, or depressed. In three-fourths of the cases death occurs during the period of algidity.

When amelioration takes place, the cyanosis disappears, the skin becomes warm, the urine increases in quantity, the pulse regains its force, the respiration becomes regular, and the cramps diminish, and so by a steady progress convalescence is reached in from ten to fifteen days. Not rarely this period of reaction is less favorable ; the anuria may persist, or more frequently typhoid symptoms may develop. In rather rare cases distinct fever, epistaxis, and even bilious diarrhœa appear. In another set of cases the symptoms of the period of reaction resemble those of meningo-encephalitis, with fever, excessive agitation, violent headache, irregular, convulsive movements, bounding pulse, and usually after one or two days death in coma. Recovery, however, is possible, even when the symptoms have seemed hopeless.

Various cutaneous eruptions, as urticaria, erythema, or roseola, develop during the period of reaction in about four per cent. of the cases. More serious are the pneumonias and other pulmonic complications, which are not rare. Convalescence is usually protracted, and almost always accompanied by dyspepsia and often by rebellious diarrhœa. Neuritis, tetany, especially after childbirth, furunculosis, and glycosuria, are among the sequelæ which occasionally occur.

The mildest forms of cholera are those which are known as *cholerine*, in which the only symptom is a slight diarrhœa ; the most severe forms, those in which there is an abrupt development of great bodily weakness with vertigo, followed in a very few hours by fatal collapse, which may be preceded by a violent intestinal flux, or in certain cases (*cholera sicca*) by a paralysis of the intestines which causes retention of the secretions, so that, although nothing has come from the body during life, after death the bowels are found full of rice-water liquid. Between the two extremes every grade exists.

Diagnosis.—During an epidemic of cholera every case of serous diarrhœa should be considered as one of cholera, and so treated with the utmost care. So far as the symptoms are concerned, there is no difference between cholera, cholera nostras, and various metallic poisonings, notably the antimonial and the arsenical. The finding of the comma bacillus is the only complete demonstration that a case is or has been one of Asiatic cholera; for even the lesions found after death from one of the diseases which simulate cholera may be indistinguishable from those of that disease.

Prognosis.—In the beginning of an epidemic of cholera the mortality usually ranges from forty to sixty or even seventy per cent., but as the epidemic progresses, either because the pathogenic agent loses its virulence or because it is the most susceptible who are first attacked, the fatality steadily diminishes. In individual cases the prognosis must always be guarded, since the mildest diarrhœa may suddenly develop an irresistible force, whilst, on the other hand, it is not rare for patients to react from the most desperate conditions. During the period of reaction any irregularity of symptoms or any appearance of cerebral or pulmonary complications is of the gravest import. The very young, the very old, the alcoholic, the insane, and persons weakened by previous chronic disease, all die from cholera in extraordinary proportion.

Prophylaxis.—From the nature and life-history of the cause of cholera, it is evident that absolute shutting out of the germ by quarantine will suffice to prevent the spread of the disease. What is theoretically easy, however, is in many cases practically impossible; but whenever an isolated case does enter an unaffected district, the utmost vigilance should be exercised to see that all the fæcal discharges are thoroughly disinfected, and that all body- or bed-linen and every garment with which they may have come in contact are either destroyed or disinfected beyond question. Absolute cleanliness will aid in arresting the spread of cholera, but will not atone for carelessness in allowing the escape of the germ. In no other disease is personal prophylaxis so effective as in cholera. For personal infection it is necessary that the germ be taken into the mouth and into the stomach, so that theoretically it is possible to live in daily contact with cholera patients without evil result.

The precautions must be absolute in their rigor: weakness in a single link or particular may do away with the value of the whole procedure.

The hands must be frequently and thoroughly washed and disinfected, especially after handling the sick or the bed-clothing from them. The food must be taken directly after it has been disinfected by fire ; the diet, therefore, must be restricted to meats, hot bread, cakes, or toast, and such other articles as shall come from the fire directly to the table, and be eaten as hot as can be borne by the palate. It is, of course, essential to avoid all indigestible food and everything tending to produce gastro-intestinal catarrh, as this condition of the alimentary canal would greatly favor the development of a stray germ which might break through the cordon of defence. No water should be taken except that which has been well boiled and is still hot, or that which has been immediately taken out of bottles into which it was put before the epidemic. It is also essential that the dishes for food and drink immediately before using be heated to such a temperature as to destroy any adhering germs. Some years ago a violent outbreak of cholera in the Insane Department of the Philadelphia Almshouse was arrested within twelve hours, without the precautions just spoken of, by the free administration of sulphuric acid lemonade. The only new case was that of a man who refused the prophylactic. In the surgical wards of the same institution the acid was used from the beginning of the epidemic, and in these wards, although in no way isolated from the other departments, there was absolute freedom from the disease. It has been experimentally proved that in dogs and guinea-pigs susceptibility to the cholera germ can be produced by rendering their highly acid stomachs alkaline, and there is reason for believing that it is possible to render the human primæ viæ an unfavorable habitat for the cholera bacillus by making them abnormally acid.

Treatment.—During an epidemic of cholera it is essential that every case of diarrhœa be treated with the utmost care, and it is certain that many cases of the disease may be thus arrested before the cholera bacillus has full possession of the intestinal tract. The patient should be put to bed, and the diet confined to strong broths and meat-essences, whilst such doses of aromatic sulphuric acid or the aromatic sulphuric acid diarrhœa mixture as may be necessary should be given. In some cases it would probably be of great service to wash out the large intestine thoroughly with distinctly acidulated water. Hayem recommends lactic acid, five drachms in twenty-four hours, in cholera, or, as a prophylactic, a drachm and a half daily well diluted. The dilute or aromatic sulphuric acid has the advantage of astringency : two drachms of it may be given in the twenty-four hours without any unpleasant effects. Naphtol, strontium salicylate, bismuth subnitrate, and other intestinal antiseptics should be freely used. Opium suppositories should be given *pro re nata*, especially when there is vomiting. Bismuth salicylate is particularly commended by some French authors, and may be efficacious.

The same treatment should be kept up during the second period of cholera.

During the algid stage of cholera the patient should be required to drink very freely of hot water, with or without the addition of small quantities of alcoholic stimulants, according as the water is made thereby more or less acceptable to the stomach. External heat should be freely applied to the extremities, or, if the temperature be below the norm, the whole body should be immersed in a bath of 105° F. As long ago as 1832 Lizars practised filling the large intestine with hot water during this stage, and the practice has recently been imitated with alleged excellent results. Instead of the saline solution sometimes employed it might be better to use an acid solution for the purpose of destroying the cholera bacillus, and a solution of tannic acid has been especially commended. In 1832 Th. Latta strongly urged the use of intravenous injections in the later stages of cholera, and their value seems to be established. They are apparently harmless, since Hayem has found that he could double the daily amount of blood in the dog by their means without producing any distress except a temporary irritation of the kidneys. Hayem's formula consists of one thousand parts of distilled water with five parts of sodium chloride and ten parts of sodium sulphate. We should prefer the saline solution without the sodium sulphate. The injection may be slowly made into the saphenous vein by means of a fountain syringe, the greatest care being exercised to see that the injected liquid and the apparatus used have been absolutely sterilized by heat. There is commonly an immediate reaction, with relief of the urinary suppression, but in most cases the symptoms return in the course of a few hours, and, although two or even three injections may have been practised, the ultimate result is affected in only a few cases. Instead of throwing the solution into the vein, it may be injected into the cellular tissue of the buttock. A large quantity can be thus taken and rapidly absorbed. Moreover, the process can be repeated until the result is secured or the method proved inefficacious.

During the stage of reaction symptoms should be met as they arise. The diet should be the simplest and most non-irritating possible, and the recurrence of diarrhœa should be strenuously guarded against.

CHOLERA NOSTRAS. CHOLERA MORBUS.

This disease may be with propriety noted in this place because the symptoms may exactly counterfeit those of cholera, and because it has been maintained by Guérin and others that it is really the sporadic form of Asiatic cholera. The symptoms often develop with great suddenness, and consist of violent vomiting and serous diarrhœa, usually without pain, although there may be great abdominal distress. The stools, at first fæcal, become more and more watery, until they may be undistinguishable from the rice-water stools of Asiatic cholera. In the severe cases there is great weakness, with whispering voice, rapid, small pulse, and cold, livid extremities, ending, it may be, in a fatal collapse.

Cholera morbus occurs exclusively in adults, or, to speak correctly, this acute serous diarrhœa occurring in adults is known as cholera morbus, in children as cholera infantum. It was affirmed in 1884 by Finkler and Prior that the disease is dependent upon a comma bacillus not to be distinguished from that of Asiatic cholera. It was, however, proved by Koch that the two organisms are distinct, and it is not certain that the bacillus of Finkler and Prior is the sole cause of cholera nostras. In three cases Gilbert and Girode have found the bacterium coli commune abundant in the stools.

The most probable view is that cholera morbus is a serous diarrhœa which may be provoked by various causes. As at least one variety, *cholera infantum*, must be looked upon as a form of thermic fever, so some adult cases may be instances of neuro-paralytic diarrhœa produced by excessive heat. Again, cholera morbus, or at least the congeries of symptoms which bears that name, may be caused by various metallic poisons. Further, we have seen the symptoms of cholera morbus induced by violent emotion.

Cholera nostras as it occurs among adults in this country is rarely fatal except in subjects who are weakened by previous disease or are of feeble constitution. It is best treated by the hourly administration of small doses, one-tenth of a grain, of calomel, with the free use of opium suppositories and of intestinal stimulants, such as camphor, the volatile oils, and chloroform. (See formula 9.) Abdominal mustard plasters should also be freely used. When there is fall of temperature the hot bath, when there is elevation of temperature the cold bath, should be employed. So soon as under the influence of calomel the passages become brownish the drug should be withdrawn. Convalescence usually follows at once.

YELLOW FEVER.

Definition.—An acute febrile disease, characterized by fever lasting from one to four days, followed by an intermission, with in severe cases a secondary exacerbation, a steady fall of the pulse, which commences during the period of fever, jaundice, a tendency to stasis of the circulation and to hemorrhage, and parenchymatous inflammations of the liver, kidneys, and stomach.

Etiology.—The question of the contagiousness of yellow fever has been investigated and discussed most extensively, so that at present it seems established that the disease is incapable of passage directly from man to man, but that the poison, whatever its nature may be, passes from the sick into some favorable locality where it develops the activity which enables it to infect another person. For the growth and development of the poison outside of the body certain conditions are necessary: these conditions probably are, first, a steady well-maintained temperature; second, the presence of filth.

It has been a wide-spread belief that this filth must have at least some animal matter in it, and that excrementitious material is especially fit for its development. It would seem that the most favorable conditions are the existence of high temperature and the presence of such mixed masses of vegetable and animal filth as prevail about seaports. The effect of cleanliness was strongly illustrated in the banishment of yellow fever from New Orleans by the rigid military sanitation enforced by General B. F. Butler during the civil war. The usual history of an epidemic is a dirty town, a single imported case, an outbreak of disease. In an instance reported by Guitéras, the man, moving from an infected district, had fever in his own house, which was kept clean, with no spread of the disease; during early convalescence he went to another village and lived in a dirty room, which room became a source of infection for a number of cases. In another case the clothing of a sailor dying of the disease was packed in his chest and sent to his wife in New York; two people were present at the opening of the chest, and were both infected.

Numerous bacilli and other organisms have been found in the discharges and tissues of yellow fever patients, but we have no knowledge of the nature of the germ, which is generally believed to be, however, an animal organism, whose life-history comprises at least two different developmental stages,—one inside, one outside, the body.

The countries which are infected by yellow fever are divided by Guitéras into—(1) the focal zone, in which the disease is practically endemic; (2) the perifocal zone, with periodic epidemics; and (3) the zone of occasional epidemics. Number 1 includes the important seaports of Havana, Vera Cruz, Matanzas, Rio Janeiro, and a small portion of the Atlantic African coast. Number 2 comprises the African Atlantic coast, a few seaports of the American Pacific coast, and the majority of the subtropical Atlantic United States seaports, including New Orleans. Number 3 includes all countries bordering on the Atlantic Ocean between 45° north latitude and 35° south latitude, below an altitude of fifteen thousand feet, not included in numbers 1 and 2. The epidemics habitually travel from the sea-coast along watercourses or along lines of railroad.

Race characteristics have been believed to be dominant in the causation of yellow fever; it has been held that, although white races are extremely susceptible to the poison, the white inhabitants of Cuba and other places where the disease is endemic are incapable of taking it. Guitéras, however, has shown that this is not because of inherited peculiarities, but because yellow fever is essentially a mild disease of childhood, and that in Cuba all the children have had attacks the nature of which has been heretofore unrecognized, but which have afforded protection in after-life.

MORBID ANATOMY.—The skin, subcutaneous tissues, and viscera are bile-stained. Large and small hemorrhages are frequent in the skin, in the gastro-intestinal and urinary mucous membranes, beneath the pleuræ, pericardium, and peritoneum, and within the lungs. The stomach contains coffee-grounds material which is largely composed of blood-corpuscles and blood-pigment. The heart, liver, and kidneys show the appearances of more or less advanced parenchymatous degeneration. The muscular substance of the heart is granular or fatty; in the latter case the myocardium is opaque yellow and flaccid. The liver is either enlarged or diminished in size, of an opaque yellow color, its cells granular, fatty, or necrotic. The kidneys are enlarged from swelling of the cortex, the epithelium of which is also granular, fatty, and necrotic. The spleen is comparatively free from alterations, although sometimes unusually soft.

SYMPTOMATOLOGY.—The period of incubation varies from a few hours to fourteen days, although it is very rare for the disease to develop after the ninth day. In a Florida epidemic nine hundred unacclimated individuals who had been exposed to the poison were quarantined for ten days and then went into healthy districts; not one of them developed the disease after the discharge. The invasion, which occurs more frequently at night, is abrupt, with repeated chills, excruciating pains in the back, head, and limbs, and an immediate rise of temperature. Vomiting is very common, and in some cases an exanthematous rash appears, especially in the scrotal region. There is usually an evening exacerbation of temperature on the first day, but on the second or third day (rarely even on the first) the characteristic fall of temperature begins and continues, though sometimes interrupted by evening exacerbations until it reaches the norm from the second to the fourth day.

During the whole period of the fever there are great anxiety, restlessness, intense suffering, and not rarely delirium, varying in degree from slight mental confusion to wild mania; in some cases there is stupor. When the remission occurs not only does the fever subside, but the pains usually disappear; the mind becomes clear, and not infrequently all anxiety is lost; there remains, however, an increasing epigastric tenderness, with continuing and increasing slowness of the pulse, perchance a little heaviness; soon jaundice appears, first generally in the forehead and conjunctiva, and rapidly increases until the whole surface is dark yellow and the deep-brown urine is heavily loaded with biliary constituents.

The period of remission may end in convalescence, but commonly there is developed a second paroxysm of fever with well-marked diurnal remissions, and sometimes hyperpyrexia. Even during the remission the failure of strength is usually marked, but in the fever of reaction, as it is called, the adynamic symptoms become more pronounced. Death may occur during the secondary fever, or, after a prolonged, irregular course, by gradual abatement of symptoms the patient may pass into convalescence.

In severe cases the jaundice deepens until the whole surface is uniformly bronzed. The vomiting recurs, and becomes uncontrollable, whilst brownish or blackish flakes appear in the matter ejected and increase in number until the whole fluid is black and opaque. The capillary circulation becomes so nearly stagnant that the dependent and extreme portions of the body, fingers, toes, scrotum, back, etc., are deep purplish. The pulse grows more feeble and irregular. The urine lessens in quantity, and may be completely suppressed. Hemorrhages occur from the various mucous membranes, even from the gums. Petechiæ, vibices, hæmaturia, bloody stools, and an intense apathy mark the complete degradation of the blood and the failure of the vital power, which deepens until a quiet death results.

Whilst the course of yellow fever is for the most part fairly uniform and consistent, the cases vary in intensity from the mildest to the most severe type. Sometimes the patient is struck suddenly with stupor and coma; sometimes walking in the streets he may be found to be pulseless, and soon develops jaundice and black vomit, ending in death; whilst in other cases, with natural tongue and natural pulse and general calm, he passes abruptly into a condition of black vomit and fatal prostration.

According to Guitéras, in young children yellow fever may be a very trivial disease, and even when severe is so lacking in characteristic symptoms that it is commonly diagnosed as an ephemeral or a thermic fever, or as a malarial attack.

The black vomit consists of gastric mucus with altered blood-corpuscles, epithelial cells, bits of food, various fungi, and black amorphous granules, evidently the last results of blood disintegration. Although there is without doubt an increased production of urea in the fever stage of the disease, yet, according to the researches of Cunisset, the elimination of urea is always less than normal, the degree of diminution being in direct proportion to the danger of the disease, and affording a very important element of prognosis. There is also a lessening in the elimination of the chlorides, phosphates, sulphates, and other inorganic salts. The amount of albumin in the urine is usually directly proportionate to the severity of the attack, but it is possible for a case to go on to death with an abundant secretion of non-albuminous urine. Severe albuminuria is connected with a diffused nephritis, and is generally accompanied by abundant casts. During convalescence parotitis, abscesses. diarrhœa, and other local disorders may be very troublesome.

Diagnosis.—The symptoms of yellow fever in their ordinary development resemble more closely those of certain malarial fevers than of any other disease, and in the early days of an epidemic it may be impossible to decide from the symptoms alone whether the patient is suffering from malarial poisoning or from yellow fever. The diagnosis must be made by determining whether the malarial organism is or is not in the blood. The mild cases of yellow fever, such as occur in children, are distinguished from thermic or ephemeral fevers by the peculiar icteric hue of the face, by the slowness of pulse in proportion to the bodily heat, and sometimes by albuminuria.

PROGNOSIS.—The mortality of yellow fever varies in adults in different epidemics from fifteen to ninety per cent. The disease is about twice as fatal in adults as in children. The existence of previous chronic disease or of alcoholism enormously increases the danger. The mortality-rate is usually much higher in hospital than in civil life, probably on account of the exposure and hardships during the earliest hours of the disease. Most favorable is a uniform lack of intensity of all the symptoms. If fever, jaundice, or renal disorder is pronounced, the outlook is serious. An initial temperature of 103° F. is a bad omen, as is also a rapid increase of albumin on the third day. Black vomit usually, but not always, presages death. Violent nervous disturbances, such as delirium or convulsions, and still more the suppression of urine, are of fatal import.

PROPHYLAXIS.—Absolute exclusion of the germ of yellow fever from any locality is an absolute preventive of the fever : hence the importance of a most rigid quarantine, the isolation of the sick, and the complete disinfection of clothing, excreta, etc. It is of the utmost importance that infected districts be immediately depopulated. The only individual prophylaxis that is of any value is keeping away from the affected locality. There is no reason for believing that the various preventive inoculations that have been lauded by Freire of Brazil and Carmona of Mexico are effective.

TREATMENT.—In yellow fever districts there is still much faith in the value of violent sweating, calomel, and similar perturbing agencies in the earlier stages of the fever. It seems probable, however, that the best results are to be obtained by the expectant treatment, including in this absolute rest in bed, especially during the remission, the administration of mild saline laxatives, the free internal use of ice-water, the external use of cold sponging and baths to reduce temperature, and the meeting of the symptoms of adynamia by stimulants. The use of bichloride of mercury internally, as suggested by Sternberg, with the idea of destroying the supposed specific agent of the disease in the intestines, does not appear to have yielded practical results. Guitéras strongly urges the use of the tincture of chloride of iron as the best means of checking the tendency to black vomit in severe cases.

When the stomach will bear it, food in the form of broths, milk, eggs, etc., should be freely administered.

Suppression of urine is to be met by the use of hot baths, digitalis, pilocarpine, and other usual remedies, the value of which in yellow fever cases, however, still remains in doubt.

ACTINOMYCOSIS.

DEFINITION.—A chronic disorder, especially attacking cattle and the pig, but capable of transmission to man, produced by the ray-fungus.

ETIOLOGY.—Actinomyces, or ray-fungus, is an organism which was discovered by Bollinger in 1877, and is considered by some authorities to be a highly organized pleomorphic bacterium allied to cladothrix, by others to be a fungus: it produces in man and the lower animals a chronic inflammatory affection, whose nature can usually be made out by detecting even with the naked eye granules varying in size from a small pea to a point, and in color from a translucent grayish to opaque gray, yellowish, or brownish, even to black. Examined with the microscope the granule is found to be a colony or clump of colonies, consisting of a central interlacing mass of branching and radiating projecting threads, with in the centre small, round, cocci-like bodies, and upon the periphery the bulbous, club-like terminations of the threads. The cocciform bodies are supposed by some to be spores, and are found both within and on the outside of the threads. Usually about the colonies are numerous bacilli of various species, and also pyogenic cocci. According to the researches of J. Israel and M. Wolff, the actinomyces grow best anaërobically. Great difficulty has been experienced in producing, either with the natural actinomyces or with the result of their culture, disease in the lower animals, but Israel and Wolff appear to have succeeded in the development of nodules containing typical colonies by introducing pure cultures into the peritoneal cavities of rabbits and guinea-pigs. Infection of human beings and of cattle has been frequently traced to the penetration of wheat, barley, oats, or other vegetable material into the tissues. Usually the part affected is the mouth, especially the neighborhood of the teeth, but the entrance may be through a wound of the skin or of any mucous membrane. The disease chiefly occurs in cattle, constituting in its ordinary form the so-called *big-jaw* or *lump-jaw*.

SYMPTOMATOLOGY.—Actinomycosis usually shows itself as a slightly painful, slowly developed growth, which finally forms fistulous orifices through which are discharged purulent matters containing the granules already described. The usual seat of these growths is the region of the lower jaw. The affected bone enlarges, the soft tissues become hardened, and finally a large mass is formed which may destroy life by exhaustion, by hemorrhage from an ulcerated vessel, or by suffocation. In some cases the course of the disease is rapid and destructive, with the formation of large abscesses.

A very rare form of external actinomycosis is that in which the ray-fungus finds lodgement under the skin and gives rise to tumors by whose suppuration extremely obstinate ulcers are produced.

The actinomyces may also find lodgement on an internal mucous membrane and produce disease. In the lungs it may cause a chronic general bronchitis or a peculiar broncho-pneumonia, which is attended usually with fever, cough, abundant, often fetid, expectoration, loss of flesh and strength, and the physical signs of bronchitis or of broncho-pneumonia, and is followed after a time by various symptoms due to metastasis and dissemination of the parasites through distant organs. Actinomycosis of the lung may closely resemble chronic tuberculosis in its symptoms, but is much less apt to be attended with hæmoptysis. Sometimes it represents a rapid chronic pneumonia. By involving the pleural cavity the ray-fungus may produce a purulent empyema. Lodged in the intestines it causes indigestion, diarrhœa, local pains, and tenderness, and is especially liable to give rise to secondary disease of the liver, which is often attended with great enlargement.

Cases have also been reported in which the fungus has found lodgement in the brain, producing symptoms resembling those of tumor, or, as in a case reported by O. B. Keller, of cerebral abscess. It has been affirmed that the lesion in the brain may be primary; but the fungus must in some way have first entered the blood.

DIAGNOSIS.—The symptoms of an internal actinomycosis vary almost indefinitely with the seat of the disease. The only diagnostic mark of either the internal or the external disorder is the presence of the actinomyces in the discharges.

PROGNOSIS.—The prognosis in actinomycosis depends upon the seat of the disease. If the lodgement is in such place that it cannot be reached surgically, death is the almost certain result.

TREATMENT.—The treatment of actinomycosis is purely surgical. The tumor and the surrounding parts should be freely removed until pieces of tissue removed show no fungus, and then the wound should be freely cauterized. It is affirmed that silver nitrate is especially deadly to the fungus, and cures are stated to have been obtained by the free use of the solid stick, and also by strong injections of carbolic acid.

MYCETOMA.

Madura foot-disease, which is endemic in certain portions of India but occurs only in natives, especially in males who work in the fields or go about with bare feet, has been shown by Vandyke Carter, confirmed by Kanthack, to be simply a form of actinomycosis, the species probably, though not certainly, being different from that which is met with in Europe and America. The part affected is nearly always the foot, but the hand, arm, shoulder, scrotum, or any other portion of the body may be attacked. The first change is swelling and redness, followed after a time by a superficial or deep papule, which finally discharges pus containing white, yellow, black, or reddish, fish-roe-like granules and black, irregular masses. As the disease progresses there are severe pain, extraordinary swelling, and distention of the foot, giving way of the arch, destruction of the bones, and the formation of numerous elevations, which are the orifices of sinuses running into the centre of the foot. The treatment is the same as that of other forms of actinomycosis. If taken early, free local excision and curetting may suffice. Later, amputation, sufficiently high up to get above the penetrating fungus, is essential. The disease always ends in death from exhaustion if left to itself.

RABIES. HYDROPHOBIA.

DEFINITION.—An acute infectious disease, occurring in various animals, and produced in man by the bite of an animal suffering from the disease, characterized by a long incubation, paroxysmal convulsions, great excitement, fever, and paralysis terminating in death.

ETIOLOGY.—Rabies is especially common in canine animals, though it occurs in cats, is said to be very frequent in the American skunk, and may be transmitted to the herbivora. The symptoms in the dog are a primary rise of temperature, followed by shyness, chilliness, irritable suspiciousness, with a tendency to run from home, and a characteristically depraved appetite, which causes the animal to eat wood, stones, fæces, and even its own tail. There is at the same time altered voice, with a peculiar howl, and a little later furious maniacal excitement; there is often at this period or later paralysis of the muscles of the jaw, with excessive salivation, causing frothy saliva to drop from the mouth; the whole attack ending in progressive paralysis and death. If the paralysis develops early, the rabies is spoken of as *dumb rabies;* if the excitement is pronounced, the case is one of *furious rabies.* It appears to be definitely proved that the bite of the animal may produce rabies as early as forty-eight hours before the appearance of the prodromic fever.

There is no well-authenticated case of the communication of rabies from man to man. The nature of the poison is unknown, but according to the researches of Pasteur and others it passes up the nerve-trunks to the nerve-centres. The virus is in many cases wiped off the teeth of the biting dog by the clothing of the bitten person: hence bites upon uncovered parts of the body are especially fatal.

MORBID ANATOMY.—The alterations of especial significance in rabies are venous injection and thrombosis, cellular infiltration of the adventitia of the veins, first observed by Kolesnikoff, and miliary abscesses, originally described by Gowers and soon after confirmed by us. These changes have been found in the medulla, in our investigations, near the floor of the fourth ventricle. In addition, we have recorded the presence of myocardial hemorrhages and œsophageal œdema.

SYMPTOMATOLOGY.—The incubation period of rabies in the dog has been experimentally proved to be from six days to eight months. In man it is usually considered as between two weeks and three months, but it is not very rarely prolonged to a year, and even three years may elapse after the bite before the appearance of the symptoms. The disease in man may be divided into three stages. The first of these, the prodromic, lasts from a few hours to six or eight days, the symptoms being malaise, slight fever, hyperæsthesia of the special senses, and occasionally pains in the scar. The first pronounced symptoms of the stage of excitement are usually stiffness of the throat and difficulty in swallowing, which rapidly increase until all efforts at deglutition produce violent spasm of the muscles of the pharnyx, larynx, and upper chest. An intense hyperæsthesia, affecting special senses and general sensibility, now develops, with an increasing area of muscular involvement by the spasms. The paroxysms by and by are produced not only by attempts at swallowing, but by the sight of fluids, especially running water, and finally by bright lights, loud sounds, or, it may be, even by irritation of the surface of the body. Although there is intense thirst, efforts at drinking are accompanied with excessive terror, and if persisted in produce violent tetanic spasms, which in rare cases have ended in death by cramp asphyxia. Excessive salivation is common. There is usually mild fever. At first the intellect is clear, but as the disease progresses delirium develops and may become violent. After from a few hours to ten days the paralytic stage appears, with increasing loss of power, which commonly commences in the muscles of the jaw and spreads to the whole body until it ends in paralytic asphyxia.

DIAGNOSIS.—The diagnosis in rabies depends upon the peculiar respiratory spasm caused by attempts at swallowing, the intense hyperæsthesia, and the concluding paralysis. These symptoms may be closely simulated in hysteria, but in all such cases that we have seen the mockery has revealed itself in an apparent exaggeration but a real lack of intensity of the symptoms.

PROGNOSIS.—The prognosis of developed rabies is fatal. The percentage of cases following the bite of a rabid animal is estimated by Horsley at sixteen, by Hunter at five, but by various other authorities much higher, even up to eighty. The bites of rabid wolves are four or five times as dangerous as those of rabid dogs; and, according to popular belief, the bite of the rabid skunk is certain death.

TREATMENT.—The treatment of developed rabies must be purely symptomatic. The question as to the inoculative treatment of Pasteur, in which the person who has been bitten is immediately subjected to a series of inoculations with the properly prepared spinal cords of rabid animals, is a most serious one for the practitioner to decide. Out of seven hundred and ten persons who had been bitten on exposed parts of the head and inoculated at the Pasteur Institute in Paris, the mortality was 3.38 per cent.,—a result which seems to justify the advising of inoculation when there is a clear history of bite by a rabid animal upon an exposed part. In every case of a suspicious dog-bite it is the duty of the practitioner as soon as possible to excise the wounded parts and to cauterize thoroughly by hot iron, caustic potash, or other penetrating caustic,—a procedure which greatly lessens the danger of infection.

ANTHRAX. MALIGNANT PUSTULE.

DEFINITION.—An infectious disease, produced by the bacillus anthracis, occurring in two forms,—an external, characterized by a peculiar pustule having a black centre and surrounded by wide-spread, hard infiltration and secondary pustules; and an internal, characterized by a rapid toxæmia, usually accompanied by hemorrhages from the mucous membranes and numerous metastatic carbuncles.

ETIOLOGY.—The anthrax bacillus (*Bacillus anthracis*), described by Pollender in 1849, and especially studied by Davaine, Pasteur, and Koch, is a non-motile bacillus, from one to two and a half millimetres broad, producing spores, and growing in the open air. Desiccated spores may survive for years, and require boiling temperature to kill them with certainty. When set free in the soil by superficial burial of carcasses or otherwise, they multiply until a local infectious area is produced. Being a saprophyte, the anthrax bacillus is capable of passing through all its life-phases in the soil outside of the animal organism, so that its entrance into animals must be looked upon as accidental. The spores usually pass into the lower animals during the grazing months, and are liable also to be transported in times of freshet and give origin to new and perhaps distant infected districts.

Almost always man becomes infected through the lower animals: consequently malignant pustule is chiefly seen among those who work about domestic animals, or who are employed in the manufacture of raw products from animals, the tenacity of life of the spores being such that they may remain active in hair, wool, etc., for many years. Thus, malignant pustule occurs chiefly among butchers, stable-boys, shepherds, tanners, wool-sorters, glue-makers, upholsterers, etc. The infection may be through a wound or an abrasion, when external malignant pustule is produced; or by inhalation or swallowing of the spores, with a resulting internal anthrax. In rare cases the infection has been conveyed by insects.

Anthrax is much more frequent in Europe and Asia than in America, where it appears to be endemic only in certain districts originally infected by importation of the germ. Owing to the robustness of the bacillus and its spores, imported hides, hair, wool, and other raw animal products may cause the disease in man.

SYMPTOMATOLOGY.—The malignant pustule usually develops upon the hands, face, or other exposed portion of the body from a few hours to twelve or fourteen days after the infection. It is a small, red, intensely itching or burning point, which soon becomes a papule surmounted by a reddish or bluish vesicle, which, after bursting and discharging its bloody serum, is followed by a dark-brown or blackish crust. The induration extends far beyond the immediate neighborhood of the inoculated point, and often has radiating from it red streaks or lines, marking progressive lymphatic inflammations. Secondary vesicles surround the central one, and gangrene may result. Nausea, vomiting, diarrhœa, fever, delirium, excessive sweating, and collapse, appear usually on the second day, and death may occur in from five to eight days; or by abatement of the local inflammation and the subsidence of the constitutional symptoms the subject may return to health, convalescence being preceded by sloughing off of the vesicle.

Under the name of malignant *anthrax œdema* a condition is described by Bollinger and others in which the pustule is wanting, and the only local symptom is a wide-spread, brawny œdema, which is usually followed by another form of gangrene, great constitutional disturbance, and death.

A *malignant œdema* has also been recorded in two cases of typhoid fever following a hypodermic injection of musk, and caused by a long, spore-forming bacillus resembling the anthrax bacillus, but narrower and with rounded ends.

In internal anthrax, or *wool-sorters' disease*, the invasion is usually sudden, with headache, rigor, nausea, vomiting, abdominal pains, and diarrhœa. Free hemorrhage may occur from the mouth, nose, and kidneys, and death, preceded by delirium, convulsions, dyspnœa, cyanosis, and heart-failure, result. In another form of internal anthrax, in which the infection is said to be in the lungs, the symptoms take on the appearance of a rapidly spreading pneumonia, with great adynamia. The pulmonic cases are especially seen among wool-sorters, carpet- and blanket-makers, weavers, and other workers in hair-like animal products; whilst the intestinal variety is produced by the eating of diseased meats.

DIAGNOSIS.—Anthrax is to be distinguished by the fact that whilst in carbuncle the local sore is formed by the coalescence of numerous points, in anthrax the origin is purely centric. Anthrax œdema is distinguished from erysipelatous œdema by its having a grayish or yellowish instead of a reddish tint, and by the induration being more distinct and less superficial. The diagnosis of internal anthrax is one of great difficulty, unless there is an outbreak of external carbuncles. Whenever the symptoms of a sudden infection appear in a person whose business exposes him to anthrax infection, the blood should be examined for the bacillus, and inoculation of a guinea-pig or a mouse with the blood, or with the local products, if there are any, should be practised. The symptoms of anthrax in the guinea-pig or the mouse are rapidly developing constitutional symptoms of a general infection, with dyspnœa and convulsions, whilst the blood swarms with the organism.

PROGNOSIS.—Under proper treatment, applied early, malignant pustule is almost invariably recovered from. Under neglect the mortality may be seventy-five per cent. The prognosis depends, therefore, on the earliness and thoroughness of the treatment. Intestinal and thoracic anthrax is in the great majority of cases fatal.

TREATMENT.—The treatment of external malignant pustule is immediate, thorough destruction by incision, followed by cauterization with the hot iron or by corrosive sublimate or carbolic acid. In the œdematous form local injection of the three per cent. solution of carbolic acid should be practised, or the parts should be abundantly scarified and dressed with carbolic acid solution. In internal anthrax there is no known specific treatment: intestinal antiseptics may be tried. In all cases of anthrax the general treatment should be supporting, stimulating, and symptomatic.

FOOT-AND-MOUTH DISEASE.

This is a highly contagious disease of domestic animals, usually occurring in epidemics, characterized by vesicles, pustules, and ulcers in the mouth, at the top and cleft of the hoof, and on the udder. The virus is in the discharges from the sores, and also in the general excretions, and is transmissible in fomites. It is probably an organism whose characters have not yet been determined. In man the virus produces disease by direct inoculation, and also through the drinking of milk. After an incubation period of from three to five days there is a rigor, followed by fever, headache, anorexia, and malaise, and after some days vesicles appear on the lips, tongue, and pharynx, and leave ulcerations behind them. Vesicles sometimes develop on the hands and feet, especially near the nails. As diarrhœa is a common symptom, the mucous membrane of the intestines is probably affected. In severe cases there may be hemorrhages from the mucous membrane. Recovery usually occurs after about two weeks of illness. In a recent epidemic the mortality was eight per cent. (Saenger).

The stomatitis should be treated locally with boric acid solution after disinfection with hydrogen dioxide solution, etc. Ulcerative points may be touched with solid silver nitrate. The internal treatment should be symptomatic, stimulating, and supporting.

As a measure of prophylaxis, during an epidemic of the disease among cattle all milk should be thoroughly boiled before being used.

GLANDERS. FARCY.

DEFINITION.—A contagious disease, produced by the bacillus mallei, especially attacking horses, but capable of transmission to man.

ETIOLOGY.—Glanders may occur in almost any animal, but chiefly attacks horses, in which it produces inflammation of the mucous membrane of the nose and upper respiratory passages, with cough, fever, purulent coryza, and ulceration of the nasal mucous membrane, attended by great enlargement of the submaxillary glands and the formation in various parts of the body of superficial glandular enlargements, which when hard are known as "farcy buds," and when ulcerated as "farcy sores," the whole ending in death from exhaustion and septicæmia. The virus which exists in the nodules and in the discharges from the ulcers and mucous membrane was discovered by Loeffler and Schütz to be a bacillus resembling, but shorter and thicker than, the bacillus of tuberculosis. The infection of man is usually by inoculation, through an abrasion on the hands or elsewhere, with the nasal discharges from the horse, but may occur through breathing or swallowing the poison. In a number of cases lions and other animals in menageries have been infected by eating the meat of diseased horses.

Glanders is capable of being transmitted from man to man, and infection of washerwomen has occurred, but the disease is chiefly seen in hostlers, coachmen, and others who work about horses.

SYMPTOMATOLOGY.—After an incubation period usually of three or four days, but sometimes protracted to fourteen days, acute glanders develops, with malaise, headache, anorexia, articular pains, and usually but not always fever. At the same time swelling, redness, and inflammation of the lymphatics come on at the point of inoculation. Within two or three days coryza appears, and rapidly becomes severe and purulent, and at or about the same time red spots come out on the face and about the joints, or sometimes over the whole surface of the body, rapidly developing into hard papules closely resembling those of variola, and going on to vesiculation, pustulation, and final ulceration. As the disease progresses, deposits occur in the Schneiderian membrane, and often also in other portions of the body, forming nodular subcutaneous enlargements, "farcy buds." These rapidly suppurate and ulcerate; the coryza becomes more and more severe, with much swelling of the nose; general symptoms of septicæmia develop; not rarely pneumonia comes on; and almost without exception death occurs in from six to twelve days.

The distinction which is made by writers between acute farcy and acute glanders has no sufficient foundation, the only difference being that in the one case the subcutaneous enlargements are pronounced, in the other they are not so strongly developed.

Chronic glanders in man ordinarily resembles in its course a severe coryza, which is distinguished from other forms by a tendency to recurring ulceration and the formation of contracting and deforming cicatrices. In one form of chronic glanders (chronic farcy of writers) there are numerous subdermal nodules with resulting abscesses and ulcerations, without much inflammation or lymphangitis. This variety is probably due to direct inoculation through an abrasion.

DIAGNOSIS.—The only disease that acute glanders resembles is smallpox; but the occupation of the patient should put the practitioner on his guard, and the nasal symptoms make differentiation easy. The recognition of chronic glanders may be extremely difficult; in any suspected case the bacillus should be looked for, and inoculation of the guinea-pig with the discharges or with cultures from the discharges should be practised: if the bacillus mallei be present, death of the animal will occur within thirty hours, with in the male enormously swollen suppurating testicles. In acute farcy the nose may not be inflamed : here, in any case of doubt, inoculation should be practised.

PROGNOSIS.—Acute glanders always ends in death. Chronic glanders may last from three to five months, and is said to have a mortality-rate of about fifty per cent.

TREATMENT.—There are no known remedies which have any control over glanders. If the case be seen early, the points of inoculation should be thoroughly destroyed by excision and caustics. Glandular enlargements should be opened early. The further treatment should be conducted on general principles, and should always be supporting and mildly stimulating.

GREASE.

Grease in horses is a peculiar ulceration between the quarters of the heel, with a thick, offensive discharge. By contact with this discharge hostlers and others suffer from a superficial pustular eruption. This yields readily to treatment, which should consist of thorough washing and disinfection and touching the points with weak astringent solution (copper sulphate four grains to the ounce, silver nitrate two grains to the ounce), or rarely with caustic.

TUBERCULOSIS.

DEFINITION.—An infectious disease due to the invasion of the organs and tissues by the bacillus tuberculosis. Interstitial and superficial inflammations follow, either circumscribed or diffuse, and the anatomical products tend to become cheesy or indurated, the cheesy dead material undergoing softening or calcification. The resulting symptoms depend partly upon the local disturbances caused by the bacilli, partly upon the absorption of the toxic products of their growth, and partly upon the modifications in the function of the organ especially diseased.

ETIOLOGY.—The bacillus discovered by Koch in 1882 is the immediate cause of tuberculosis in man or in animals, although the infectious nature of this disease was ascertained by Klencke in 1843, whose observation was forgotten. It was suspected from the anatomical appearances by Buhl in 1857, and rediscovered by Villemin in 1865. Tuberculous man is the chief source of human tuberculosis, yet the disease may be derived from the tuberculosis of the domesticated animals. It is rare in dogs and cats, in sheep and swine, and prevails among the bovines, but the so-called tuberculosis of fowls is not due to the bacillus of tuberculosis. Of 4093 cattle in Massachusetts tested for tuberculosis by means of tuberculin. 1081 reacted positively, and were killed, and anatomical evidence of tuberculosis was found in all but two. Of 132,294 cattle slaughtered in Copenhagen, seventeen and seven-tenths per cent., and of 142,872 killed in Berlin, fifteen and one-tenth per cent., were tuberculous. In this connection it is interesting to note that the average mortality from tuberculosis in man is in the vicinity of fifteen per cent. The bacilli transferred from man to man are generally considered to be largely derived from cases of pulmonary tuberculosis, while those transmitted from cattle are probably conveyed by contaminated milk, dairy products, and more rarely by meat. The degree of risk of the transfer of tuberculosis from animals to man is lessened in the preparation of food. The milk from tuberculous animals, even if the udders are not tuberculous, may be dangerous, and should be avoided, since Ernst has shown that the milk may contain the bacilli although the udders are free from tubercles. Milk from cows with tuberculous udders is dangerous, and must be condemned, and Bang has shown also that butter made from the milk of tuberculous cows is infectious.

The bacilli of tuberculosis enter the body through the skin or by means of the respiratory and digestive tracts, and perhaps by the uro-genital canal. The skin is invaded when bacilli lodge in a wound, as a scratch or tear from the sharp edge of a broken receptacle containing tuberculous sputa. The wound occurring in the ritual performance of circumcision may become infected with bacilli present in the mouth of a tuberculous operator. The surgeon may be infected with bacilli during an operation upon tuberculous bones, joints, or glands, and the anatomist may become inoculated with the living bacilli in a dead body. The inhalation of dried tuberculous sputa, however, is the chief source of tuberculosis of the respiratory tract. The swallowing of tuberculous sputa and of food containing virulent bacilli is the principal source of infection by means of the alimentary canal. The food most often contaminated is milk from a tuberculous cow, especially one whose udders are tuberculous. It is also true that the nursing mother may give infected milk from a tuberculous breast. The entrance of bacilli by means of the uro-genital tract, though rare, is possible in the case of genital tuberculosis of husband or wife. The above-mentioned sources of invasion of the body are usually included under the term acquired tuberculosis. Congenital tuberculosis, on the other hand, sometimes exists, perhaps more often than is generally supposed, and is to be explained by the transmission of bacilli from the mother to the fœtus through the vascular walls in the placenta, which organ has been found to contain evidences of tuberculosis.

The bacillus once lodged in the body and finding favoring opportunities for its growth multiplies, and is transferred from the place of entrance along mucous surfaces or through lymphatics and blood-vessels, and thus gives rise to anatomical changes remote from those which may have been produced at the point of entrance. A distinction is hence drawn between primary and secondary tuberculosis. The bacilli are present throughout the world, since tuberculosis exists everywhere and prevails wherever large collections of individuals exist. The disease is less frequent in high altitudes. and, as was first announced by H. I. Bowditch and by Buchanan, is of increased frequency in low-lying, damp regions. It is more frequent in cities and towns than in the country. It abounds in prisons and asylums, and Cornet has frequently found the bacilli in the dust of hospital wards. It is, however, maintained that the nurses and attendants in hospitals for tuberculous cases are not especially likely to become diseased, although Strauss has found the bacilli in the nostrils of physicians and nurses caring for tuberculous patients.

The bacillus of tuberculosis is a non-motile, narrow rod, usually somewhat curved, from one-half to two-thirds the diameter of a red blood-corpuscle. It is found alone or in groups, and its presence has been demonstrated in the sputum, urine, fæces, blood, pus, lymph, and tissues of tuberculous patients. It grows slowly at the temperature of the body, and when cultivated attains the maximum of its development in four weeks. Tuberculin is a toxic product of its cultivation, and is of value as a means of testing the presence of tuberculosis in animals. Its therapeutic use in man has been productive of harm, and a positive reaction may follow its injection into healthy persons. On the other hand, tuberculous patients do not always react when it is tried. With possible danger and uncertainty of results, its employment in the diagnosis of human tuberculosis is to be regarded as generally inexpedient. Koch endeavored to free tuberculin from its injurious properties, and Klebs announces that he has extracted a harmless and beneficial tuberculocidin from tuberculin. Such an extract is now offered, under the name of antiphthisin, as a remedy in the treatment of phthisis. Especial clinical importance is to be attached to the demonstration of the bacilli in sputum and in urine, since the absolute diagnosis of the presence of tuberculosis may thus be established. Even in those instances in which the bacilli cannot be shown in material of supposed tuberculous origin their presence may often be proved by the production of tuberculosis in inoculated animals.

Predisposing causes of tuberculosis are also important, since of all exposed to the invasion of the bacilli some or many do not become diseased. The predisposition may be inherited, since it is universally recognized that the children of tuberculous parents are in especial danger of becoming tuberculous. Such children are commonly called scrofulous or strumous. This inherited predisposition is to be explained rather by increased vulnerability of the tissues and a diminished power of resistance than by the actual transmission of the bacilli from the parent to the offspring. A congenital stenosis of the pulmonary artery or malformation of the heart may act as a predisposing cause, since these apparently promote the progress of pulmonary tuberculosis by unfavorably modifying the pulmonary circulation. The young are more prone to become tuberculous than the aged, although a localized pericardial tuberculosis is sometimes found in extreme old age. Acquired local predisposing causes are also important by enfeebling the power of resistance of the individual. Among these are faulty hygienic surroundings, insufficient food, insanitary occupations, especially those favoring the inhalation of irritating dust, acute or chronic inflammation, particularly of the respiratory tract, and acute infectious diseases, chiefly those producing disturbances of the respiratory organs, as measles, whooping-cough, and influenza. Traumatism favors the growth of the bacilli by diminishing the power of resistance of the tissues. Chronic debilitating diseases, as syphilis and diabetes, are also of importance in etiology.

MORBID ANATOMY.—The anatomical changes due to the presence of the bacillus of tuberculosis are essentially of an inflammatory character. An exudation is produced, the quality and quantity of which depend upon the number of bacilli present, the structure of the organ, and their localized or disseminated presence upon the surface or within the tissues of the diseased part. The word tubercle, whence the term tuberculosis is derived, is descriptive, and is applied to a little knot, node, or granule, the result of the irritation due to the presence of a circumscribed collection of the bacilli of tuberculosis within the tissue. Such a tubercle was called miliary from its resemblance in size to that of a millet-seed, although smaller tubercles, submiliary, may be present, and others even smaller can be found with the microscope. The typical structure of a tubercle consists of one or more polynucleated giant cells in which the bacilli may be found. These are surrounded by large endothelioid or epithelioid cells in which also the bacilli may be present, and these in turn are surrounded by small mononuclear corpuscles resembling those prevailing in lymphatic glands. These various corpuscles lie within the meshes of a net-work, the reticulum, which is elaborated from the fibrous tissue in which the bacilli have become lodged. The submiliary and miliary tubercles are composed of agglomerations of the minute and microscopical tubercles, which may congregate into masses as large even as the fist, and are then designated nodular and conglomerate tubercles.

Variations in the structure and shape of the miliary tubercle may arise. The younger the tubercle the more numerous the cells, the less the fibrous reticulum. In the pia mater of the brain the tubercles are composed almost entirely of small round cells, which are accumulated in the adventitia of the arteries entering the cerebral cortex and in which they appear as fusiform swellings. In the lungs the miliary tubercles may represent irregular thickenings of the interstitial tissue at the junction of the bronchioles with the infundibulum or of the alveolar walls projecting into it. Diffused and extensive growth of fibrous tissue may result from the presence of the bacillus. The chief characteristics of such growth are the abundant production of endothelioid cells, and the presence of the bacilli.

When the bacillus multiplies upon a surface it produces an exudation which is largely cellular, but may be fibrinous or hemorrhagic. The cells are both those desquamated from the surface, alveolar epithelium, for instance, and migrated leukocytes resembling either pus-corpuscles or lymph-corpuscles. An important characteristic of the cellular inflammatory products due to the invasion of the organs and tissues by the bacillus is their tendency to early death. A necrosis occurs, usually beginning at the centre of the tubercle or in those cells of a superficial exudation farthest removed from their source. It is manifested by a homogeneous glistening appearance of the cell, which is readily stained with fuchsine, although the nuclei lose the power of becoming differentially stained. An absorption of fluid takes place from the dead material, which thus becomes transformed into a homogeneous opaque yellow mass in which the structural details are lost. To this condition the term *caseation*, *cheesy degeneration*, or *cheesy metamorphosis* is applied. The minute tubercle or the agglomerated myriads of tubercles may undergo caseation, and the inflammatory products on the surface, both cells and fibrin, may become similarly metamorphosed. In those organs in which an association of superficial and interstitial inflammation is possible the cheesy masses often consist of the interstitial tubercle and the superficial inflammatory exudation. The group of changes is designated tubercle or tuberculosis, the anatomical characteristic the tubercle giving way to the etiological unit the bacillus. Tuberculosis thus no longer means a tubercle, but the various lesions, superficial or interstitial, due to the invasion of the bacillus of tuberculosis.

The cheesy material tends to become softened or hardened. Softening apparently occurs from a soaking of the dead material with fluid, a molecular disintegration being the result. The softening occurs more rapidly in those parts to which the air is freely accessible, as the lungs, and it seems probable that it may be accelerated by the presence of other bacteria than those of tuberculosis, and also by the entrance of amœboid corpuscles into the cheesy masses. The softened cheesy material may be carried along the passages communicating with the surface of the body, and ulcers or cavities result, or be absorbed by means of the lymphatics and blood-vessels, or be evacuated through the skin either spontaneously or by a surgical operation. In the displacement of softened cheesy material from one organ to another a renewal of infection is favored, since the cheesy detritus contains the bacilli.

Cheesy material becomes hardened by the deposition within it of lime salts, calcification. The cretaceous mass remains embedded in fibrous tissue often throughout the life of the individual, and is usually productive of no further disturbance.

A fibrous transformation of the tubercle may occur, when many of the cells are destroyed, others are transformed into permanent connective-tissue cells, and the fibrous reticulum becomes thickened and broadened. The fibrous transformation of the tubercle results in the production of a sort of scar-tissue, which may retain the shape of the original tubercle or appear as irregularly defined collections of dense fibrous tissue, whose origin from the bacillus of tuberculosis may be a matter of pure inference.

The *symptoms* depend upon the manner and method of the invasion and upon the existence of other disturbing agencies. There are certain common features, however, which, although varying in severity and rapidity of development, are present whatever part of the body may be invaded. Such manifestations are conspicuously fever and wasting, and in former times gave rise to the designations hectic or hectic fever and consumption, phthisis or tabes, which were further qualified according to the structures affected as pulmonary, renal, intestinal, or mesenteric, and according to the rapidity of progress as acute, galloping, or chronic.

With the recognition of tuberculosis as an infectious disease, stress has been laid upon the conspicuous affection of one or more organs or sets of organs, since the local symptoms resulting from the disturbance of function of these organs are the more especial causes of complaint. Although the local manifestations of tuberculosis are usually described in connection with the organs diseased, repetition is avoided and the importance of the process as a whole is more readily appreciated by the consideration of the various manifestations of the presence in the body of the bacillus under the one title tuberculosis, with subdivisions classifying the important clinical characteristics of the disease.

VARIETIES.—The most important clinical distinction is that drawn between general and local tuberculosis. General tuberculosis affects various organs simultaneously or in rapid sequence, and the bacilli are distributed chiefly by means of the circulation. Local tuberculosis occurs in an organ or an apparatus, and the bacilli are admitted to the region concerned largely from the surface exposed to the air, although in part through the circulation. A further practical distinction is made between acute and chronic tuberculosis. In the former the tubercles are usually miliary or submiliary in size, of a gray color, and are generally associated with but little evidence of a superficial inflammatory exudation. In chronic tuberculosis extensive agglomerations of tubercles in nodules and patches occur. The tubercular masses are opaque yellow from cheesy degeneration. Softening, calcification, and fibrous induration are of frequent occurrence, and acute and chronic inflammatory changes are often associated. General tuberculosis is usually acute, and is often called disseminated from the scattered distribution of the tubercles; local tuberculosis is commonly chronic, although it may become complicated by an acute outbreak of disseminated miliary tubercles.

GENERAL ACUTE OR DISSEMINATED TUBERCULOSIS.

This designation is given to the presence of tubercles in various parts of the body due to the more or less rapid dissemination of considerable numbers of the bacilli.

ETIOLOGY.—Acute general tuberculosis is a secondary process depending upon the existence somewhere in the body of a tubercular lesion, either apparent or concealed, from which the bacilli are admitted into the circulation. The entrance of the bacilli usually takes place by the extension of a local tubercular process in an organ commonly the seat of chronic tuberculosis, as the lungs or the lymph-glands, into a blood-vessel, especially a vein, or into a lymphatic, notably the thoracic duct. This variety is more common in children than in adults, and not infrequently immediately follows an acute affection of the respiratory tract, as influenza, measles, or whooping-cough. These diseases apparently exercise a favoring influence in promoting the admission of bacilli into the blood, perhaps by diminishing the resistance of the tissues to the rapid growth of the bacilli towards the nearest vessels. In like manner typhoid fever has occasionally been reported as preceding the symptoms of a general tuberculosis.

MORBID ANATOMY.—The lesions present are miliary and submiliary granules, either projecting from the surface of an organ and easily recognized or lying within its substance, as in the lungs, spleen, or kidney, and readily apparent or to be appreciated only by close scrutiny, even requiring the use of the microscope for their identification. Few or many tubercles are present, and, except in the cerebral pia mater, lungs, and kidneys, are usually unaccompanied by other inflammatory exudation. The pia mater, lungs, liver, and spleen are the organs in which the largest number of tubercles is to be found, although they are also present in the kidneys, choroid, heart, thyroid gland, serous membranes, and bone-marrow. The predominant localization of the tubercles, as far as the symptoms are concerned, is in the pia mater or the lungs.

SYMPTOMATOLOGY.—The general symptoms of acute disseminated tuberculosis closely resemble those of typhoid fever, and are often of rapid onset in a person apparently in previous good health, or they may be preceded by a gradually increasing sense of malaise. Not infrequently signs or symptoms exist of chronic or latent tuberculosis somewhere in the body. Chilly sensations often followed by flushing are complained of. The patient loses appetite and becomes weak. The symptom which usually first calls attention to the nature of the disease is the elevation of temperature, which frequently has evening exacerbations and morning remissions perhaps with considerable differences, as in the third week of typhoid fever. An evening temperature of 103.5° or 104° F. is common. Higher degrees are sometimes observed, especially in children, shortly before death. The range is often exceedingly irregular, perhaps with intervals of a few days of relatively normal temperature to be followed by almost periodical remissions or intermissions. The morning record may be higher than that in the evening, although this is only an occasional feature. Very exceptionally there may be little or no elevation of temperature, and still more rarely it is found subnormal. The elevated temperature, accelerated respiration, and rapid and weak pulse in the absence of localizing symptoms are directly suggestive of typhoid fever. The associated debility, hebetude, sopor or mild delirium, dry tongue, and occasional diarrhœa offer additional suggestive evidence of the same process. A rash also somewhat resembling that in typhoid fever is sometimes observed.

The area of splenic dulness is increased, although the enlargement of the spleen is usually moderate. The bacilli of tuberculosis have been found in the blood, but the search for them is so often unsuccessful as to be of little or no avail in diagnosis. A moderate leukocytosis may exist. The urine is scanty, high-colored, and of high specific gravity, corresponding to the elevated temperature. A trace of albumin is frequent, and the diazo-reaction is often present. The characteristic bacilli have been found in the urine, but so inconstantly and under such difficulties that they are usually not sought for diagnostic purposes.

Acute Tubercular Meningitis.—In the further progress of acute general tuberculosis the cerebral symptoms may predominate over the pulmonary, or the reverse may occur. The cerebral symptoms are often so pronounced at the outset that the disease is to be regarded rather as one of meningitis than of acute tuberculosis. Although tubercular meningitis, as a rule, is the conspicuous localization of a general tuberculosis, the occasional occurrence of tubercular meningitis independent of tuberculosis elsewhere in the body may be mentioned. Tubercular meningitis is especially frequent in children, in whom it is usually secondary to tuberculosis of the lymph-glands, tubercular caries of the mastoid, or tuberculosis of the brain. The tubercles are most numerous at the base of the brain, especially near the optic chiasm and the pons Varolii, in the fissures of Sylvius, and upon the upper surface of the cerebellum. They appear particularly in the vicinity of injected blood-vessels as granules, nodules, and patches, and are to be found as fusiform thickenings of the adventitia of the minute arteries entering the cerebral cortex. They are also to be found, especially in children, in the choroid plexuses of the lateral ventricles, whose cavities are dilated with an opaque fluid and the ependyma thickened and softened. To this condition the term *acute hydrocephalus*, or dropsy of the brain, was formerly applied. The more abundant the distention of the ventricles with fluid, the drier the brain, the more flattened the convolutions, and the more obliterated the furrows. In the meshes of the pia mater is a more or less extensive infiltration of an opaque yellow fibrino-serous material, especially abundant in those parts in which the tubercles are numerous, although it may be abundant and but few tubercles be observed. Minute hemorrhages may be found in the pia mater and in the brain. Tubercles of the choroid are more likely to be present when acute tuberculosis is especially marked in the cerebral membranes. Similar alterations may be found in the membranes of the spinal cord, especially at the upper and anterior portion.

The symptoms of tubercular meningitis may develop suddenly and progress so rapidly, especially among children, that death may quickly occur without warning. Usually, however, they are preceded during a week or two by the general symptoms of acute tuberculosis above mentioned. Headache is conspicuous, is sometimes intense, and is usually aggravated by light and noise. Nausea, vomiting, unexpected or not, and constipation are more or less constant. Delirium and restlessness are frequent, and in children grinding of the teeth and convulsions are common. Among them a sudden outcry, sometimes a shriek or scream, the *hydrocephalic cry*, is often heard. The head is drawn back, the anterior wall of the abdomen becomes concave, and spasmodic contractions of various muscles occur. The skin readily shows the *taches cérébrales.* The patient rapidly loses flesh and strength. In the further progress of the disease the irritative symptoms diminish, and those of intracranial pressure, the paralytic symptoms, appear. The patient is more quiet, but shows increasing dulness of mind. The stupor becomes more profound, and Cheyne-Stokes breathing may precede the death of the comatose patient. Strabismus and conjugate deviation of the eyes or drooping of the lids occur, and the pupils previously contracted become dilated or irregular. Localized pareses or paralyses may take place, and Osler notes the existence of aphasia and brachial monoplegia. The pulse is frequently irregular. Tubercles may be found in the choroid, and the appearances of optic neuritis may be observed with the ophthalmoscope. In acute general tuberculosis in which the cerebral membranes are especially diseased death may occur, as already mentioned, with such suddenness as to suggest cerebral hemorrhage. More often the symptoms of a meningitis persist during a period of two or three weeks, and sometimes, especially when the tubercular meningitis is sharply localized, the symptoms are mild and are continued over a period of months.

Acute Tuberculosis of the Lungs.—With a predominant localization of the tubercles in the lungs the condition becomes one of acute miliary tuberculosis of the lungs. These organs are distended, injected, somewhat increased in weight. The air does not readily escape even on pressure, and the tubercles are often to be felt as minute shot-like bodies. The pleuræ frequently show numerous minute and agglomerated tubercles. On section of the lungs the miliary and submiliary tubercles, either gray and translucent or yellow and opaque, are disseminated in greater or less abundance throughout, often projecting above the surface. They are not infrequently associated, especially at the apices, with large cheesy, sometimes softened nodules, due to an earlier localized tubercular process. In the rapidly progressing cases the interstitial miliary tubercles are unaccompanied by exudation in the alveoli. In the protracted cases more or less cellular or fibrino-cellular, perhaps cheesy, exudation is present in various quantity within few or many alveoli.

The onset of acute miliary tuberculosis of the lungs may take place so rapidly as to suggest an acute diffuse bronchitis or fibrinous pneumonia. More often with the early prodromal symptoms of general tuberculosis the conspicuous affection of the lung becomes manifested by cough, rapid breathing, and pleuritic pain. The cough is frequently persistent, sometimes incessant, the sputum usually scanty, at first consisting of mucus, but soon becoming muco-purulent, and is sometimes streaked with blood. Characteristic bacilli, if present, are to be accounted for rather by the softening of an antecedent tubercular focus than by the acute invasion of the interstitial tissue. The rapid breathing, perhaps sixty inspirations per minute, is due in part to the presence of a diffused bronchiolitis and to the projection of the tubercles into the alveoli and bronchioles, and in part to the tuberculosis of the nervous system. As a result of the dyspnœa, the patient becomes cyanotic out of all proportion to the physical signs of affection of the lungs or heart. The pleuritic pain is of frequent occurrence, resulting from the presence of tubercles in the pleura, and is accompanied by the sound of friction. On physical examination of the chest the resonance is either normal or increased, the tympanitic resonance corresponding to the frequent hyperdistention of the lung found after death. The existence of spots of dulness is suggestive of chronic foci of tuberculosis or of acute broncho-pneumonic complications. On auscultation fine moist and dry râles are distributed throughout the lungs, while towards the end of the disease coarse dry and moist râles are numerous. Absence of respiratory sounds or tubular breathing and bronchophony may be evident in the regions of localized dulness.

In acute general tuberculosis in which the pulmonary symptoms are conspicuous, death may occur in the course of a fortnight after the appearance of these symptoms, or the progress of the disease is subacute or chronic, then extending over a period of weeks or months. The course essentially depends upon the sudden or gradual entrance into the blood-vessels of few or many bacilli during a longer or shorter period. Although this variety is called acute, and generally is so, its progress may extend over a period of months as well as of weeks.

In *chronic general tuberculosis* as distinguished from acute general tuberculosis the symptoms, although similar, are less severe, and periods of intermission and exacerbation of various duration occur. Such variations are especially indicated by the changes in temperature, periods of normal temperature alternating with atypical elevations or periodical intermissions, perhaps accompanied with chills, and are for the time being suggestive of malarial attacks. Progressive loss of flesh and strength and increasing pallor are more conspicuous than severe cerebral or pulmonary symptoms.

Diagnosis.—The diagnosis essentially depends upon the persistence of atypical fever without obvious local cause, during the progress of which meningitic symptoms occur or dyspnœa and cyanosis arise, the latter without sufficiently explanatory physical signs on examination of the lungs. It is strengthened by evidence of an antecedent tubercular affection in some part of the body, and is definitely established by the discovery of tubercles in the choroid or by the presence of the characteristic bacilli in the sputum.

During the early or typhoid stage indicative of the general infection, the range of temperature is the chief means of distinguishing acute miliary tuberculosis from typhoid fever. It is atypical and irregular in the former, more definitely characteristic in the latter. The presence or absence of rose spots, constipation or diarrhœa, meteorism and right iliac pain, enlargement of the spleen, and the diazo-reaction, are not significant of either affection. The presence of leukocytosis is in favor rather of acute tuberculosis than of typhoid fever. With the predominant development of meningitic symptoms the tubercular nature of the meningitis is to be inferred from the absence of traumatism, of inflammation of the middle ear, and of epidemics, the usual causes of meningitis. The onset of tubercular meningitis is more gradual, the irritative stage is more prolonged, the convulsions are more frequent, and evidence of an extensive bronchitis is more often present in meningitis of tubercular origin than in the other varieties of this disease.

If the acute tuberculosis progresses with conspicuous localization in the lungs the local physical signs are those of a capillary bronchitis. The latter affection, however, is of more sudden onset, with immediate development of the signs of a diffuse bronchitis. The cough is more severe, while dyspnœa and cyanosis are less extreme.

PROGNOSIS.—Acute general tuberculosis is a universally fatal disease. The rare cases of reported recovery are based upon the disappearance of symptoms. Post-mortem examinations have never shown any anatomical evidence by means of which the assumption of recovery from an antecedent attack of acute general tuberculosis could be maintained.

LOCAL TUBERCULOSIS.

This term is applied to indicate the conspicuous presence of tubercles or other inflammatory products due to the presence of the bacillus of tuberculosis in limited portions of the body, and represents the most frequent cause of death in mankind. These products are usually combined both within the tissues and upon the surfaces. Instances of the former are not only the miliary and submiliary tubercles, disseminated and in clusters, but also endothelioid or epithelioid cells from blood-vessels and lymphatics, perhaps from other tubes also,—bile-ducts, for example. The superficial products are the hyperplastic epithelial cells from glands, tubules, ducts, and other surfaces lined or covered with epithelium, while blood-corpuscles, fibrin, and serum may be found as a part of the exudation both within the tissues and upon the surfaces. Local tuberculosis may be conveniently considered under the head of tuberculosis of the skin, of the mucous membranes. digestive organs, and uro-genital apparatus, of the serous membranes, of the ductless glands, of the nervous and vascular systems, and of the bones and joints, although two or more of these regions may be simultaneously affected.

Tuberculosis of the Skin.—The skin becomes tuberculous by the direct inoculation of the bacilli from without, or may arise from the extension towards the surface of a subcutaneous tubercular affection, as may be seen near the outlet of fistulæ communicating with softened cheesy lymphatic glands and tuberculous bones and joints. The miliary tubercles are embedded in greater or less number in a congested fibrous tissue containing abundant endothelioid cells.

According to the circumstances of the infection, the method of the extension of the local process, and the nature of the secondary changes, various terms are applied. The *anatomist's tubercle* is represented by a sharply defined, reddish, translucent nodule, generally limited to the superficial portion of the skin, pertinacious, but not prone to undergo extensive secondary changes. In *lupus* numerous nodules of a reddish-brown tint are present. They increase in size, project considerably above the surface, and are associated with the abundant formation of epidermis or with extensive necrosis. The necrotic tissue becomes softened and separated, while a renewal of the infection takes place both laterally and below the surface. Extensive scars, with extreme deformity, notably in lupus of the face, result from the tendency of the cicatricial tissue to contract. The term *scrofuloderma* is applied when the miliary tubercles and diffused tubercular tissue form slightly projecting nodules of a purplish color, which undergo necrosis and softening and result in the formation of ulcers with a cheesy base. These ulcers usually occur in the vicinity of a deeper-seated tubercular affection, especially of the bones or lymph-glands, and are believed to result from the transmission by means of the lymphatics of the bacilli from such sources to the skin. *Tuberculous ulcers* are also recognized independent of scrofuloderma. They are characterized by an irregularly rounded and congested margin and an indurated base, in which are opaque yellow specks. Miliary tubercles are present both in the base and edges. Further consideration of cutaneous tuberculosis is to be found in works on dermatology and surgery.

Tuberculosis of the Mucous Membranes.—Tuberculosis of the mucous membranes forms the most important group of the tubercular affections, and the resulting disturbances are associated with so much wasting that they in particular are those which have been usually described under the head of phthisis, consumption, or tabes. The processes are essentially the same whether the bacilli are inhaled, swallowed, admitted in copulation, or eliminated through the kidneys. The usual result is that more or less of the tract in continuous relation with that part of the mucous canal first diseased is simultaneously affected : hence in tuberculosis of the respiratory mucous membrane the lungs are usually diseased, and in tuberculosis of the urinary tract the genitals, especially in man, are frequently similarly altered.

In tuberculosis of the mucous membranes miliary tubercles result from the invasion of the bacillus. They are seated superficially, and tend rapidly to become cheesy. The more abundant and the more liquid the outward flow over the mucous membrane, and the more superficial the tubercles, the more quickly is the cheesy material removed: hence the so-called lenticular ulcers arise, sharply defined and shallow, but tending to spread laterally by the confluence of neighboring ulcers. The base of the confluent ulcers may be of a grayish-yellow color, from cheesy material not washed away. Such ulcers are especially to be found upon the posterior surface of the epiglottis, in the larynx and larger bronchi, and in the ureters and bladder. The smaller the canal the less readily removed are the cheesy products, which then become inspissated and often form an obliterating plug, as in tuberculosis of the smaller bronchi, the Fallopian tubes, and the vasa deferentia. In the intestine large and indurated ulcers result from the extensive formation of tubercles in the base as well as in the edges of the ulcer. The clinical importance of the appreciation of tuberculosis of the mucous membranes and its complications requires a separate consideration of pulmonary, laryngeal, intestinal, and uro-genital tuberculosis.

Pulmonary Tuberculosis.—This term includes the various disturbances that result from the invasion of the respiratory tract by the bacillus of tuberculosis. Acute miliary tuberculosis of the lungs has already been considered as of hæmatogenous origin and a part of general miliary tuberculosis, although it is possible that a localized acute miliary tuberculosis of the lungs may arise and be disseminated throughout the lungs from the inhalation of large numbers of the bacilli in minute subdivision. This condition, however, is rare, and its manifestations would not essentially differ from those described in connection with acute general tuberculosis.

The more important varieties of pulmonary tuberculosis are those in which bacilli are inhaled, the hæmatogenous form having already been considered, and the pleurogenic variety being, in the main, of no practical importance. One or more foci of localized tuberculosis are produced, from which the lung becomes more and more extensively invaded. Although the term pulmonary tuberculosis indicates the seat and cause of the disturbances, the lesions are so various that their separate anatomical diagnosis is often simply problematical. The term phthisis is therefore to be preferred as indicative of the especial clinical characteristic,—namely, the emaciation. Fibroid phthisis has no necessary connection with tuberculosis.

PULMONARY PHTHISIS. PULMONARY CONSUMPTION. PULMONARY TUBERCULOSIS.

Etiology.—In the etiology of pulmonary phthisis there are certain features which have been already mentioned as important in the etiology of tuberculosis. The invasion of the lung by the bacillus is essential, and it is chiefly introduced by means of the inhalation of particles of dried sputum. Less frequent is the evacuation into the lung of a softened tubercular gland or a softened tubercular abscess from caries of the spine or ribs. The effects of the invasion vary. Of a number of individuals equally exposed the bacillus will find suitable conditions for its growth and propagation in some and not in others. Such conditions are to be found in an inherited or acquired vulnerability of the tissues. Local causes are also important, as is seen in the frequency of pulmonary phthisis in persons with a malformed thorax, congenital stenosis of the pulmonary artery, previous pleurisy, and following measles and whooping-cough, in which contagions the catarrhal affection of the respiratory tract apparently acts as a localizing cause.

MORBID ANATOMY.—As a result of the presence of the bacilli upon the respiratory surface, a series of local changes arise both superficial and interstitial. To the former the terms *cheesy pneumonia* and *cheesy bronchitis* are applied in virtue of the appearance and localization of the anatomical products. In cheesy pneumonia the alveoli become filled with large cells of an epithelial character, either desquamated alveolar epithelium or transformed leukocytes. Such cells are prone to undergo rapid necrosis, and soon present the caseous appearance. The alveolar walls and the neighboring interstitial tissue become thickened, and a formation of large endothelioid cells takes place in them. The smaller branches of the pulmonary artery and the alveolar capillaries become obliterated. The affected portion of the lung thus contains no air, is solidified, and deprived of blood, a condition which has received the term cheesy hepatization. These alterations are distributed over smaller and larger areas. Thus, one may find miliary foci of cheesy pneumonia in which few alveoli are affected, or the solidification may exist as a lobular or a lobar pneumonia, or as a broncho-pneumonia. The extent of the primary distribution of the lesions depends largely upon the number of bacilli inhaled and the length of time during which such inhalation has been taking place. Bacilli may proceed from such superficial exudation into the interstitial tissue of the lung and give rise to miliary tubercles as a complication of the process. These may abound in the vicinity of the superficial changes, but are so incorporated with them that they are often not to be identified except when present in relatively normal portions of the lung, and especially when seen in the pleura.

A similar superficial exudatory process occurs in the smaller bronchi and results in a cheesy bronchitis, the canal of the bronchus being filled with a necrotic exudation, the wall also becoming necrotic after being infiltrated with endothelioid cells. An inflammation of the pulmonary alveoli surrounding such bronchi may take place, and foci of broncho-pneumonia thus arise. These alterations are often earliest found at the apices of the lungs, and this localization, according to Orth, depends upon the favorable opportunity for the retention of the bacilli, the lessened resistance of the tissues from the diminished blood-supply in these portions of the lung in consequence of their incomplete expansion in ordinary respiration, the frequent presence of pleural adhesions, and the liability of the bacilli to be forced into the apex by deep and violent inspiration during fits of coughing. The bacilli once having produced these disturbances in any particular part of the lung, the cheesy masses may become encapsulated in cicatricial tissue or impregnated with lime salts and indefinitely remain inert. The cretaceous nodules and puckered cicatrices so often found, especially at the apices of the lungs, are thus explained.

On the other hand, the chief source of danger from the localized foci of cheesy pneumonia is from softening. The softened, cheesy material escapes into the larger bronchi, and a *cavity* results, at first with an irregular, opaque yellow wall, the free surface of which continues to become softened perhaps more rapidly, as especially suggested by the experiments of Prudden, from the presence and growth of other bacteria than the bacillus of tuberculosis, while at the same time the surrounding alveoli become the seat of the advancing cheesy inflammation. The enlargement increases, neighboring cavities become fused, and eventually an entire lobe may become cavernous. The larger cavities have a relatively smooth wall, upon which an opaque gray pyogenic membrane is adherent and from which slight but frequent hemorrhages readily arise. Such cavities are crossed by trabeculæ, which represent the persistence of branches of the pulmonary artery whose tissue offers the most resistance to the advance of the destructive process. Small aneurisms of these arterial branches may be found, and are the source of the extreme, and sometimes immediately fatal, hemorrhage which occurs in pulmonary phthisis.

With an extension of the softening process to the surface of the lung the pleura frequently becomes necrotic, is torn during the act of coughing, and the contents of the cavity enter the pleural cavity, producing a pneumothorax rapidly becoming a pyopneumothorax from the associated pleurisy. As the softened contents of the cheesy mass, in which bacilli in large quantities are present, pass along the larger bronchi, the mucous membrane is infected, and miliary tubercles and cheesy bronchitis arise. The bacilli are also aspirated, especially during the act of coughing. into smaller branches, hitherto uninfected, of the main bronchus through which they are passing. New foci of miliary, broncho-, and lobular cheesy pneumonia are formed, undergo softening, and are transformed into cavities which become confluent, and thus the destruction of the lung extends.

Other changes than those above described are circumscribed cellular and fibrino-cellular exudations, which are attributable to the presence of other bacteria than the bacillus of tuberculosis. Certain parts of the lung are collapsed from a plugging of the respective bronchi, while other portions are in a state of collateral emphysema. Bronchial dilatation is frequent, and new-formed fibrous tissue abounds.

The pleuræ are usually thickened, sometimes being of almost cartilaginous density, and adhesions are formed between the costal and pulmonary pleuræ, at times so firm that in the removal of the lung the costal pleura is torn from the wall of the chest. The thickened pleura is often crowded with miliary tubercles, at times agglomerated into cheesy masses. The bronchial glands are increased in size, and usually contain miliary tubercles and cheesy nodules. The latter tend to become fused, softened, or calcified, and are often of a dirty gray color from the presence of particles of black pigment.

In many cases of chronic phthisis evidences of tuberculosis are to be found elsewhere, especially in the intestine, kidneys, and spleen. Laryngeal and tracheal tuberculosis are frequent complications, while tubercular endocarditis sometimes is present. Fatty infiltration of the liver is common, and amyloid degeneration of the spleen, kidneys, liver, and intestine is frequent.

SYMPTOMS.—In the consideration of the symptoms of pulmonary consumption a distinction of practical importance is to be drawn between two classes of cases. In the one the disease rapidly progresses, and terminates fatally in the course of a few weeks or months; in the other the disease slowly advances, and is continued over a period of years. The former is the acute, quick, or galloping consumption; the latter, the usual form, is chronic consumption.

ACUTE PULMONARY PHTHISIS.

The symptoms of *acute consumption* may arise in a person previously in apparent good health, or may appear in one who has for some time suffered from the symptoms and signs of a latent or incipient pulmonary tuberculosis, when the unexpected rapid progress of the disease places it definitely in the group of acute phthisis. Young persons, particularly children after measles, whooping-cough, or influenza, are especially liable to this variety. The disease may begin suddenly, perhaps after exposure to cold or in the sequence of an attack of hæmoptysis. Chilliness or a chill is followed by fever, accompanied by cough, a rapid pulse, dyspnœa, and pain in the chest. In other cases the symptoms suggestive of infection of the respiratory apparatus may be delayed for several days or a few weeks, the febrile condition not being accompanied by other than general symptoms. The temperature rapidly rises to 103° or 104° F., with morning remissions and evening exacerbations, often with daily differences of two or three degrees, which course is likely to remain continuous throughout the disease. This continued elevation of temperature is often suggestive of typhoid fever, which suggestion is favored by the occurrence of hebetude or delirium. The patient sweats freely, especially during the daily fall of temperature.

There are loss of appetite, rapidly progressive emaciation, and marked failure of strength. The respiration becomes accelerated and remains quickened, and there may be but little cough or thoracic pain. The cough at the outset is simply irritative, with but little expectoration, and may be due to an associated laryngitis, in which case there is also hoarseness. This laryngitis of the earlier stages of phthisis is the result rather of a catarrhal inflammation than of tubercular ulceration. In the latter stages of the disease, however, extensive tubercular lesions may be present as a result of infection of the larynx by tubercular sputa. Pseudo-membranous ulcers of the larynx may also occur in phthisis, presumably as the result of the presence of other bacteria than the bacillus of tuberculosis.

The alterations within the lungs are usually found at the apices at the onset, but rapidly involve other portions, either continuously or discontinuously, so that the physical signs at first may be indicative of a broncho-pneumonia or a lobular pneumonia, but quickly present the characteristics of a lobar pneumonia affecting one or more lobes, perhaps the greater part of both lungs. An important characteristic of these physical signs is their persistence. The dulness, tubular breathing, and increased vocal resonance and vocal fremitus in those regions in which the bronchi are not obstructed, with perhaps tympanitic areas from collateral emphysema or solidification over large bronchi, are also the physical characteristics of acute pneumonia. Moist râles are to be heard in various parts of the lung.

The secretion from the lungs may be scanty throughout the course of the disease. It is at first mucous, then generally becomes more and more purulent, and is usually viscid. It presents the physical characteristics of the sputum of a bronchial catarrh. If the patient lives long enough for softening of the cheesy products to occur, the sputum becomes more abundant, is largely purulent, and contains elastic fibres as well as numerous bacilli. The latter are to be sought in the sputum raised from the lungs, especially in the opaque yellow pus. If but few bacilli are present and pus is abundant, Biedert recommends that a drachm of the sputum be diluted with three drachms of water and fifteen drops of caustic potash and be heated on a sand-bath for two hours. The pus-corpuscles become dissolved, and the bacilli, even if few, are readily found in the sediment. To detect their presence, thin cover-glasses are to be carefully cleansed in water and strong alcohol. A bit of purulent sputum is to be removed by a platinum wire freshly heated and spread upon the cover-glass in as thin a layer as possible. The smeared glass is then to be carefully dried by being held in forceps over the flame of a Bunsen burner. The essential characteristic of the bacillus of tuberculosis is that it becomes stained by solutions of the aniline dyes to which a mordant has been added, and is not decolorized when acted upon by acids and alcohol. The stained bacillus sometimes assumes a beaded appearance, attributed to the presence of unstained spores or vacuoles. The method of staining recommended by Gabbet is the most convenient, since it requires the least time. The cover-glass smeared with the sputum, after being dried, is placed for a minute or two in a warm solution of one part of fuchsine, five parts of carbolic acid, ten parts of alcohol, and one hundred parts of water; it is then to be washed in water and put for a few minutes into a solution of methylene-blue two parts, sulphuric acid twenty-five parts, and water one hundred parts. It is again to be washed in water, and may be examined in this fluid, or may be dipped in alcohol, dried with filter-paper, and examined in oil of cedar or any other essential oil. If the preparation is to be permanently preserved in Canada balsam it should not be examined in oil of cloves, since this agent in time causes the color to fade. The bacilli are stained red; the rest of the specimen is blue. The coloring fluids are subject to changes when long preserved, and fresh solutions should be occasionally made.

The progressive involvement of unaffected areas of the lung, or the absence of a critical fall of temperature during the second week of the disease, may first excite the suspicion of the phthisical nature of the process. The presence of the characteristic bacilli usually gives the first positive evidence of the tubercular nature of the process. Death may occur in a few weeks during this stage of consolidation, or take place in the course of two or three months, in which time cavities may arise as the result of the rapid softening of the inflammatory product.

Diagnosis.—The symptoms at the outset being those suggestive of typhoid fever or pneumonia, the former is to be excluded by the absence of a characteristic range of temperature, the rash, abdominal symptoms, a palpable spleen, and perhaps the presence of a leukocytosis. The physical examination of the chest shows persistent areas of consolidation, to be found only in typhoid fever in the later stages of this disease and in the dependent portions of the lung. Acute pulmonary phthisis is to be differentiated from acute fibrinous pneumonia by the persistence of the signs of consolidation, the considerable daily differences in temperature, the lack of a critical fall of temperature, the frequent presence of a moderate instead of a considerable leukocytosis, and the eventual appearance in the sputum of characteristic bacilli. Important suggestive evidence of the phthisical nature of the disease may be found in the previous history or personal characteristics of the patient.

Prognosis.—Although cases of acute phthisis usually terminate fatally in the course of six weeks, a temporary arrest of the process may take place and death be delayed for a few months, or chronic phthisis supervene.

CHRONIC PULMONARY PHTHISIS.

The symptoms of chronic pulmonary phthisis are usually of gradual development, and it is of the greatest practical importance to recognize them at the earliest possible moment. A distinction is thus generally drawn between incipient and advanced phthisis.

In *incipient phthisis* the patient complains of being run down, assigning no cause for the increasing weakness. His friends notice slowly increasing emaciation and pallor. The appetite fails, and digestive disturbances are often complained of. The patient speaks of feeling chilly even in a warm room, and admits that slight exertion causes shortness of breath. In such persons the use of a thermometer indicates an elevation of temperature, a local cause for which first becomes manifest on examination of the lungs. In other cases with similar increasing weakness, hæmoptysis, perhaps after slight exertion, takes place, and is often found to be associated with localized physical signs, although months may elapse before they appear. In a third series of cases the patient, perhaps after some slight exposure to cold or wet or to a draught of air, feels chilly and suffers from a frequent cough, at first dry and hacking, then moist, which persists in frequency and remains obstinate to treatment. This persistence of even an apparently slight attack of bronchial catarrh often leads to the recognition of its cause by examination of the sputum, even before areas of consolidation are found in the lung. Again, the early phthisical symptoms may follow an attack of pleurisy, although in such cases the probability of the pleurisy being of tuberculous origin is directly suggested. It is, however, possible that a lung prevented from expansion by pleuritic effusion may offer suitable opportunities for the growth of the bacillus of tuberculosis.

In brief, the most important suggestive, because the most constant, sign is persistent elevation of temperature without obvious cause, often associated with chilly sensations and perhaps with frequent sweatings, especially at night. More characteristic is the hæmoptysis, which is the initial suggestive symptom in a large number of cases, although the physical examination of the chest may not reveal its source nor that of the sputum indicate its cause.

Sooner or later the physical evidence of the incipient stage of pulmonary phthisis is obtained. In the great majority of cases the pulmonary signs appear at the apices and near the anterior border of the upper lobe, rather more frequently in the right than in the left lung. The evidence obtained by percussion is less constant and significant than that resulting from auscultation. Dulness should be sought in the fossæ immediately above and below the clavicle, also in the supraspinous fossæ, although absence of dulness in either of these regions does not indicate that the subjacent apex is free from disease. On auscultation of the same regions the signs of chief importance as evidence of incipient phthisis are prolonged and harsh expiration and fine moist subcrepitant râles at the end of the inspiration, which is often feeble and jerky. Such signs are significant merely of a bronchiolitis, while solidification is indicated by bronchial or tubular breathing with exaggeration of the whispered voice. The cause of this circumscribed bronchiolitis is demonstrated if characteristic bacilli are found in the sputum. At this stage repeated examinations may be necessary, and it is often difficult to obtain sufficient sputum for the purpose.

In *advanced phthisis* the symptoms of the pulmonary affection are more characteristic. Cough and thoracic pain are conspicuous, and the respiration becomes constantly quickened. The cough varies in severity in the individual cases. At times it may be so slight as to attract no attention. Again, it may be so severe and persistent as to awaken the patient, produce vomiting, and cause him to fear suffocation. It is usually more frequent in the morning, and is sometimes so annoying in the night as to prevent sleep. The paroxysms of coughing often occur without the raising of sputum, and in persons of sensitive nervous temperament may be almost incessant until the attention of the patient is diverted.

The sputum which is raised from the lung varies in character according to the stage of the disease and the severity of the process. As already stated, in incipient phthisis there may be little or no sputum. As the disease advances, that which first appears is rather mucous than purulent, perhaps containing minute opaque yellow flocculi, but later mucous sputa may alternate with thick purulent sputa, or all sputum may be largely purulent. The more abundant the sputum, the more likely the presence of cavities due to the softening of caseous masses, the size of which may be suggested by the quantity of sputum coughed up. Defined masses of purulent sputum but little aerated and sinking in water are called *nummulated sputa,* and are considered to be evidence of the presence of cavities. The sputum is often streaked or stained with blood. In the former case the congested bronchial mucous membrane is regarded as the source of the bleeding ; in the latter, the pyogenic membrane lining the wall of cavities is the source. The sputa are sometimes of an extremely offensive odor, from the presence of putrescent bacteria in the pulmonary cavities. The diagnostic importance of the microscopic examination of the sputum for the bacilli has already been mentioned. Elastic fibres are often sought for, and frequently with negative results. Their presence is indicative of the formation of cavities, although cavities may exist and elastic fibres not be found. They are most conveniently shown by the examination of a portion of the sputum in caustic potash. If they are present in small numbers they are more satisfactorily isolated by boiling the sputum in a ten per cent. solution of caustic potash ; after some hours the fibres are found at the bottom of the glass.

Thoracic pain, usually sharply defined, is more frequent as an early than as a late symptom of advanced pulmonary phthisis. Its presence is indicative of an associated pleurisy, and is the more severe the more acute and extensive the latter. The sudden onset of an intense pleuritic pain, associated with a rapid and difficult respiration, a rise of temperature and pulse, and marked prostration, is suggestive of the rupture of the wall of a cavity and the production of a pneumothorax, which usually becomes a pyopneumothorax. The symptoms and signs of this affection are more fully considered in connection with the subject of pleurisy. (See Pleurisy.) Pleuritic pain is aggravated by coughing, and often necessitates rapid and superficial breathing; but as the pleura becomes thickened or the pleural cavity obliterated the pain ceases to be conspicuous. Its situation varies from time to time as previously unaffected portions of the pleura become involved, and it is now referred to the apex, now to the interscapular or axillary regions. The shortness of breath so apparent on inspection of a phthisical patient is not accompanied with evidence of suffering. The respiration readily becomes increased on slight exertion, but the breathing is difficult only when large quantities of secretion obstruct the bronchi and vigorous efforts are required to cause their removal.

When the phthisical alterations of the lung have become advanced, *hæmoptysis* is so frequent as often to be a matter of jest among patients in health-resorts frequented by consumptives. The spitting of blood may occur suddenly and unexpectedly, perhaps waking the patient from sound sleep, or it may follow exertion, excitement, or a severe paroxysm of coughing. The mere streaking or staining of sputa with blood hardly excites comment, and gives rise to no general disturbance. The hæmoptysis which is of greater significance is the escape of bright frothy blood, apparently flowing into the mouth and rather spit out than coughed up. Considerable quantities of blood may thus escape in a short time and cause the patient to become decidedly weakened. In general, the loss of blood is not an immediate source of danger, and Flint has stated that recoveries were more numerous in cases in which hæmoptysis occurred than in those in which it was absent, though recurring and considerable hemorrhages produced a corresponding degree of anæmia with its various symptoms. Immediately fatal hemorrhage from the rupture of aneurisms into cavities may take place, death being in part occasioned by the immediate loss of blood, but being also in part dependent upon asphyxia from the filling up of the lung by the blood.

PHYSICAL EXAMINATION.—On inspection, the chest is usually either long and narrow or flattened in the antero-posterior diameter. This thoracic formation is regarded rather as a cause than as the result of the disease. The greater the degree of emaciation the more prominent are the ribs, clavicles, and scapulæ. The ribs usually project, and the intercostal spaces are conspicuous. The fossæ above and below the clavicle and the supraspinous fossæ are abnormally deep. One or both supraclavicular spaces may be shrunken and incapable of distention, from a thickened, adherent, and retracted pleura and from solidification of the pulmonary apex. Defective expansion of the chest may be seen on the side of the affected lung. The skin, especially over the sternum, frequently shows a yellowish-brown discoloration, from pityriasis versicolor. Clubbing of the fingers and incurvation of the nails are frequent in chronic cases, and similar changes may take place, though to a less degree, in the toes.

Palpation, as well as inspection, reveals a defective expansion, especially of the apices. By palpation during phonation may be recognized the modification of the tactile fremitus, which is increased over solidified portions of the lung and diminished in the presence of pleural effusion or thickening. Normally, the tactile fremitus is somewhat more marked at the right than at the left apex.

Percussion gives evidence of solidification with or without a thickened pleura, and of the presence of cavities when of considerable size and of superficial seat. In solidification there is dulness tending towards flatness and accompanied by a sense of increased resistance; if the dulness is slight, it is best recognized at the end of prolonged inspiration. If there is a superficial cavity, percussion elicits a high-pitched tympanitic tone, the pitch being higher, as shown by Wintrich, when the mouth is open, and, according to Gerhardt, also changing with a change of the patient's position from the dorsal to the upright or the reverse. The resonance on percussion may be but little altered in case of minute disseminated areas of solidification or in miliary tuberculosis, and may be tympanitic over the apices from the presence of cavities. A dull tympanitic note may be present in the absence of cavities when extensive solidification of the lung overlies the large bronchi near the root of the lung. Absolute flatness is indicative of a combination of extensive solidification of the lung and a thickened pleura with or without the presence of fluid. If fluid is absent, the chest is retracted; if it is present in sufficient quantity, the ribs are separated and there is no motion of the intercostal spaces.

Beginning consolidation of the lower lobes is usually first apparent at their upper portions, which lie in the interscapular regions on a level with the fifth dorsal vertebra.

On auscultation the evidence of obstruction to the entrance of air into the bronchioles is furnished by a diminished and often jerky inspiration. More constant and important is prolongation of the expiratory sound. The respiration assumes a tubular character when the lung-tissue surrounding the open bronchi is consolidated. Râles are to be heard even before dulness or tubular breathing is apparent. In the earliest stages the fine crackling, crepitant, or subcrepitant râle is to be heard at the end of inspiration, especially on coughing or when a long breath is drawn. As the disease advances and the sputum resulting from the associated bronchitis is raised, coarse and fine, moist and dry râles are numerous, either localized or widely distributed, according to the extent of the process. When the cheesy material softens and cavities are formed, coarse bubbling or gurgling râles are to be heard, and, at times, sounds suggesting a metallic tinkling. Increased vocal resonance gives important evidence of consolidation, and in the early stages of phthisis is best determined by listening at the apices for the whispered voice. The vocal resonance transmitted through a cavity often assumes a bleating sound, to which the term ægophony or pectoriloquy is applied. Over the unaffected portions of the lung, especially when extensive solidification exists, the normal broncho-vesicular murmur becomes harsh, rude, or puerile. The presence of an associated pleurisy is indicated in its early stage by the sound of friction or rub, while a chronic thickening of the pleura or an accumulated exudation produces a muffling or an obliteration of the respiratory and vocal sounds.

The existence of a cavity is to be determined by the following group of sharply defined physical signs. Percussion produces a dull tympanitic note, amphoric in character in large cavities with thin walls, and perhaps presenting a cracked-pot sound best to be heard when the mouth is open, especially if the orifice of the stethoscope is placed in front of the mouth. On auscultation the respiration is tubular, cavernous, or amphoric, according to the size of the cavity. The râles are bubbling or gurgling, and may have a metallic sound. The vocal resonance is hollow, amphoric, or ægophonic.

A hæmic systolic murmur is frequent both at the apex and in the region of the valves of the pulmonary artery, and the signs of acute endocarditis sometimes occur. The heart-sounds are freely transmitted in the region of large cavities, and a subclavian murmur is often heard, attributable to pressure upon or traction of the subclavian artery. The cardio-respiratory murmur or systolic souffle ceasing when the breath is held may be heard over the larger bronchi.

COURSE AND DURATION.—During the progress of chronic phthisis the persistent cough and fever and the loss of flesh and strength are the conspicuous symptoms. The fever pursues an extremely irregular course, and frequent observations during the twenty-four hours may be necessary to determine its character. There are times when the exacerbations are so periodical as to suggest malaria, and again these may alternate with days of normal temperature. Indeed, in many cases of chronic phthisis the temperature may remain normal or subnormal for long periods of time. The intermittent or remittent types of fever are called *hectic*, and are usually associated with flushing of the cheeks and considerable sweating. The latter is more frequent at night, although it may occur by day or night, usually while the patient is asleep. The night-sweats in particular not infrequently form a most annoying symptom.

The various functions of the body are disturbed. The condition of the mind is notably one of activity, even of exhilaration, and, despite grave symptoms, the patient remains hopeful. The general or local symptoms of intracranial tuberculosis already mentioned arise when meningeal or cerebral tuberculosis occurs as a complication, and the numbness or pain characteristic of a peripheral neuritis sometimes is present. There is no other disturbance of the circulation than is made evident by the rapid pulse associated with the fever and by the diminished force attributable to the accompanying atrophy of the heart. According to Henry, the anæmia of phthisis does not progress as this disease becomes more severe. Acute endocarditis is rare, but thrombosis of the pelvic venous plexus and of the veins of one or both legs occasionally occurs in the latter stages of the disease. The venous thrombosis is a cause of œdema in one or both legs, also of tenderness along the course of the femoral vein, and may prove a cause of sudden death by the production of embolism of the pulmonary artery.

There are loss of appetite, nausea and vomiting, distress from food, and diarrhœa. The vomiting is often induced by coughing, and the diarrhœa may become persistent and severe. In the latter case tubercular ulcers or amyloid degeneration of the intestinal mucous membrane is to be suspected. Tubercular ulcers of the intestine may infect the peritoneum and cause a general or a circumscribed peritonitis, resulting in fistulous communications between neighboring portions of intestine or between the intestines and a hollow organ or the abdominal wall. Anal fistula is a frequent complication of both the early and the late stages of phthisis, and may be of a tubercular nature, as is suggested by its occasional resistance to therapeutic measures.

The urine not infrequently shows a trace of albumin, especially during the febrile exacerbations, and in the latter stages of the disease may contain abundant albumin, one-half per cent. or more, from a complicating amyloid degeneration of the kidney. The quantity is then increased, the color pale, the specific gravity in the vicinity of 1010, and hyaline and fatty casts are present. If tuberculosis of the uro-genital tract exists as a complication, the urine may contain pus, blood, and the characteristic bacilli.

The menstrual function is eventually suppressed in advanced phthisis. During the earlier stages, although the catamenia are scanty, pale, and watery, pregnancy may occur, in which case the progress of the disease is frequently arrested and temporary improvement in the condition of the patient takes place. After the birth of the child, however, the disease is likely to advance with renewed activity.

The duration of chronic phthisis is very uncertain. The frequency of the evidence of healed tuberculosis in persons who have recovered from the disease is familiar to all pathologists, and the indefinite arrest of incipient tuberculosis is a fact of daily observation. The gravity of chronic tuberculosis is known as well from personal experience as from the mortality statistics. The disease advances, comes to a stand-still, is retrograde, and again advances, its course extending over many months or a few or an indefinite number of years. Death usually results from increasing debility and eventual pulmonary œdema, or it may occur from a complicating acute disease, as pneumonia or typhoid fever. In certain cases anæmia and debility are accelerated by amyloid degeneration, and the patient dies with conspicuous symptoms of amyloid nephritis. In other cases delirium, sopor, and coma due to tubercular meningitis may be the signs of impending death, while in still other instances the patient may die of cerebral anæmia or suffocation from sudden and severe pulmonary hemorrhage.

DIAGNOSIS.—The early stage of chronic phthisis is alone difficult of diagnosis, which ultimately depends upon the discovery of the characteristic bacilli in the sputum. The physical signs of a persistent bronchiolitis, or of consolidation at one or both apices or elsewhere in the lung, especially when associated with an elevation of temperature, are significant of phthisis. They are of especial importance in those cases in which the bacilli are not discovered, owing either to the absence of a characteristic sputum or to the presence of the bacilli in very minute quantities. The persistent presence of the bacilli is essential to the diagnosis of a tubercular phthisis, since they may at times be in the sputum without having obtained a permanent lodgement in the lung. Even with the signs of cavity and chronic cough the presence of the bacilli is essential to the diagnosis of phthisis, since chronic bronchitis and bronchiectasis of non-tubercular origin may give rise to such signs. The discovery of elastic fibres is less essential to the diagnosis of tuberculosis than to the recognition of the destruction of lung-tissue.

PROGNOSIS.—The prognosis of phthisis becomes the more grave the farther advanced the disease. In the incipient stage it is usually relatively favorable provided the patient can sufficiently control his surroundings. Even with the lack of such control recovery is not infrequent, as is shown both by clinical observation and by anatomical investigation. Unfavorable prognostic signs are persistent elevation of temperature, progressive loss of weight, repugnance to food, and chronic diarrhœa. The occurrence of such complications as tubercular meningitis, laryngitis, pneumothorax, and amyloid disease of the abdominal viscera is usually indicative of a speedy termination.

LARYNGEAL PHTHISIS.

This term is indicative of chronic tubercular affections of the larynx. Such are usually secondary in character to pulmonary tuberculosis, and are attributable to the passage through the larynx of sputa containing the bacilli of tuberculosis. As a rule, superficial miliary tubercles arise, which rapidly become necrotic, and lenticular ulcers result, tending to spread laterally. The older and larger ulcers have an opaque gray or grayish-yellow base, from necrotic tubercles, and are present throughout the larynx, but are most abundant upon the epiglottis and in the vicinity of the arytenoid cartilages and the false vocal cords. Extensive destruction of the epiglottis may take place, and an arytenoid perichondritis is of frequent occurrence, resulting in sequestration of the cartilages, which may be coughed up. The mucous membrane is injected and swollen, and hemorrhages may be present.

Another variety of tuberculosis of the larynx is to be seen in lupus of this organ, which is usually the result of the extension inward of a cutaneous lupus. This affection of the larynx is characterized by papillary outgrowths from the epiglottis and the vicinity of the vocal cords. Such outgrowths become thickened and nodular, and undergo necrosis, and ulcers result which may heal with the formation of extensive cicatrices.

Laryngeal phthisis may develop in the early stages of pulmonary phthisis before extensive lesions are apparent, in which case its primary nature is suggested. More frequently it develops late in the disease. Hoarseness may occur, the degree of which may vary from time to time, and even complete loss of voice arise. Frequent rasping cough, laryngeal pain, and difficulty in swallowing are significant symptoms. The difficulty in swallowing may be such that excessive irritation of the larynx arises and spasm of the glottis follows. Regurgitation, perhaps vomiting, occurs, and suffocation is threatened. The secretion from the larynx is of slight or moderate quantity, is purulent, and often contains specks of blood, while characteristic bacilli are present. Laryngeal phthisis is rarely recovered from except in the earliest stages, although lupus of the larynx may heal. The duration of the symptoms is largely dependent upon that of the pulmonary affection, and, like the latter, when not especially severe, may extend over years. The diagnosis is based upon the recognition of the anatomical changes by means of the microscope, and confirmed by the discovery of typical bacilli in the secretion removed from the larynx.

FIBROID PHTHISIS.

This term denotes chronic alterations of the lung resulting in the formation of abundant fibrous tissue with a corresponding atrophy of the parenchyma. The condition is essentially one of chronic fibrous pneumonia, and has received the designation of cirrhosis of the lung. The limited formation of fibrous tissue is of frequent occurrence in the course of chronic phthisis, due to the presence of the bacilli of tuberculosis, but in that affection the wasting of the individual and that of the lung predominate over the formation of fibrous tissue. In fibroid phthisis, so called, the fibrous changes in the lung predominate over the wasting of the individual. It is possible that the bacillus of tuberculosis may produce extensive diffused thickening of the fibrous tissue of the lung in the absence of its more characteristic results, but when the bacilli are found in fibroid phthisis they are usually associated with cheesy conditions and are to be regarded as a complication. Fibroid phthisis is to be considered as the result of an acute pneumonia, a chronic bronchitis, or broncho-pneumonia from the inhalation of dust, or it may follow a chronic pleurisy. The symptoms are somewhat similar to those occurring in phthisis of tubercular origin. Cough, abundant expectoration, and some shortness of breath on exertion, with, in general, absence of fever and fairly good health, are characteristic of the condition. The further consideration of this subject will be found in connection with that of chronic fibrous pneumonia.

TUBERCULOSIS OF THE ALIMENTARY CANAL.

Tuberculosis may affect the alimentary canal from the mouth to the anus, although it is rare in all parts except the small and the large intestine. It occurs both as a primary and as a secondary affection. The former is to be seen in the indurated tubercular ulcers of the lips and tongue, which are often attributed to syphilis or cancer. Secondary tuberculosis is the rule, and the infection of parts above the pharynx usually results from the extension of lupus, cutaneous tuberculosis, into the mouth and pharynx, with the production of nodules, ulcers, and scars. Tuberculosis limited to the pharynx is usually secondary to that of the larynx, and the tonsils, base of the tongue, soft palate, and posterior wall of the pharynx may be affected, the disease even extending into the œsophagus. This portion of the alimentary canal may also become tuberculous from the evacuation into it of a tuberculous abscess of the vertebræ or of a softened tubercular bronchial gland. As elsewhere in the mucous membranes, the initial miliary tubercles become necrotic, are disintegrated, and the débris is carried away, leaving superficial ulcers tending to become confluent, and extending in depth by the invasion of the subjacent tissue by the bacilli. Sharply defined ulcers result, having an indurated base, speckled with gray and yellow, and showing but little inclination to hemorrhage. Tubercular ulcers may also be found in the stomach, although their presence in this viscus is rare. In most cases they are the result of the escape into the organ of the softened contents of neighboring cheesy glands. A tuberculous fistula may be established between the colon and the stomach, and tuberculosis of the stomach may result from the extension inward of a tuberculosis of its serous covering. The resulting ulcers are usually small and not associated with symptoms, although Orth states that they have caused death by producing hemorrhage and perforation.

INTESTINAL TUBERCULOSIS.

Next to the lungs the intestines are the most frequent seat of tuberculosis, being affected in about one-half the cases of this disease.

Etiology.—Intestinal tuberculosis usually results from swallowing bacilli, although it sometimes occurs from the extension of a tubercular peritonitis to the mucous membrane or follows the evacuation through the intestinal wall of a softened tuberculous gland. According to the source of the bacilli a distinction is drawn between a primary and a secondary variety of intestinal tuberculosis. Primary tuberculosis is found more especially among infants, and is considered to be due chiefly to the use of infected milk. Secondary tuberculosis is more frequent in adults than in children, and is chiefly due to tuberculous sputa, although milk, dairy products, and meat from tubercular animals sometimes furnish the bacilli.

MORBID ANATOMY.—In primary intestinal tuberculosis the lesions of the intestine consist of ulcers similar to those to be mentioned as due to secondary tuberculosis. In primary tuberculosis, however, the infection of the mesenteric glands from the intestinal mucous membrane may be so extreme that the alterations of these glands predominate over the changes in the mucous membrane. To this condition as found in children the term *tabes mesenterica* or *tabes meseraica* was formerly applied. The enlarged cheesy glands were considered to be manifestations of scrofula, but the tubercular nature of the alterations in the mesenteric glands is not only suggested by the microscopical appearances, but has also repeatedly been proved by the discovery of the bacilli and by the production of tuberculosis by inoculation of the cheesy material. Such tubercular glands are usually, though not necessarily, associated with tubercular ulcers of the intestine. The relation is thus analogous to that observed when cheesy bronchial glands are found without evidences of tuberculosis of the lungs.

Ulcers are the essential characteristic lesions of a tuberculosis of the intestine. They are usually largest and most numerous at the lower end of the ileum, but may be found disseminated throughout both the large and the small intestine, or may be limited to various portions of either, especially to the cæcum, the appendix, and the flexures of the colon or rectum. The primary lesion results from the presence of the bacilli of tuberculosis within the lymph-follicles, whether these are solitary or agglomerated in Peyer's patches. The tubercular tissue is produced, becomes necrotic and softened, and the overlying mucous membrane gives way, thus permitting the softened cheesy material to be evacuated into the intestine. A tubercular ulcer results, crater-like, with swollen injected edges and a gray or grayish-yellow base. The bacilli extend into the immediate vicinity, producing new tubercles, which also become caseous: hence the spread of the ulcer in width and depth, especially the former. In Peyer's patches the progress of the ulcer follows rather the width than the length of the patch, in accordance with the course of the lymph-vessels, and when the edge of the patch is reached the continuous mucous membrane becomes infected, and thus annular or girdling ulcers result. The peritoneum overlying the ulcer often contains translucent or opaque miliary tubercles, and, in the more chronic varieties, beaded and varicose opaque yellow lines, tuberculous lymphatics, are seen through the peritoneum to extend from the ulcer to the mesentery, and even along the latter to the nearest lymphatic glands, which become enlarged and cheesy from tubercular infection.

The usual tendency of the ulcer is towards extensive destruction of the mucous membrane, at times resulting in perforation, and it is the most frequent cause of tubercular peritonitis. Partial or complete healing of the ulcer occurs, although rarely, and then for the most part in the case of single or few ulcers. A pigmented scar remains, which, if the result of a girdling ulcer, may produce an extreme degree of narrowing of the intestine.

SYMPTOMS.—Chronic diarrhœa is the characteristic symptom of primary intestinal tuberculosis, and often alternates with temporary periods of relatively normal evacuations, and even with occasional constipation. The dejections contain abundant slimy material, which at times is blood-stained, and they are usually rather pultaceous than watery, either white, yellow, green, or brown, according to their frequency, the medicines used, and the nature of the food. The odor is often extremely offensive. The more numerous the dejections the more likely are the stools to be fatty and to contain undigested food. The movements of the bowels are often excited by food or drink, and are usually preceded by colicky pains, which are relieved by the evacuation. The appetite is frequently excessive, and the desire for solid food greater than that for liquid nourishment. The child becomes pale and emaciated, but the abdomen swells and is tense from the presence of gas in the intestine. Except over the abdomen, the skin is flaccid, its surface dry and rough, and the wrinkled face suggests that of old age. The eyes become sunken, and in infants the fontanelle also is depressed. The fever presents the general characteristics of the fever of tuberculosis, periods of intermission and remission being frequent, while intervals of subnormal temperature occur. During the febrile exacerbations profuse sweating takes place, especially from the head and back. Rather a sense of discomfort than suffering from pain is the rule. The distended abdomen is usually not tender, and as the tension is relieved by the escape of gas, the enlarged mesenteric glands, the especial characteristic of primary tubercular enteritis, are made apparent. They are to be recognized as resistant, somewhat movable tumors of a lobulated character, perhaps as large as pigeons' eggs, most frequently in the right iliac fossa and in the vicinity of the navel. The lymphatic glands in the groins, axillæ, and neck may be enlarged. Circumscribed abdominal tumors may also result from the extension of the tuberculosis to the peritoneum, with the formation of adhesions and exudations in the region of the appendix, between coils of intestine, and in the omentum. The course and results of primary intestinal tuberculosis are essentially those of the secondary variety.

Secondary tuberculosis of the intestine is frequently combined with pulmonary tuberculosis in both young and old. It may give rise to no symptoms, or may be characterized by diarrhœa, constant or occasional, in either case not readily yielding to treatment, while exceptionally actual constipation may be associated with extensive tuberculosis. The stools are usually painless, and often occur by night as well as by day. They are, as a rule, light-colored, and may contain abundant slime, especially when the large intestine is the seat of the ulcers, and blood is sometimes present in small quantity. The bacilli of tuberculosis may be found in the intestinal contents by the method employed for their recognition in the sputa. No diagnostic importance, however, is to be attached to their presence if the lungs are simultaneously affected, because of the frequency with which sputa are swallowed. Abdominal pain is usually slight, although occasional attacks of colic occur. The pain when present is often referred to definite parts of the abdomen, especially to the lower half, and tenesmus occurs when tubercular ulcers are present in the lower part of the rectum. The abdomen is retracted, and may be the seat of tender points indicative of extension of the ulcers towards the peritoneal surface of the intestine. If the tuberculosis is localized in the appendix or at the lower part of the rectum, the symptoms may be those of an appendicitis, a proctitis, or an ischio-rectal abscess, the tubercular nature of which is to be suspected only from the persistence of the symptoms and their failure to disappear under appropriate treatment. If intestinal obstruction follows the healing of the tubercular ulcer of the small intestine, its origin may be obscure, since solitary ulcers usually progress without symptoms.

Diagnosis.—The diagnosis of primary intestinal tuberculosis is based upon the persistence of the diarrhœa, the progressive anæmia and emaciation, and the discovery of the enlarged mesenteric glands. The latter feature, the most essential in diagnosis, may not be made out until towards the end of life. In like manner the diagnosis of the tubercular enteritis may first appear probable with the development of signs of tuberculosis elsewhere, especially of a tubercular peritonitis or meningitis. The diagnosis of secondary tubercular enteritis is based upon the occurrence of chronic diarrhœa in pulmonary tuberculosis. Amyloid degeneration of the intestine also occurs in the course of pulmonary phthisis and produces a diarrhœa. In such cases, however, evidence of amyloid degeneration is likely to be found in the spleen, liver, and kidneys. It is to be remembered that both amyloid degeneration and tuberculosis may be present in the intestine. The results of the extension of tubercular ulcers of the intestine to the peritoneum will be considered in connection with tubercular peritonitis.

TUBERCULOSIS OF THE LIVER.

In all cases of general tuberculosis, in most cases of abdominal tuberculosis, and frequently in pulmonary tuberculosis, the liver contains tubercles, the bacilli probably being introduced by means of the blood-vessels and the lymphatics. Disseminated miliary tubercles are oftenest present, and may occur in enormous numbers, although many are invisible without the microscope. They are to be seen with the unaided eye beneath the peritoneal covering and on section of the liver as minute, opaque white spots seated near the periphery of the lobules. They are easily distinguished when the liver, as is frequently the case, contains abundant blood, and, on the contrary, are recognized with difficulty when there is fatty infiltration of this organ. The tubercles are sometimes fibrous, and may then be associated with a cirrhotic condition of the liver.

Nodular tubercles also occur, either alone or with miliary tubercles, and are intimately connected with the bile-ducts, as is evident from the bile-stained softened centre of these nodules. Such tubercles are often larger than cherry-stones, and are cheesy at the periphery and softened at the centre. Their appearance suggests that the wall of the bile-ducts is first infected, and that the formation of tubercles takes place around them, producing results analogous to those occurring in tubercular peribronchitis.

Finally, a localized tuberculosis of the liver sometimes, though rarely, is present and forms a tumor as large as an egg. This is of a yellow color throughout, and miliary tubercles are to be found at the periphery. There are no symptoms characteristic of hepatic tuberculosis.

Tuberculosis of the Pancreas.—Tuberculosis of the pancreas is rare, whether in the form of disseminated or of localized tuberculosis. Miliary tubercles may be found in the vicinity of tubercular lymph-glands lying near the pancreas. Like hepatic tuberculosis, the pancreatic affection has no symptoms of clinical importance.

URO-GENITAL TUBERCULOSIS.

The uro-genital tract is often the seat of tuberculosis, and the infection is generally considered to take place by the admission of bacilli through the blood-vessels. This is suggested by the frequency of miliary tuberculosis and tubercular nodules in the kidney without any tuberculosis of the ureters or bladder, and also by the occasional occurrence of extensive tuberculosis of the apices of the Malpighian pyramids without any affection of the mucous membrane of the pelvis. The possibility of an ascending affection from below, either by the admission of the bacilli through the urethra or from a prostatic tuberculosis, has previously been mentioned. In the male a concurrent affection of both the urinary and the genital tract is common, owing to their unification at the neck of the bladder, while in the female such concurrence is infrequent, the urinary tract being diseased more often than the genital organs. Hence tuberculosis of the kidneys, prostate, seminal vesicles, vasa deferentia, epididymis, and perhaps of the testicle, is frequently associated. In the female, however, the tuberculosis is commonly limited to the urinary or to the genital tract. In the latter case the infection of the genital mucous membrane may be hæmatogenous, or the bacilli may enter the tubes from the peritoneal cavity and infect both them and the uterus from the mucous surface. The cervix and vagina are rarely tuberculous except in the far advanced cases of tuberculosis of the body of the uterus and the Fallopian tubes. Local tuberculosis of each part of the uro-genital apparatus is of practical importance and demands separate consideration.

Renal Tuberculosis.—Miliary tuberculosis of the kidneys has long been recognized, but the tuberculous nature of the so-called scrofulous kidney, or renal phthisis, has become a matter of general acceptance only since the discovery of the bacillus of tuberculosis as the cause of this affection.

Disseminated miliary tuberculosis of the kidney is part of a general miliary tuberculosis, and is manifested by the presence of few or many minute, gray, more or less opaque tubercles surrounded by an injected border. They are more abundant in the cortex, and readily observed on removal of the capsule. On section linear clusters of the tubercles are often to be seen continued towards the pyramids. Such disseminated miliary tuberculosis of the kidneys is usually bilateral and associated with a like affection of the bladder, prostate, or testicles, and there are no symptoms especially calling attention to this localization in the kidneys.

Chronic renal tuberculosis, renal phthisis, or scrofulous kidney, occurs oftenest during the middle third of life, although the extremes of life are not spared. It is frequently unilateral, and is manifested by the extension of cheesy masses from the apices of the Malpighian pyramids upward into the cortex of the kidney. At the periphery of these cheesy portions miliary and agglomerated gray and cheesy tubercles are to be seen. On microscopic examination the tubules are filled with necrotic epithelium, and large numbers of the bacilli of tuberculosis are often found. The interstitial tissue is also infiltrated with cells and is necrotic. The unaffected portions of the kidney become invaded by the bacilli, either along the tubules or by the entrance of the bacilli into the lymphatics or blood-vessels.

Softening of the parts first involved—namely, the apices of the pyramids—takes place as the disease advances into the substance of the kidney. A series of cavities thus arise continuous with the pelvis of the kidney, the intervening septa of normal tissue becoming more and more narrow as the invasion of the kidney progresses, till eventually the kidney may become a mere fibrous bag filled with liquid and curds, or both wall and contents may be so infiltrated with lime salts as to form a calcareous shell enclosing a mortar-like material. More often the kidney becomes enlarged and forms a tumor which is usually symmetrical, although the tubercular process may localize itself in a definite portion of the organ.

As the process in the kidney advances, the mucous membrane of the pelvis of the kidney, ureter, and bladder is invaded, miliary tubercles form in the superficial portion of the mucous membrane, and, becoming necrotic, their detritus is washed away in the urine. The infection extends in depth especially by means of the lymphatics, so that the entire wall eventually becomes a cheesy ulcer in which islets of relatively unaffected mucous membrane may exist. The contrast between the cheesy tubercular ulcer and the relatively normal mucous membrane is most conspicuous in the ureter and bladder. Both the pelvis of the kidney and the ureter frequently undergo extreme degrees of dilatation, and extension of the process to the paranephric tissue may occur and a perinephric abscess result. Amyloid degeneration is not infrequent in the course of chronic renal tuberculosis.

The symptoms which call attention to the disease of the kidney are of gradual onset, and are usually preceded by symptoms referable to the bladder, especially by frequent micturition. There is discomfort or pain, and eventually a resistant swelling, apparently a symmetrical enlargement, in the region of one or both kidneys. The urine is usually acid, albuminous, and contains pus-corpuscles, increasing in number as the disease progresses. Blood, epithelium, and granular material are also present. The bacilli of tuberculosis are to be sought in the sediment by the method followed in the examination of the sputum. Repeated examinations may be necessary, and in cases where the disease is not far advanced and the sediment is but slight, the centrifugal apparatus is of value in concentrating the bacilli. It is to be remembered that in the preputial smegma a bacillus has been found which resembles the bacillus of tuberculosis in appearance and in reaction to stains. The former, however, becomes immediately decolorized in alcohol, while the bacillus of tuberculosis retains its stain when exposed for several minutes. The general symptoms of chronic tuberculosis—namely, irregular fever, night-sweats, progressive emaciation and debility—are present.

Chronic renal tuberculosis affecting both kidneys is generally fatal during the course of from one to three years after its recognition. The possibility of recovery from unilateral tuberculosis has long been recognized, through the discovery, after death from other causes, of the remains of an old tuberculous process, which may be sometimes so extensive as to have led to the complete destruction of the kidney. Of late years the extirpation of a tuberculous kidney has repeatedly been followed by the recovery of the patient.

Tuberculosis of the Bladder.—Tuberculosis of the bladder is usually due to infection from the kidney, although it may proceed from tuberculous disease of the prostate, a hæmatogenous miliary tuberculosis of the bladder as part of a general tuberculosis being very rare. The anatomical appearances are essentially similar to those of the lesions occurring in the larger bronchi or the larynx. Superficial lenticular ulcers, at first discrete, coalesce and form ulcers of various size with a crenated outline and injected margin. These ulcers are most abundant at the neck of the bladder, in the vicinity of the orifices of the ureters, but may also be found at the fundus. As the disease progresses and infection of the wall of the bladder extends in depth, destruction of the mucous membrane occurs, the muscular coat is exposed, the subperitoneal tissue may be invaded, and peritonitis follow, or paracystic abscesses and fistulæ result.

The symptoms are those of a persistent vesical catarrh gradually increasing in severity. The neck of the bladder is especially sensitive: hence there is frequency of micturition associated with vesical tenesmus and painful flow of urine, the pain often radiating from the perineal region. Slight or considerable hæmaturia, often unexpected, is frequently the first sign suggestive of serious vesical disease. The urine is acid, with a trace of albumin, and contains but little sediment, which is composed of leukocytes, vesical epithelium, and blood-corpuscles. If the tuberculosis is limited to the bladder the bacilli are found with difficulty, and failure to discover them does not affect the diagnosis. Eventually the symptoms of a chronic febrile cachexia and those of tuberculosis elsewhere, especially in the uro-genital tract, become apparent, and the urine is likely to contain an abundant sediment, in which are the curds and blood-stained clots perhaps containing the bacilli. In the early diagnosis of tubercular cystitis suspicion should be aroused by the occurrence of chronic cystitis without obvious cause, resistant to treatment, while the urine is acid.

Tuberculosis of the Prostate and the Seminal Vesicles.—Acute miliary tuberculosis of the prostate is extremely rare. Chronic cheesy tuberculosis, on the contrary, is common, and is often associated with renal and vesical tuberculosis and with tuberculosis of the testis. This affection of the prostate is more frequently the result than the cause of tuberculosis of the urinary apparatus, while it may be either a cause or a result of an accompanying tuberculosis of the testicle. The bacilli are supposed to enter the gland-ducts, infect the wall, and produce desquamation and necrosis of cells and a tubercular infiltration of tissues. As a result the prostate becomes more or less enlarged on one or both sides, nodular or not, hard or soft, according to the size and number, necrosis, calcification, or softening of the tubercular masses within the prostate. The softened caseous masses are discharged into the bladder and leave cavities with cheesy walls. Perforation of the wall of the prostate, resulting in a tubercular infiltration of the neighboring fibrous tissue, takes place. Similar changes are found in the seminal vesicles, and in extreme cases the prostate and the vesicles may be transformed into a convoluted capsule filled with cheesy material in which lime salts are abundant.

The symptoms of prostatic tuberculosis resemble those of vesical tuberculosis. There is long-continued pain of gradual onset, referred to the perineum, and to the neck of the bladder or the rectum, and perhaps radiating towards the testis. The chief symptoms are a frequent scanty, opaque white or yellow urethral discharge, occasionally scanty hæmaturia, especially at the beginning of micturition, and a urine resembling that found in tuberculous cystitis.

The diagnosis is largely based upon the extreme sensitiveness of the prostatic portion of the urethra to the passage of a sound, and the eventual nodular enlargement with considerable variations in density of the prostate and seminal vesicles. The diagnosis of the tubercular nature of the palpable prostatic changes is strengthened by evidence of tuberculosis elsewhere in the uro-genital tract, and may be established by the discovery of the bacilli in the urine. Rectal examination of the prostate may often first indicate the existence of renal or vesical tuberculosis, or it may corroborate a diagnosis of tuberculosis of the testis.

Tuberculosis of the Testis.—Miliary tubercles may be found in the testicle as part of a general miliary tuberculosis, and they may also be found as the result of the extension of a tubercular process from the epididymis. Chronic tuberculosis of the testis usually proceeds from the epididymis, the latter becoming infected by the passage of bacilli from the bladder or prostate along the vas deferens. Tuberculosis may remain for a long time limited to the epididymis and the vas deferens, the wall of which is thickened, the surface infiltrated with necrotic tubercular tissue, and the canal filled with cheesy plugs. The epididymis may thus be transformed into a dense mass nearly as large as the testis which it encircles. In time the tubercular infection is likely to progress from the epididymis into the testis, which then becomes enlarged from the presence of tubercles and the formation of fibrous tissue. With the occurrence of softening the body of the testis may represent a series of cavities divided by fibrous septa and filled with cheesy material. The sac of the tunica vaginalis may contain more or less fluid or may be obliterated by adhesions. The softened cheesy material may escape through the adherent skin, in which are formed one or more chronic fistulæ with tuberculous walls. Such is the so-called *scrofulous testicle*, which often diminishes in size and increases in density with the persistence of the discharge.

There may be but little pain associated with tuberculosis of the testicle except during the stages of softening and beginning ulceration of the skin, although discomfort results from the weight of the considerably enlarged gland. The importance of palpation of the testicle in possible uro-genital tuberculosis is obvious, and this gland should be examined although the patient may be unaware of any abnormality connected with it.

Tuberculosis of the Sexual Organs of the Female.—The primary nature of tuberculosis of the genital tract of the female is suggested by the occasional isolated affection of the Fallopian tubes, while its extension as a secondary process from the peritoneal cavity is evidenced by the predominant alteration of the tubes when the uterus is involved, the rarity with which the ovaries are simultaneously tuberculous, and the usual freedom from disease of the cervix uteri and vagina. Observations of acute miliary tuberculosis of the mucous membrane of the uterus and

Fallopian tubes and of the ovary are rare. Chronic tuberculosis of these organs, with the exception of the ovaries, is by no means infrequent, and the alterations of the genital tract may predominate over the evidence of tuberculosis in other parts of the body. Usually they are accompanied by tuberculosis elsewhere, especially in the lungs.

Tuberculosis of the uterus is indicated by the presence of miliary tubercles in the mucous membrane, which coalesce and degenerate while the infection extends deeper into the wall. The uterus becomes enlarged, its cavity is dilated, and an extensive cheesy ulcer is formed as in chronic tuberculosis of the kidney or the lungs. The tubercular affection of the uterus usually begins in the region of the tubes and ceases at the internal os, although the cervical portion of the uterus may become affected when the uterine tuberculosis is long continued or in the rare event of the occurrence of an extension of the tuberculosis from the vagina upward. Tubal tuberculosis is usually double, and the tubes become elongated, dilated, and tortuous. Tubercular infiltration of the wall exists, and the canal is filled with cheesy material. Tubal tumors thus arise, the tubercular nature of which is usually unsuspected, since the symptoms are those common to chronic salpingitis.

Primary tuberculosis of the tubes may be productive of the hectic fever of chronic tuberculosis extending over a period of years, as in the case reported by Channing, and the symptoms of perforation into the rectum may eventually occur.

The *ovaries* may be the seat of miliary tubercles or may form tumors of considerable size with softened cheesy contents, as in tuberculosis of the testicle. If ovarian tuberculosis exists, it is usually accompanied by tubal and uterine tuberculosis, although the tubes and the uterus are often tuberculous when the ovaries are normal. The symptoms of tuberculosis of the genital tract of the female are those of chronic endometritis, salpingitis, or ovaritis, and the tubercular nature of the disease is usually first made known by the examination of the specimen removed at a surgical operation or at a post-mortem examination.

TUBERCULOSIS OF THE MAMMARY GLAND.

Of late years attention has been directed to the occurrence of tuberculosis of the mammary gland, which in the main has been found in females, especially during the period of child-bearing. The bacilli in certain cases are brought from a remote region probably by the blood-vessels, while in others they are directly introduced from neighboring tuberculous ribs or skin. Few or several cheesy nodules are present in the gland, and when softened are evacuated through the skin with the production of fistulæ. The skin of the breast also may become tuberculous, and the corresponding axillary glands are usually affected. The disease, when deep-seated, is to be suspected from the constant presence of circumscribed nodules of hard or soft consistency, which tend towards ulceration and the formation of fistulæ, and from the association of permanent enlargement of the axillary glands. An important diagnostic feature is the evidence of tuberculosis elsewhere, and the diagnosis is to be definitely established by the recognition of typical bacilli in the pus or scrapings from the wall of the sinus, or by the inoculability of the discharge from the fistulæ.

TUBERCULOSIS OF THE SEROUS MEMBRANES.

The pericardium, pleuræ, and peritoneum are frequently infected by the bacillus of tuberculosis. Although tuberculosis of the serous membranes may be part of a general infection in which the bacilli are transferred to the diseased membrane by means of the blood-current, usually each serous membrane becomes infected by the extension of tubercular processes from a neighboring part into the overlying membrane. Thus, pericardial tuberculosis commonly arises by the passage of bacilli from a tuberculous bronchial lymph-gland or from tubercular lungs, sternum, or spine. Pleural tuberculosis results from the passage of bacilli from a tuberculous lung or a tuberculous process in the spine, ribs, or sternum. Peritoneal tuberculosis most frequently is due to the passage of the bacilli from a tuberculous ulcer of the intestine, from the lungs through the diaphragm, from tuberculous mesenteric glands, or from tubercular Fallopian tubes. When tuberculosis affects one of these serous membranes the bacilli are often readily transferred to the others: hence tuberculosis of the several serous membranes often concurs. In acute miliary tuberculosis of these membranes gray translucent tubercles may be present, with no other inflammatory products or evidences of inflammation than a certain quantity of serous fluid in the cavity affected. Usually, however, the tubercles are associated with the products of exudative inflammation,—namely, serum, fibrin, or cells. The anatomical condition is therefore one of tubercular pericarditis, pleuritis, or peritonitis of a serous, fibrinous, or cellular character. The affection may be both acute and chronic, the latter being further characterized by the formation of fibrous tissue. All tuberculosis of serous membranes is essentially indicative of an inflammatory cause: hence the distinction between tuberculosis and tubercular inflammation of the membrane concerned is simply one of degree.

Tubercular Pericarditis.—Tuberculosis of the pericardium is usually acute, and is characterized by the presence of tubercles, fibrin, and more or less liquid exudation, the latter often being hemorrhagic. The tubercles may be readily overlooked, but are made evident when the fibrinous adhesions are removed from the thickened pericardium, in which they appear as minute, opaque gray specks often so closely approximated as to form plates, which are seen to best advantage on transverse section of the pericardium. Rarely the pericardial exudation is purulent.

Especial interest is connected with the occurrence of tubercular pericarditis in very old people, in whom it may be the immediate cause of death, being the sole conspicuous lesion found. In such cases the presumption is strong that the bacilli of tuberculosis have remained for many years inert in some part of the body, oftenest in the bronchial lymph-glands, and, suddenly entering the pericardium, cause its inflammation. It is also possible that they enter the circulation at a remote point and are engrafted upon a pericarditis due to other causes. In chronic tubercular pericarditis the tubercles are present as well in the fibrous adhesions as in the thickened pericardium. The opposed surfaces are firmly adherent to a greater or less extent, and cheesy masses composed of necrotic tubercles and inspissated exudation are to be found embedded in the adhesions. The symptoms and signs of acute tubercular pericarditis are largely those of acute pericarditis of non-tubercular origin, while those of chronic tubercular pericarditis are usually masked by the symptoms of tuberculosis elsewhere.

Tuberculosis of the Pleuræ.—In pleural tuberculosis the conditions are essentially the same as in pericardial tuberculosis. The exudation is usually abundantly serous, and is frequently hemorrhagic. As in pericarditis, so in pleurisy the tubercles may first be made evident after removal of the fibrinous false membrane, when they are seen mottling the surface or forming thick layers. Although tubercular pleurisy is usually secondary to tuberculosis in the immediate vicinity, it is probable that many cases of acute exudative pleurisy are due to a tubercular infection of the pleuræ. The bacilli may be in such limited quantity as not to be found on microscopical examination of the exudation, but the inoculation of guinea-pigs with the latter has frequently resulted in the production of tuberculosis. Eichhorst, for instance, has recently reported positive results in fifteen out of twenty-three inoculations of serum from patients attacked with acute idiopathic pleurisy. Acute tubercular pleurisy may be suppurative both in children and in adults, and in pyopneumothorax of phthisical origin the combination of suppurative and tubercular pleurisy is often seen. Chronic tubercular pleurisy is the combination in the pleuræ and adhesions of recent and cheesy tubercles and of inflammatory products inspissated, softened, or calcified. This variety is the frequent result of extensive chronic pulmonary tuberculosis, and also represents a terminal stage of acute tubercular pleurisy and tubercular empyema. Since the local manifestations of acute or chronic pleurisy are essentially the same whatever the cause of the inflammation, their clinical features will be considered in connection with the subject of pleurisy.

TUBERCULOSIS OF THE PERITONEUM.

The presence of tubercles in the peritoneum is associated with the variety of inflammatory products found in the other serous cavities. On account of the size of the cavity, the extent of surface, and the number and variety of the organs covered by the peritoneum, the anatomical results form a more complex grouping. The relative significance of the primary source of peritoneal tuberculosis may be seen from the statement by Pribram that of one hundred and sixty-five cases of peritoneal tuberculosis examined after death, eighty-seven were attributed to intestinal tuberculosis, sixty-five to pulmonary and glandular, eight to tubal and uterine, and five to osseous tuberculosis. In one hundred and seven autopsies of tuberculosis in which the peritoneum was affected, the lungs were simultaneously diseased in ninety-nine cases, the pleuræ in sixty, the intestines in eighty, the retroperitoneal glands in forty-four, the spleen in forty, the kidneys in thirty-eight, the liver and suprarenal capsules each in sixty-six. According to Osler, tubercular peritonitis is most common between the ages of twenty and forty years, and is rare in old age. Although pulmonary tuberculosis affects either sex with equal frequency, the statement of König, that of one hundred and thirty-one cases of laparotomy in which tubercular peritonitis was found ninety-two per cent. were females, is significant that the distinction was not critically drawn between tubercular and chronic granular peritonitis. On the other hand, it is not to be denied that the granules which have been seen in chronic granular peritonitis may have been fibrous tubercles whose specific characteristics had become lost. The often reported concurrence of fibrous hepatitis and tubercular peritonitis is of interest as suggesting that a non-inflammatory pathological process involving the peritoneum may favor the invasion of the latter by the bacilli of tuberculosis from elsewhere in the body and result in the production of their characteristic disturbances.

MORBID ANATOMY.—The lesions of tubercular peritonitis, like those of the non-tubercular variety, are diffused or circumscribed, and are represented by the association of tubercles and exudation. The appearances vary in accordance with the predominance of the one or the other, and are influenced by the extent and duration of the process. The diagnosis to be beyond question demands the appreciation of more than the gross appearances of the tubercle. These are sufficiently indicated by the meaning of tubercle,—namely, little knob or node. Similar nodules, even miliary in size, occur without other evidence of a tubercular nature in chronic peritonitis. In doubtful cases, therefore, the diagnosis of the nature of the tubercle demands a microscopical examination with reference to structure and to the presence of characteristic bacilli. In the absence of the latter, inoculation of the suspected material is necessary before the absolute diagnosis of tuberculosis can be made.

A circumscribed growth of tubercles is often unexpectedly found when the abdomen is opened either by the surgeon or at an autopsy. It may be limited to the peritoneum overlying a tuberculous ulcer of the intestine, or may be found in the vicinity of a tuberculous Fallopian tube or in the peritoneal covering of the diaphragm in tubercular pleurisy or pericarditis. Such localized tubercular peritonitis is of little clinical significance unless active or sufficiently extensive to result in the production of tumors, the characteristics of which are soon to be stated.

If tubercular peritonitis is part of a general acute miliary tuberculosis, the peritoneum may be smooth, shining, transparent, at the most injected, but is studded with gray, glistening, translucent granules smaller than a pin's head, and projecting slightly above the surface. There may be but a few ounces of clear yellow fluid in the peritoneal cavity.

When tubercular peritonitis becomes the principal tubercular lesion in the body, the alterations of the peritoneum are far more extreme. The peritoneum is thickened and opaque, sometimes pigmented from extravasated blood. The tubercles are opaque, grayish-white, or yellow, and tend to become clustered into patches and nodules. Serum, fibrin, and sometimes pus are found in the exudation, and red blood-corpuscles may also be present. As a rule, the more abundant the serum the less the fibrin. The former may lie free in the peritoneal cavity or be enclosed within fibrinous false membranes. Fibrin is present as a layer covering the peritoneum and often concealing the tubercles lying within the latter, or it forms bands or cords uniting different portions of the peritoneum. Fibrous adhesions and fibrous thickenings of the peritoneum, both of which may contain tubercles, eventually result. Owing to the contraction of this fibrous tissue and the abundant formation of tubercles, tumor-like masses may be produced. The omentum may be shrivelled into a dense sausage-shaped mass, or adherent coils of small intestine may form a globular mass closely attached to the spine by the contracted mesentery. Tumors composed of serum, fibrin, and tubercles may be formed between adherent peritoneal surfaces, especially in the pelvis and in the iliac fossæ. The adjacent intestine may become perforated from without, perhaps in several places, and more or less of the softened and disintegrated exudation be discharged into the intestine. The abdominal wall may also be perforated, and the contents of the intestine escape through such openings. If the tumor is in the right iliac fossa, the vermiform appendix may lie at the bottom of such a sinus in the abdominal wall, and its tip, if non-adherent, be exposed by slight muscular exertion, as in coughing. If there are extensive adhesions and little serous exudation, the peritoneal cavity may become largely obliterated and the abdominal wall retracted.

Symptoms.—The onset of a tubercular peritonitis is often so gradual, the symptoms so latent, and the course so prolonged, that the disease may be unsuspected until enlargement of the abdomen is apparent. Even then the tubercular nature of the process may be recognized only after the abdomen has been opened for the removal of a supposed abdominal, usually ovarian, tumor. In other cases severe symptoms may develop suddenly and progress rapidly, and directly call attention to the probable tuberculous nature of the affection. A frequent early symptom is a sensation in the abdomen, either localized or diffused, rather of discomfort than of pain. Severe pain may be an early symptom, in which case it is associated with tenderness, and may be prolonged, with intermissions of comparative comfort.

Irregular elevation of temperature is an important symptom, although there may be prolonged intervals of normal, perhaps subnormal, temperature. The attacks of abdominal pain are likely to be accompanied with elevations of temperature, and both pain and fever are often associated with alterations in the size of the abdomen.

The enlargement of the abdomen is due, at the outset, to the exudation of liquid, which in the course of time may become so abundant as to produce considerable distention. The intestines may float freely, and a wave be transmitted throughout the abdomen as in ascites. The enlargement is to be partly explained by the presence of gas in the intestines, variations in the quantity of which largely account for temporary modifications in the size of the abdomen. With the formation of fibrinous and eventually fibrous adhesions the liquid exudation is usually diminished and often encapsulated, thus causing the circumscribed tumors already mentioned, which, according to the predominance of tubercles and exudation or of intestinal gas, are flat on percussion or present a modified, perhaps tympanitic, resonance. The fixedness and elasticity as well as the resonance of these tumors give satisfactory evidence of their nature and origin. They are thus not to be confounded with the resistant, sausage-shaped masses of thickened and shrivelled omentum or with the indurated products of a tubercular peritonitis to be felt on pelvic examination.

As the disease progresses, digestive disturbances become especially conspicuous. There are loss of appetite, nausea, perhaps vomiting, and diarrhœa or constipation. The last may be so severe as to lead to a diagnosis of intestinal obstruction. Ultimately loss of flesh and strength becomes conspicuous, and is especially marked in those cases in which intestinal fistulæ have arisen, or in which extensive tuberculosis exists elsewhere, especially in the lungs or the intestine.

DIAGNOSIS.—The frequency of a gradual onset of the disease and the latency of the symptoms have often made an early diagnosis difficult. This is conspicuously shown by the fact that in the majority of cases in which tubercular peritonitis has been discovered at a laparotomy a previous diagnosis of some other affection, particularly of an abdominal tumor or an ovarian cystoma, has been made. Ascites, chronic peritonitis with abundant effusion, and abdominal tumors are especially to be differentiated. For this purpose an appreciation of the etiology of the several affections is of importance.

Ascites is simulated when there is abundant free fluid in the peritoneal cavity. In tubercular peritonitis, however, there may be a certain degree of abdominal pain and tenderness, more or less fever, even if slight, and frequent indurations, while in ascites jaundice or gastro-intestinal hemorrhages and enlargement of the spleen are to be expected.

Most cases of subacute and chronic general peritonitis of non-traumatic origin, independent of antecedent acute peritonitis and cancer or sarcoma, are possibly of tubercular origin. Evidence of pre-existing or associated tuberculosis of the lungs, intestine, genitals, kidney, lymphatic glands, or bones, also an associated pleuritic effusion, favors the tubercular nature of the peritonitis. If such evidence is lacking, a differential diagnosis between chronic tubercular peritonitis and chronic peritonitis with abundant serous effusion may be as impossible as is the differential diagnosis between chronic granular peritonitis and miliary tubercular peritonitis when based on the gross appearances alone after the abdomen has been opened, either during life or after death. Fortunately, this distinction is of little practical importance in treatment.

A parovarian or a unilocular ovarian cyst may be differentiated with difficulty. The fluid from the former is sufficiently characteristic, while the growth of the latter is likely to be slower, with less disturbance and without emaciation and debility. Malignant disease of the peritoneum may closely resemble the graver forms of tubercular peritonitis, especially when tumor-like masses are present, and is to be differentiated by its more rapid progress and by the presence of probable malignant disease elsewhere, especially of some abdominal organ.

Prognosis.—The mortality in cases of tubercular peritonitis has until recently been considered extremely high, most cases being thought to prove fatal within a year after the recognition of the disease. So excessive a death-rate was largely attributed to the existence of severe tuberculous disease elsewhere, and to the complications, especially the intestinal fistulæ, resulting from the abdominal disease. At the same time, cases have been recorded in which the exudation of supposed tubercular peritonitis has disappeared. In one instance the patient was alive three years later; in another the patient died of general tuberculosis after two years; in a third, as reported by Pribram, tubercles and characteristic bacilli were found at the end of a year and a half when laparotomy was performed for the removal of a parovarian cyst. Of late years frequent recoveries from assumed or assured tubercular peritonitis have been announced. Casinari states that of eight hundred and forty cases which have been reported two hundred and eight died, giving a mortality of about twenty-four per cent. The diagnosis of tubercular peritonitis, to be beyond criticism, demands the recognition with the microscope of Koch's bacilli or evidence of their presence by inoculation experiments. Such information has been possible only since 1882, and has been furnished in comparatively few of the numerous reported instances of tubercular peritonitis cured by laparotomy. But the few indubitable cases, in addition to the experimental evidence furnished by Stchegoleff, make it evident that under suitable conditions the prognosis of tubercular peritonitis may be favorable. It is generally recognized that the variety of tubercular peritonitis which presents the least danger is that with abundant serous exudation and latent symptoms.

TUBERCULOSIS OF THE DUCTLESS GLANDS.

Tuberculosis of the Spleen.—The spleen, like the liver, is a frequent seat of tubercles, the bacilli being brought by means of the blood or entering from the peritoneal cavity. In acute miliary tuberculosis thousands of tubercles may be present, which are at times with difficulty distinguished from the Malpighian corpuscles. The spleen is enlarged, moderately firm, of dark-red color. On section the pulp is increased and studded with innumerable grayish-white specks slightly projecting above the surface, as if the section were finely sanded.

Chronic tuberculosis of the spleen occurs as cheesy nodules more or less rounded and varying in size from that of a grape-seed to that of a hazel-nut, the additional presence of miliary tubercles at the periphery indicating a progressing stage. Fibrous adhesions are frequent between the spleen and the abdominal wall when the solitary tubercles lie near the surface of the spleen, and tubercles may be present in the adhesions. The demonstrable enlargement of the organ in acute miliary tuberculosis is so constant as to be a valuable diagnostic sign of this affection.

Tuberculosis of the Lymph-Glands.—The lymph-glands are constantly diseased in tuberculosis. They are usually affected in virtue of tubercular changes in the regions from which they receive lymph, although they may become tuberculous without evidence of a pathological process in such regions. When one set of glands is affected, tuberculosis of other lymphatic glands is likely to follow. The glands oftenest diseased are the cervical, bronchial, and mesenteric; but those in the axillæ and the groins are not exempt. The occurrence of tuberculosis of the cervical glands as a sequence of chronic cutaneous inflammations of the face and scalp, of naso-pharyngeal catarrhs, and of carious teeth, suggests that glands inflamed from whatever cause are prone to become infected with the bacilli. In like manner, the presence of bronchial and mesenteric tuberculous glands without evidence of pulmonary or intestinal tuberculosis indicates that irritations of the respiratory and intestinal tracts may be followed by the admission of the bacilli to the neighboring glands without the production of surface lesions. Local tuberculosis of any part of the body is likely to be speedily followed by tuberculosis of the nearest group of lymphatic glands.

The bacilli of tuberculosis when lodged in a lymph-gland cause a productive inflammation, a lymphadenitis. Two varieties of this result occur,—the one, tubercular lymphadenitis, conspicuously characterized by the presence of visible and structural tubercles containing the bacilli, and the other, cheesy lymphadenitis, chiefly manifested by extensive necrosis of the gland with or without visible tubercles. The failure to discover with the microscope the bacilli in such glands does not contradict their tubercular nature, since the inoculation of guinea-pigs with the cheesy material is usually followed by the production of tuberculosis in them.

In tubercular lymphadenitis the gland is moderately enlarged by an increase in the number of its cells. Miliary tubercles appear, increase in number, and become confluent and cheesy. Multiple nodules may thus form, and the gland eventually grow cheesy throughout. The individual tubercles may also undergo a fibrous transformation, and the entire gland become indurated by the increased formation and contraction of fibrous tissue.

In cheesy lymphadenitis the glands are swollen, perhaps to the size of a pigeon's egg, and on section are of a reddish-gray color and somewhat translucent. The enlargement is due to an increase in the number of the large and small lymphocytes, and giant cells may form ; but miliary tubercles are lacking, the process being one rather of diffuse than of circumscribed inflammation. A necrosis of the cells takes place, and the enlarged gland in the course of time is transformed into a homogeneous cheesy mass, whilst softening or calcification may subsequently occur. In the former the evacuation of the cheesy detritus takes place through the skin or into a neighboring cavity or hollow organ or into the blood-vessels or lymphatics. The discharge through the skin produces fistulæ with tuberculous walls, and if the bronchi are perforated a pulmonary tuberculosis results. Evacuation into the intestine may give rise to intestinal tuberculosis, while the passage of the contents into a serous cavity results in a pericarditis, pleurisy, or peritonitis. The escape of the softened cheesy material into a blood-vessel or into a large lymphatic, as the thoracic duct, is a most important cause of general tuberculosis, and such a result offers a direct demonstration of the tuberculous nature of the softened lymph-gland. Calcification of the cheesy lymph-gland represents an arrest of the process, the lime salts being deposited in the cheesy material of the previously enlarged gland, which is also fibrous and atrophied.

SYMPTOMATOLOGY.—Tuberculous adenitis is most common in children, but may be manifested at any time in life when a local tuberculosis in the affected region occurs. The symptoms vary in accordance with the number of glands involved and the rapidity of the infection. Osler mentions a case of general tubercular lymphadenitis, with a continued elevation of temperature extending throughout a year, in which enlarged and cheesy glands were the sole significant lesions. As a rule, the enlarged lymph-glands produce but little local disturbance. They remain quiescent, or soften and are evacuated, the fistulæ also often giving little or no trouble. Persons with tuberculous glands are likely to suffer from catarrhal and cutaneous affections and from digestive disturbances. They are apt to be anæmic and debilitated, especially early in life, but, except in case of febrile disturbance, are comparatively free from discomfort. The deformity is usually more distressing than the disease.

DIAGNOSIS.—Enlarged lymph-glands are to be recognized as tuberculous only when the bacilli of tuberculosis are found in them or when inoculation from them produces tuberculosis in animals. The tuberculous nature of the enlarged lymph-gland is to be inferred when evidence of a local tuberculosis is found in the vicinity, as tuberculous ulcers of the skin or of the pharynx, or when the bacilli of tuberculosis are found in pus from the middle ear or from carious bone or teeth. The tubercular nature of visible enlarged glands may also be inferred if evidence of tuberculosis is found elsewhere, as in the lungs, intestine, serous cavities, or uro-genital apparatus.

PROGNOSIS.—The presence of tuberculous glands is always a source of anxiety, from the liability of the extension of the infection from gland to gland, or to some important organ, or throughout the body. Glandular tuberculosis, when limited, may be recovered from, either as a result of treatment or in consequence of the softening and evacuation of the tuberculous gland or of its induration and calcification.

Tuberculosis of the Thymus and Thyroid Glands.—Recorded instances of tuberculosis of the thymus gland are very rare. According to Jacobi, who reports three cases of general tuberculosis in which the thymus was diseased, there was but one of isolated primary tuberculosis, that of Demme. Miliary tubercles alone may be present, also cheesy nodules with miliary tubercles at the periphery.

The *thyroid gland* may also be the seat of miliary or nodular tuberculosis, in both instances associated with the presence of tuberculosis elsewhere. There are no known symptoms resulting from tuberculosis localized in the thymus or the thyroid.

Tuberculosis of the Suprarenal Capsules.—This affection is usually chronic, and may exist alone, although generally associated with tuberculosis of other organs. Its chief clinical interest is due to its frequent presence in Addison's disease. (See page 41.)

TUBERCULOSIS OF THE VASCULAR SYSTEM.

Tuberculosis of the Heart and Blood-Vessels.—Miliary tubercles as part of an acute miliary tuberculosis are rarely found in the heart, but pericardial tuberculosis is often continued into the myocardium, in which cheesy patches and nodules may thus be produced. Gummata of the myocardium have repeatedly been mistaken for cheesy tubercles, in virtue of similar gross appearances. The bacilli of tuberculosis have been found in the vegetations of acute endocarditis in cases of chronic pulmonary tuberculosis. It is not unlikely in such cases that the bacilli lodge in vegetations due to other causes.

The arteries are frequently invaded by the bacilli from tubercular processes in the vicinity, and miliary tubercles in the adventitia of the arteries of the pia mater are a constant feature in tubercular meningitis. Tubercles are also to be found projecting from the intima of the pulmonary artery and even from that of the aorta in cases of chronic tuberculosis, there being no tubercular lesions in the vicinity. Mallory has observed the bacilli in the base of aortic ulcers and between the lamellæ of the elastic coat. It is probable that the tubercles of the intima of arteries become necrotic and softened and thus aid in the dissemination of bacilli. Tuberculosis of the veins also may occur, the bacilli being either transferred from the immediate vicinity when a tubercular inflammatory process reaches the wall of the vein, or entering directly from the blood, the latter probably being the source of the miliary tubercles which project from the intima in general tuberculosis. The relation of such venous tubercles to general tubercular infection is the same as in the case of arteries, the admission of the bacilli into the veins being the more frequent.

TUBERCULOSIS OF THE BRAIN AND SPINAL CORD.

Cerebral tubercles as distinguished from meningeal tubercles occur in the form of cheesy nodules, single or many, varying in size from that of a cherry-stone to that of a walnut. The larger, solitary tubercles more often occur in the cerebellum, pons, and cerebral peduncles. The multiple nodules are usually intimately connected with the pia mater of the convexities, and are essentially a globular variety of chronic meningeal tuberculosis, although the more frequent manifestations of the latter are cheesy plates, at the base or upon the convexities, oftener in the former situation. The presence of miliary tubercles at the periphery or in the vicinity of such cheesy formations, or the discovery in the latter of the characteristic bacilli, is indicative of the nature of the lesions, whose gross appearance often suggests that of sarcoma or gumma. This difficulty of diagnosis is especially marked in the case of the solitary tubercle, which may increase in size periodically, with intervals of quiescence, miliary tubercles being present at the periphery during the stage of growth and being absent at a later period. Cerebral tubercles are more frequent in children than in adults, and are usually associated with tubercles elsewhere. They are of probably hæmatogenous origin, except in those cases in which the infection is extended from a chronic tuberculosis in the immediate vicinity, as in nasal or auditory tuberculosis.

Tuberculosis of the spinal cord also occurs in the form of cheesy nodules, which may long remain latent, although gradually increasing in size until significant symptoms are present. The clinical characteristics of tuberculosis of the brain and spinal cord are essentially those of tumors of these organs, and are to be found in the consideration of the latter subject.

TUBERCULOSIS OF THE BONES AND JOINTS.

Tuberculosis of the bones throughout the body is a very frequent accompaniment of acute miliary tuberculosis, the tubercles being present in the marrow as miliary granules, which often, though with difficulty, are recognized without the aid of the microscope. More important is the occurrence of localized chronic tuberculosis, *osteomyelitis tuberculosa*, which takes place particularly in the vicinity of joints, especially of the lower extremities. The frequent association with tubercular arthritis suggests that the bacilli may enter the bone-marrow from the joint, although the absence of disease of the joint in other cases is evidence that the infection of the bone-marrow likewise takes place through the circulation. In chronic local tuberculosis of the bones extensive hyperplasia and necrosis of the marrow are present. The trabeculæ become disintegrated, fragments of bone of various size are sequestered, and cavities arise eventually communicating with the surface and leading to the formation of fistulæ. With the occurrence of chronic tubercular osteomyelitis the periosteal growth of bone is often increased and aids in the retention of sequestra in the interior of the shaft. Such bone tuberculosis forms a frequent basis for cranial and vertebral caries and for the carious and club-shaped bones of the extremities.

Tuberculosis of the joints may result from the entrance of bacilli into the joint from the synovial membrane, or may be due to the extension to the joint of a tubercular osteomyelitis. Large and small joints may be affected, especially those of the lower extremities. The synovial membrane is thickened, reddish-gray, and translucent, and contains minute gray or yellow miliary tubercles. As the disease progresses and the tubercles become caseous and softened, concurrent destruction and proliferation of the synovial membrane take place. The ligaments and the surrounding tissue are thickened and fibrous, tuberculous fistulæ extend from the joint towards the skin, and the familiar white swelling results. Destruction of the cartilage and adjacent bone may occur, and sinuses are formed which lead, often through spongy bone, towards and into the diseased joint. The articular cavity may contain much or little exudation, which is serous, fibrinous, or purulent. The tuberculous nature of osteomyelitis and arthritis is rendered positive by the discovery of the typical bacilli in the exudation or in the diseased parts ; but the clinical history of the cases suggests that all are not due to tuberculosis, and the bacilli have repeatedly been sought for in vain.

Tuberculosis both of bones and of joints is more frequently found in the young, especially in children, and its further consideration belongs to surgery.

SCROFULA.

The intimacy of relation between scrofula and tuberculosis is such that many writers regard them as absolutely identical ; but scrofula as distinguished from tuberculosis is rather indicative of a state of the tissues and their relation to nutritive processes than of an infectious disease. The term was originally invented on account of the resemblance of the swollen neck of affected individuals to that of swine, *scrofa*. Such swelling was eventually found to be chiefly due to enlargement of the lymphatic glands or to an increase in the size of the thyroid gland. The enlargement of the lymphatic glands proved to be due to a variety of causes, but cheesy conditions were most frequently observed. The cheesy glands were called scrofulous, and similar cheesy appearances found elsewhere in the body also were designated scrofulous, hence scrofulous kidney and scrofulous testicle, and were regarded as the manifestations of a like condition of the body, the scrofulous diathesis or constitution. The enlarged thyroid gland received the term struma, and English writers were accustomed to use strumous and scrofulous as synonymous. As it appeared, especially in children, that cutaneous eruptions, obstinate catarrhs, and inflamed bones and joints were frequently associated with enlarged cheesy glands, such affections were regarded as scrofulous, and thus arose the terms scrofulous lichen, scrofulous ulcers, scrofulous ophthalmia, scrofulous ozæna, scrofulous bones and joints.

From the frequent association of the cheesy alterations with tubercles of various size and appearance the cheesy conditions eventually were regarded as a manifestation of tuberculosis. Thus scrofula and tuberculosis became identified. Virchow, however. showed that the cheesy appearances might be the result of various pathological processes, and Koch demonstrated that the presence of the bacillus of tuberculosis is the essential characteristic of tuberculous material. Although much cheesy material contains this bacillus or produces tubercle when inoculated, exceptions frequently arise. By those who would identify scrofula and tuberculosis it is held that positive results follow inoculation only when a certain number of bacilli are present, and the older the cheesy material the less numerous the bacilli ; but it is maintained, on the other hand, that more bacilli are at times to be found in old than in fresh cheesy material. It is also argued that the negative results of inoculation are attributable to an enfeebled virulence of the bacilli,—a view which receives support from the experiments of Arloing, who found that rabbits were insusceptible to the inoculation of cheesy material which infected guinea-pigs, although the virus from such guinea-pigs, passed through successive series, became so increased in virulence as to produce tuberculosis in the rabbit. It is furthermore maintained that the scrofulous constitution, if not the scrofulous lesion, is due to the early lodgement in the tissue of the bacilli of tuberculosis. In favor of this view are the discovery of the bacilli in fœtal blood and the production of tuberculosis by the inoculation of blood from the umbilical vein of the infants of tuberculous mothers. Cheesy nodules containing bacilli have likewise been found in the viscera of an infant so young as to make probable the prenatal origin of these nodules.

Since cheesy degeneration is not due to tuberculosis alone, and since the bacilli of tuberculosis are not present in all cheesy material, it is obvious that caseation is not absolute evidence of tuberculosis. In certain so-called scrofulous eruptions of the skin, lichen, for example, and scrofulous catarrhs, notably conjunctivitis, the bacilli of tuberculosis are absent: hence so-called scrofulous inflammations are not necessarily tuberculous. The question of the identity of scrofula and tuberculosis is, therefore, to be regarded as still open. There is to be recognized a condition of the body which Virchow has defined as a feeble power of resistance of the tissues and a persistence of the disturbances in them. The pathological processes and products are to be found in the skin, mucous membranes, bones, and joints, and are usually associated with chronic enlargement of the lymph-glands. These pathological products are prone to become invaded by the bacillus of tuberculosis, which finds in them suitable conditions for its growth and dissemination. The term scrofula is thus to be applied to those individuals who through inheritance or by exposure to faulty hygienic surroundings acquire such a vulnerability of the tissues that trivial causes produce persistent lesions, and in whom exposure to tubercular infection is frequently if not always followed by the harboring and propagation of its bacillus. The scrofulous person thus is one who is prone to become tuberculous in virtue of vulnerable tissues, but who is not tuberculous until infected by the bacillus of tuberculosis.

ETIOLOGY.—This vulnerability of tissues is of both congenital and acquired origin. Congenital causes are to be found in scrofulous or tuberculous parents, or in those enfeebled by severe chronic diseases, as syphilis, cancer, or nephritis, or by the abuse of alcohol. Early and late marriages and those of near blood relations have been considered important in etiology. Causes of acquired vulnerability are bad air, poor food, insanitary dwellings, the crowding together of children in institutions, and insufficient attention to hygiene. Although scrofula is more common among the poor, it is not limited to them. The manifestations of the condition often appear in early infancy, but usually become pronounced in childhood.

MORBID ANATOMY.—The products of chronic inflammation of the skin and mucous membranes, of the lymphatic glands, and of the bones and joints are the lesions likely to be present in scrofula. Enlargement of the lymph-glands, especially when associated with cheesy degeneration, has always been regarded as the essential anatomical feature of scrofula. It is obvious that this view is not absolutely correct, and in the light of our present knowledge the scrofulous as distinguished from the tuberculous lymph-glands, whether caseation is present or absent, are those in which the bacilli are absent, as indicated by the results of inoculation as well as by the microscopical examination. Practically, when caseation is present the scrofulous glands have become tuberculous. The scrofulous lesions of bones are especially to be found in the vertebræ and the long bones of the extremities. They are manifested by an osteomyelitis which progresses with destruction of the bone, the abscesses extending towards distant parts, as in the prevertebral abscess of spinal caries. The evacuation of the pus leads to the formation of fistulæ and sinuses in the vicinity of or at some distance from the diseased bone. In like manner a scrofulous inflammation of the joint is represented by a chronic thickening of the tissues, destruction of the cartilage, and fungous granulations. In many cases, however, of chronic osteomyelitis and arthritis caseation of the inflammatory product occurs, and tubercles and bacilli are present: hence the distinction between a scrofulous and a tuberculous joint depends also upon the results of the search for the bacilli. The recovery from such inflamed joints as well as from a scrofulous osteomyelitis offers suggestive evidence of the absence of bacilli, as the persistent progress of destruction implies their presence. In the latter event both the scrofulous joint and the carious bone may be considered to have become probably tuberculous.

SYMPTOMATOLOGY.—Among the earliest symptoms suggestive of scrofula are cutaneous inflammations, especially lichen, which is characterized by clusters of miliary, red or reddish-yellow papules in the vicinity of the hair-follicles, especially upon the chest and the abdomen. The rash may persist even for years, new papules being formed as those earlier formed disappear. Chronic eczema, both moist and squamous, is frequent upon the scalp, face, and ears, and the pustules of impetigo are to be seen upon the face and extremities. Lupus and scrofuloderma, formerly regarded as manifestations of scrofula, are known to be tubercular lesions. Subcutaneous abscesses often occur, either as complications of cutaneous inflammations or of independent origin, and are prone to remain quiescent for a long time, the pus being eventually absorbed or evacuated.

Catarrh of the mucous membranes is common and often persistent. Conjunctival catarrh tends to become granular, and is often associated with eczema of the lids, inflammation and opacity of the cornea, and pannus. The nasal catarrh is obstinate, and produces copious secretion, which either macerates the skin of the nostrils and the upper lip, causing redness and swelling and often eczema of the lip, or forms crusts at the nasal openings. Chronic or recurrent swelling of the tonsils and enlargement of the pharyngeal lymph-follicles are conspicuous features. The pharyngeal catarrh extends to the middle ear, often resulting in perforation of the tympanum and extension to the mastoid cells, with continuous or recurrent discharge from the meatus which frequently continues into adult life. Catarrh of the respiratory mucous membrane leads to repeated attacks of laryngitis, tracheitis, and bronchitis, while the catarrhal infectious diseases, as measles, influenza, and whooping-cough, tend to the production of broncho-pneumonia. Catarrh of the stomach and intestines is also of frequent occurrence.

Swelling of the lymph-glands is dependent upon cutaneous or catarrhal inflammations or affections of the bones and joints: hence the glands affected are those nearest the seat of the inflammation, and frequently several are implicated. The glands oftenest concerned are those beneath the jaw and in the neck. The axillary and inguinal glands are enlarged when inflammations of the trunk or extremities occur, and the bronchial and mesenteric glands become inflamed in consequence of catarrh of the bronchi and intestine. The glands at first form lumps of the size of beans, but may be as large as walnuts. They are neither painful nor tender, and may persist for years without undergoing further changes. With a recurrence or continuance of the affections of the skin or mucous membranes they further enlarge. They may subsequently shrink, but are likely to grow until they form tumors, sometimes of considerable size, which become cheesy, calcified, or softened. When the softened material is evacuated, sinuses are produced, which heal with difficulty and usually with the formation of extensive scars. In such cases the scrofulous glands are tuberculous, but the quiescent glands are associated with no symptoms in virtue of which the presence of the bacilli of tuberculosis can be recognized.

Affections of the bones in scrofula are those of a chronic character tending towards caries. Although the most frequent cause of caries of the bone is tuberculosis, in a scrofulous person the infectious or traumatic causes may produce an osteomyelitis not to be distinguished from that due to tuberculosis except by the absence of the bacilli of tuberculosis. A carious bone in the scrofulous person is apt to become tuberculous even if it were not so from the outset. If the osteomyelitis arises in the vicinity of a joint, the joint frequently is secondarily involved. The inflamed joint of the scrofulous person is usually announced by vague pain, impaired mobility, and a gradually increasing swelling. Such joints are likely to become tuberculous, in which case the persistent œdema of the surrounding tissues is a suggestive symptom. The especial consideration of the osteitis and arthritis of scrofulous persons belongs to surgery.

Two types of scrofulous children are usually described, although it is generally considered that they are not sufficiently constant to be regarded as characteristic. The one is represented by a stunted growth, coarse, flabby, clammy skin, thick nose and lips, and a swollen abdomen. Mental and physical action are characterized by slowness and deliberation. Such children are prone to cutaneous and catarrhal affections. The other type includes the tall children, usually blondes, with small bones, thin, pale skin, and prominent veins. The eyes are large and expressive, and the cheeks readily flush. These children are lively both in mind and in body. Febrile disturbances are frequent in them, and they are especially likely to become tuberculous.

DIAGNOSIS.—The suggestion of scrofula is presented by children with the characteristics last mentioned, and especially when the etiological causes exist; the diagnosis of scrofula is to be made when the previously mentioned recurring or chronic affections of the skin, mucous membranes, lymph-glands, bones, and joints are present, and the bacilli of tuberculosis are absent. Some of the manifestations of congenital syphilis may be mistaken for those of scrofula, but the cutaneous manifestations of syphilis are more general, the tendency to ulceration is more frequent, the enlargement of the glands is less considerable, and the affections of the bones and joints are of early occurrence.

PROGNOSIS.—Recovery from scrofulous lesions, even from those of bone, is frequent, although the tendency to scrofulous inflammations usually persists till puberty. The especial danger is the liability to tubercular infection, which often occurs before puberty and may manifest itself at a later period in life. The prognosis in the individual case becomes the more serious the more permanent the nature of the lesions: hence enlarged glands, caries of the bone, and chronic arthritis have a graver prognosis than cutaneous eruptions or catarrh. That the more serious lesions are not hopeless is evidenced by the numerous persons who have reached middle life despite deforming scars of the neck, curvature of the spine, and stiff joints originating during a scrofulous childhood.

PROPHYLAXIS.—The prophylaxis of tuberculosis may be either from the point of view of the individual already suffering or from that of the person free from the infection. In order to prevent himself from becoming a centre of infection, and also to diminish the possibility of reinfection of himself, the tuberculous person should destroy by burning or boiling all discharges from diseased parts, whether such parts be internal, as the lung or the bowels, or external, as open glands or joints. Local cleanliness is essential, and in phthisis it is important that the sputa be not swallowed, but expectorated and immediately destroyed. Portable spit-cups are in the market; or small pieces of rag may be used, put in a special receptacle, and finally burned.

The tuberculous subject should never sleep in the same bed with another person, and absolute cleanliness of the person should be enjoined; the occupied apartment should have a hard-wood floor, with mats instead of carpets, and should be thoroughly scrubbed at short intervals. If these precautions be observed, and if the apartment be at all times well ventilated, the risk to a healthy person involved in nursing a consumptive will be very slight. In a family, however, in which the tendency to tuberculosis is strong, this risk is appreciable.

From the point of view of the non-infected person with an hereditary tendency to the disease there are two desiderata: first, to increase the resistive power of the tissues; secondly, to avoid infection with bacilli. Of these the most important is the first mentioned, at least if the individual is to live within the confines of civilization, since the tubercle bacillus is so universally present as to make escape from it hopeless. At the same time it is important to avoid inoculation as far as possible, and the person who has a strong hereditary tendency to tuberculosis should shun unnecessary exposure to the contagium: thus, a physician should not take a resident position in a consumptive hospital; a nurse should decline tubercular patients. There can be no doubt that, especially in our large cities, there are houses and rooms in houses which are infected with the bacillus of tuberculosis. A person with lack of resistive power should never live in a room or even in a house which has been inhabited by a tubercular patient, unless such apartment or house has been cleansed and disinfected in a most thorough manner.

It has been demonstrated that the tubercle bacillus may be transmitted to the human being with animal food, yielded by tubercular animals. The danger of infection from tubercular milk is greater than from tubercular meat, evidently because before it is eaten meat is almost uniformly subjected to a sufficient temperature to kill the bacillus. The inspection of milk and of meat by municipal authorities in large cities ought to be, but is not, sufficiently rigorous to guarantee immunity; and precaution should be taken by the susceptible individual against tuberculous foods, especially to see that the milk habitually used is obtained from healthy cows.

The child with hereditarily feeble resistive powers should from the beginning be brought up with the purpose of developing the muscular and circulatory system and of obtaining that vitality which is given by continuous life in the open air. At the same time it must be carefully guarded from the various infectious diseases, especially such as measles, which have a tendency to provoke catarrhal inflammations, and it should be continually watched to prevent the development of mucous membrane catarrhs, which experience has shown have a pronounced tendency to aid in the development of tuberculosis. Obstruction of the nose and throat by malformations, adenoid glands, or enlarged tonsils should be promptly relieved by surgical or other treatment. The clothing should be warm, woollen in winter: it is the height of folly to attempt to harden such a child by insufficient clothing and exposure. Habitual cold bathing is excellent. The food should be abundant, simple, nutritious, largely but not altogether farinaceous, with a full supply of milk, and, if possible, of fats. Almost invariably the child can be brought to like cod-liver oil, and advantage is often gained by making this fat an habitual article of diet in cold weather.

In selecting a climate the question of degree of temperature is a minor one; that of moisture and equability of temperature is dominant. A dry equable climate is always preferable. Dry cold is not dangerous, and is, indeed, preferable to enervating warmth.

Prolonged life in high mountainous regions during childhood, if associated with habits of exercise, has a distinct tendency to develop the lungs and heart, and is very beneficial. When there is any special failure in the chest development, gymnastic exercises directed to the development of this part of the body may be very useful ; but no in-door exercise will take the place of out-door work ; and it is not probable that any artificial system is better than or even equal to the natural gymnastics of an active child. Running up and down mountains, herding goats and sheep, following the chase, fishing mountain streams,—these are the methods of restoring vitality to an exhausted family stock. It is affirmed by recognized authorities that a long-continued life at high altitudes so greatly increases the respiratory movements as to cause dilatation of the air-vesicles and a permanent increase in the size of the chest, which is a great disadvantage when such persons attempt to live at the sea-level. If this be correct, it constitutes no reason against bringing up the hereditarily feeble upon the mountains, but is a strong one for keeping them there during their after adult life. The out-door life is the one dominant feature: this must be insisted upon when the individual is forced to take what he can get, not being able to get that which is best. Thus, a seafaring life entered early, though less beneficial than a mountain life, is much better than a city life, provided its hardships are not too great. If the subject is forced to live in the city and earn his daily bread, the safest occupation is probably that of a car-driver or a motorman, with its continual open-air exposure and partial protection from the elements.

The habitual use of any drug is probably more injurious than beneficial, but, when there is any excessive susceptibility of the mucous membrane, continuous courses, spreading over months, of minute doses of arsenic are certainly worthy of trial. The arsenic-eaters of the Styrian Alps, if accounts can be trusted, are remarkable even among mountaineers for their pugnacity, their endurance, and their long-windedness.

TREATMENT.—*Acute general tuberculosis* always ends fatally, and, as its course cannot be distinctly modified by any known method of treatment, the effort of the physician should be especially directed to the obtaining of euthanasia. All possible moral support should be given to the patient. Disagreeable drugs and disturbing agencies should be sedulously avoided unless the relief of pain by them would be distinctly greater than the suffering produced by them : thus, a blister by relieving a pleuritic pain may be a great relief. Morphine and other narcotics should be used without hesitation to relieve suffering. The general course of treatment must be symptomatic and palliative.

In the treatment of *chronic tuberculosis* it is essential that the physician free his mind from the thought that the disease is necessarily fatal, so that he may be able to impart hopefulness to the patient under his care,—since to the ordinary individual there is no more depressing agent than the belief in oncoming death.

The first indication for treatment is removal of the infected part, when possible. In the case of glands and other parts which can be destroyed by the surgeon's knife, no time should be lost in dallying when once the diagnosis has been made clear. Unfortunately, in the larger proportion of cases excision of the affected part is impossible, so that the case must be treated entirely medically. The growth of surgery, however, has gradually widened the scope of surgical treatment. Thus, it is probable that tubercular peritonitis should be considered a local surgical disease.

Many attempts have been made to destroy colonies of bacillus by local or other medicinal treatment. In external tuberculosis the application of concentrated germicides or the free use of iodoform may be of service, but in internal tuberculosis these local agents are never of any value. Creosote, carbolic acid, and other drugs have been administered by the mouth, but when so given have no specific action on the tubercular bacillus. No known dose of any germicide which can be borne by the human system has any distinct power in even inhibiting the growth of the tubercle bacillus when once lodged in the body.

At one time extraordinary hopes were excited by the publications of Koch,—hopes which were, however, entirely beyond what ought to have been reasonably expected from Koch's own assertions. It is now known that the tuberculin of Koch was an impure toxin; or, in other words, that it was a poisonous agent produced during the growth of the tubercle bacillus, the cause of the hectic fever and many of the constitutional symptoms of phthisis. It was stated by Koch that the hypodermic injection of tuberculin in the tubercular patient would produce a febrile reaction, and at the same time would soften all tissues containing the tubercle and lead to the throwing off of both tissues and parasite. These assertions are correct, but the changes do not benefit the patient. If the tuberculin or tubercle-toxin be injected into the normal individual in the dose recommended by Koch, no distinct symptoms usually result; if, however, a certain amount of toxin be already in the tissues about the bacilli and in the blood, the addition of the amount of toxin used will be sufficient to bring about a hectic fever,—*i.e.*, febrile reaction,—and also to injure or to kill the tissue which is in immediate contact with the bacilli and already nearly saturated with toxin. The treatment, however, does not kill the bacilli, but simply liberates them by destroying the lung or other part in which they are situated. It puts the organisms in an excellent position for entrance into blood-vessels or lymphatics, and in this way has frequently led to a dissemination of the bacilli and to a consequent conversion of a localized into a generalized tuberculosis. It is possible that in the future a tubercular antitoxin may be produced, but, as tuberculosis is not a self-limited disease which produces an immunity, the prospects for success with its antitoxin do not seem brilliant.

As at present we have no known method of directly attacking the bacilli in internal tuberculosis, the indications in the treatment of the local chronic forms of the disease are—first, to increase the general nutrition and the resistive power of the individual; second, to reduce to a minimum the local irritation and changes produced by the bacillus at the seat of infection; third, to combat constitutional symptoms as they arise.

First Indication.—The method of meeting the first of the indications just spoken of must vary with the stage and condition of the tubercular patient. In the advanced disease absolute rest in the open air in a mild climate may constitute the chief measure which can be adopted with advantage, but in the following discussion it will be considered that the subject is in the earlier stages of the disease, with only a moderate amount of local disease in the lung. Under these circumstances high feeding, exercise, and life in the open air, with as much sunshine as possible, are the chief agencies with which the tendency to disease must be combated. The food should always be simple, thoroughly well cooked, palatable, the most nutritious and digestible that can be obtained, with a fair proportion of farinaceous articles, very little sugar, and a large amount of fats. It is a matter of the utmost importance, however, not to overfeed the patient,—that is, not to give more food than can be digested. Vegetable fats are useful, but are probably inferior to animal fats. Sweet oil is an excellent food, but cod-liver oil is preferable. In the selection of fats, as of other foods, the question of digestibility is dominant, and in the use of cod-liver oil it is essential to see that no more is taken than can be absorbed without difficulty, this being the only limit to the amount of oil to be ingested. Alcohol may be considered under these circumstances as a food, and is of the greatest value; taken in large quantity, however, it becomes a deadly poison, and in tuberculosis, as in all other forms of chronic disease, overshadowing the use of alcohol is always the danger of the formation of the alcoholic habit. The judgment of the physician must be applied to the individual case. When the digestion is strong, malt liquors are often preferable to spirits; but when there is any feebleness of the digestive organs, whiskey, brandy, or other distilled liquor is superior to either wines or malt liquors. Both for physical and for moral reasons, the alcoholic drink should always be given with food, and never in such amounts as to get its narcotic effect. An excellent combination is with cod-liver oil: thus, half a fluidounce each of the two substances may be given together after meals. It is essential that the patient be instructed never to use alcoholic liquor as a stimulant against the ever-recurring feeling of exhaustion.

In prescribing exercise the points to be borne in mind are that the exercise should be regular, day after day, with no paroxysms of excess to be followed by hours of exhaustion. It should never be violent, but should be continuous; it may be adapted to the individual needs in developing the chest or other part, but always should be, if possible, in the open air, and always should be kept within the strength of the patient: slight tire producing quietness and sleep is advantageous, excessive tire is very injurious. Continuous life in the open air is of the utmost importance; even in the advanced stages of phthisis life will be protracted and made more comfortable by having the bed of the patient from sunrise to sunset on a porch or in the open air, if the temperature be suitable.

The choice of climate for a patient is a most important part of the treatment. Usually the first decision to be made is whether the patient shall or shall not go away from home. The proper rule is, the milder and apparently more insignificant the local disease the more important the seeking out of a suitable climate, because the more is to be hoped from climatic treatment. If both apices are involved, the chances of life are so reduced that the physician is hardly justified in urging a change of habitation at every sacrifice. If with the involvement of each lung there be softening and formation of cavities, or if there be wide-spread tubercular infiltration with softening in one lung, change of climate can only be expected to give relief; and whether it should or should not be sought must depend upon the environment and circumstances of the patient. In such a case removal to a mild climate not too far from home is often preferable to choosing a more distant though more salubrious region.

In selecting for a patient the best possible region the individual peculiarities must be carefully studied. We have known tubercular patients whose only comfort and progress towards health were found in continuous life at sea. In the great majority of cases, however, a dry, equable climate, with abundant brilliant sunshine and pure air, constitutes the desideratum. The temperature and the character of the air must be such that the patient can be out of doors the whole time of daylight; and if the day and night can be spent practically out of doors the chances are much better. The climate in the Eastern United States which best suits the majority of cases is that of the Adirondacks; Florida is too damp, enervating, and malarious in most of its parts for the ordinary case of incipient phthisis; the high sand-ridge in the centre of the State is the best situated, but is probably inferior to the pine district of Southern Georgia. The high mountain districts of North Carolina rank next to the Adirondacks, and are even superior in those cases in which there is a tendency to feebleness in the heat-making function. If a patient feels the cold of the Adirondacks, Asheville is preferable; or the winters may be passed in Asheville and the summers in the Adirondacks. Southern California in some of its parts is undoubtedly a good climate, but we believe it to be inferior to the central arid tract in the United States commencing in San Antonio, Texas, and running north to Colorado and Arizona.

The height of the locality above the sea is a serious consideration to the consumptive. Although individual peculiarities here, as elsewhere, are important, the majority of tubercular patients will do best at a height of from three to six or even seven thousand feet above the sea. Among the modifying influences in regard to altitude is the tendency of the patient to hæmoptysis: when this exists a rapid ascent to a considerable height greatly increases the danger of bleeding. It is probable that this is due to the extension of the air-vesicles by the increased efforts at respiration produced by the altitude. The very cause of the benefit of the altitude becomes the source of danger. Hemorrhagic cases should therefore begin their life in the arid tract at a low elevation. Again, in the northern portion of this region, and especially in the higher elevations, the cold is severe in winter; on the other hand, in the San Antonio region the summer heat is excessive. It is plain that the first selection of a climate depends upon the character of the case and the season of the year. In the winter it is usually preferable to send the patient to the southern portion and have him travel northward with the season, so that the following winter can be spent in the high and colder districts.

It is of the utmost importance in a case of consumption to maintain the integrity of the digestive apparatus, and in selecting the place of abode the possibility of getting properly cooked food suitable to the individual case is of importance. Further, what may be termed extraneous considerations often enter into the problem of choice of locality: very frequently the opportunity for making a living, if not in the immediate present, in the near future, is of vital importance. In Texas, Southern California, Colorado, Wyoming, or other localities, ranch life or the cultivation of the soil in some way is open to many and gives work in the open air. In other cases the attractions of a city like Denver are dominant. The altitude above the sea of San Antonio is 650 feet, of Santa Fé, which may be looked upon as the next important stopping-place, 6840 feet, of Denver 5196 feet, of Colorado Springs 6000 feet. For a young man with pecuniary means we have no doubt that travel, hunting, and tent life in this region afford the best obtainable chance of recovery.

Professor C. B. Penrose, of the Medical Department of the University of Pennsylvania, spent two years roaming over this arid district, and states as the result of his personal observation that a greater proportion of cases get well in New Mexico than in any other Western Territory or State.

In the climatic treatment of phthisis it is important that the patient remain at the favored locality not for weeks or months, but for years.

The monotony of existence may sometimes advantageously be broken by travel in suitable localities. The question of the return of the apparently cured patient to his home is always a very serious one; in the majority of cases permanent residence in a proper climate is essential.

Second Indication.—Under the second indication—namely, the reduction to the minimum of the local irritation and changes produced by the bacteria—may be considered the use of—first, certain pulmonic gymnastics; second, counter-irritation; third, inhalations; fourth, internal medicaments.

In some cases of incipient phthisis the inhalation of compressed air in the so-called "pneumatic cabinet" gives good results, which are probably produced by a distention of the air-vesicles. It would seem that such inhalations are a very inferior substitute for high mountains, and should be used with great caution where there is a tendency to hemorrhage. It is further probable that as much good can be obtained by means of a simple device consisting of a glass or other tube a quarter of an inch in diameter and six or seven inches long, with a pin-hole made at one side; the patient inspires through the tube, places the finger so as to close the aperture, and forces the air through the pin-hole with all the expiratory force at his command, repeating the act many times over. Similar results may be obtained simply by the habit of frequent forced inspiration and expiration.

Counter-irritation is often of value in phthisis pulmonalis: it combats the local inflammation, but not the bacillus. In the earlier stages of the disease, when a small amount of tuberculosis in the apices produces much irritation with catarrhal pneumonic consolidation, the continuous application of croton oil over the upper chest may be very serviceable. Again, sinapisms or dry cups when there are congestive exacerbations, blisters in times of acute pleurisy, chloroform or other irritating embrocations when there are neuralgic or muscular pains, and other similar remedies or measures, may be very useful if judiciously employed. Even wet cupping or other local blood-letting may sometimes be advantageous, although it has a tendency to lessen the strength of the patient.

As inhalations and expectorant remedies are serviceable only by affecting the catarrh of phthisis, the remarks about the use of such remedies in the articles upon acute and chronic bronchitis are equally applicable to their employment in chronic pulmonic tuberculosis, and should be read in connection with the present subject. The checking or the supporting of the cough is governed by exactly the same principles in the two disorders. Morphine must, however, be used with especial care in phthisis, on account of its tendency to derange digestion and of the danger of the formation of the morphine habit: moreover, there is often a necessity of husbanding this remedy as a means of euthanasia in the later stages of the disease. Sometimes sedative expectorants are demanded by the patient to "loosen the phlegm;" it is necessary, however, to be guarded in their employment, lest the stomach be disturbed. No greater abomination exists than the multitudinous "syrups" which formerly more than at present were employed in this disease, much to the detriment of the patient's digestion and well-being. Creosote and guaiacol are valuable remedies, especially applicable to cases with very free expectoration; the latter may be used hypodermically. Sulphuretted hydrogen, terebene, and the various expectorant volatile oils are all useful when there is much chronic catarrh or softening. In dry chronic cases with little catarrhal tendency the long-continued use of smaller doses of arsenic (one or two drops of Fowler's solution three times a day) may be very advantageous.

Third Indication.—In any case of phthisis it is a matter of vital importance to study carefully the digestive organs, to adapt the food to the individual needs and condition of the patient, and to treat any symptoms of digestive failure very carefully as soon as they manifest themselves. A catarrhal state of the stomach and bowels must be at once met by appropriate remedies. (See articles on Gastric and Intestinal Inflammations.) Especially in the advanced stages, not only comfort but also distinct advantage is sometimes obtained by a system of forced feeding, consisting in a daily lavage of the stomach, followed by an injection into the stomach of quantities of nutritious, concentrated, easily digested foods. In some cases, in children more than in adults, free inunction with cod-liver oil seems to aid in the prevention of emaciation.

When *hæmoptysis* occurs, the patient should be put to bed, and forbidden to talk or to make any exertion whatever. Opium should be given in sufficient quantities to allay the nervous excitement and erethism which are almost invariably present, and to quiet cough if it should exist. At the time of the hemorrhage the taking of a large dessertspoonful or small tablespoonful of dry salt into the mouth is sometimes effective: it evidently acts reflexly by irritating the mucous membranes of the mouth and pharynx. If the seat of the bleeding is apical, ice or other form of cold may be applied locally. If the case be severe, extract of ergot should be given hypodermically (fifteen grains with ten minims of glycerin and twenty minims of water), as well as by the stomach, if the latter be retentive. As ergot is an entirely safe remedy, it should be exhibited in large doses. If there be call for immediate haste, two to four fluidrachms of the fluid extract may be exhibited at once. Afterwards the solid extract should be administered in capsules, as less apt to disturb the stomach; from ten to twenty grains (equivalent to five times the amount of the fluid extract) may be given every half-hour to every two hours, according to the degree of emergency. Not more than an ounce of the ergot should be taken in the twenty-four hours. Other efficacious remedies are gallic acid, ten grains every one to four hours, and oil of erigeron, ten minims every one to four hours. Very frequently good results are obtained by alternating these remedies with each other or with ergot. If there be excitement of the circulation and a full bounding pulse, aconite should be exhibited ; on the other hand, the use of stimulants, such as alcohol and digitalis, though in some cases imperative, must be very cautious, lest by increasing the force of the circulation they aggravate the hemorrhage. The older remedies, such as sulphuric acid, plumbic acetate, and oil of turpentine, are of very inferior rank to those above noted.

"*Night-sweats*" are in no way peculiar to phthisis, and may occur in the daytime when the patient is awake. The antihidrotic drugs which are of value are atropine, extract of ergot, agaricin, gallic acid, and sulphuric acid, named in the order of their power and general applicability. Of these, atropine has the disadvantage of producing great dryness of the throat and mouth and disturbance of the circulation if given in full dose. Unless there be a tendency to sweat during the day, it is best administered at bedtime, one-hundredth to one-sixtieth of a grain, often associated with some somnifacient ; if the tendency to sweating be present at all times, one-hundred-and-twentieth of a grain may be given every eight hours. Extract of ergot is incapable of doing harm to the patient except by disturbing the digestion, and if administered in five-grain capsules it rarely does this. Ten grains may be given every two to four hours during the day, its action not being immediate like that of atropine, but rather continuous. Agaricin is often very effective given (three to five grains) in capsules every six hours ; it has some tendency to irritate the intestinal tract, but otherwise seems to act purely as an antihidrotic, exerting no perceptible influence upon the system. The action of the antihidrotic drugs may sometimes be aided by bathing the patient with alcohol at bedtime : some of the older authorities recommend the use of baths of decoction of oak bark, but we have had no experience with them. As after the sweat the bodily temperature is frequently subnormal, care should be taken in changing the underclothing to see that the patient is rapidly well dried, that the fresh underclothing is warm, and that the whole process is gone through as quickly as possible, so as to avoid the danger of taking cold.

The combating of the hectic fever in phthisis is often hopeless. The moderate use of antipyrin, phenacetin, and allied antipyretic drugs is sometimes advantageous. If, however, moderate doses fail, it is not right to employ larger amounts, because of their tendency to produce depressing sweats. Quinine is rarely effective. When the temperature rises above 103° F. no hesitancy should be felt in the use of cold sponging or of the tepid bath.

The treatment of *intestinal tuberculosis* must have for its basis the general hygienic management and treatment already specified for chronic tuberculosis. Bismuth with carbolic acid, silver nitrate in capsules, creosote, chalk mixtures containing tannic acid, lead acetate, and other remedies suitable for the relief of intestinal catarrh and for the checking of diarrhœa when it becomes severe, must be used *pro re nata.* Opium is in many cases valuable. Externally, turpentine stupes, spice plasters, and all the milder counter-irritants may be employed upon occasion. Warm poultices or fomentations often give the most relief.

The medical treatment of *tubercular peritonitis* consists, first, in the employment of such hygienic measures as have already been spoken of as useful in tuberculosis; second, in the continuous use of mild counter-irritation, with careful feeding and the treatment of diarrhœa or constipation as may be needed. When diarrhœa exists, it may be considered to be the outcome of intestinal irritation or catarrh. Iodide of iron, especially in the case of children, is thought by some practitioners to be of great value. Most of the time the counter-irritation should be mild and continuous. Fomentations, poultices, and spice plasters may be used. Blisters are sometimes very effective in periods of exacerbations. Tapping in ascitic cases has been long practised, often with advantage. Simple aspiration often does good. The most recent method of treatment is by laparotomy.

Roers has analyzed three hundred and fifty-eight cases of chronic peritonitis in which laparotomy was performed. There were only twenty deaths from the operation. Two hundred and fifty-three cases were reported as cured; of these, fifty-three were still alive at the end of two years. It is not probable, however, that these cases were all tubercular, and there seems to be no light as to how far the residuum of fifty-three cases had originally been tubercular. Aldibert alleges thirty-nine cures in fifty cases in which tubercle was histologically or bacteriologically proved to be present. The accuracy of these figures can hardly go unchallenged, but the evidence is sufficient to warrant laparotomy in favorable cases of tubercular peritonitis. When there is tubercle elsewhere in the body, operative treatment is unjustifiable, and it is stated by Richardson, of Boston, to be inadvisable when a localized peritoneal infection has produced fistula with general matting together of the intestinal viscera. The form of operation should vary with the form of the disease; but for these surgical details the reader is referred to the paper by Richardson in the fourth volume of Dennis's "System of Surgery." Tuberculosis of the kidneys, bladder, prostate gland, seminal vesicles, testes, mammary glands, lymph-glands, bones, and joints, all may be considered to be surgical disorders, especially to be treated by radical local measures when possible. The constitutional management of such cases is similar to that of chronic internal tuberculosis.

In *lupus* or in *scrofuloderma*, when there is derangement of the general health, hygienic measures, with cod-liver oil and other nutritive stimulants, should be freely used. Especial value is attached by some to small doses of Lugol's solution (two drops *ter die*). Tuberculin has been largely used in lupus, but does not seem to have sustained the claim originally put forward for it. It would appear, however, that it does aid in removing the thickened and hardened tissues, and it may be employed from time to time, especially as a preliminary to severe local treatment. An effect similar to that of tuberculin has been alleged by H. von Hebra to be produced by thiosinamine. The local treatment is of especial value. The scraping off of the diseased tissues by means of the sharp dermal curette, as originally advised by Volkmann, may be done with local or general anæsthesia, the after-hemorrhage being controlled by pressure with absorbent cotton. After the scraping, dermatological authorities recommend various caustics: strong carbolic acid, fuming nitric acid, zinc chloride, chromic acid, pyrogallol (ten to fifty per cent. ointment), acid nitrate of mercury, and silver nitrate have each their supporters. Electrolysis has been used to a considerable extent with good results when the patches are small. Some specialists recommend that the local treatment be followed by tuberculin injections.

LEPROSY. LEPRA. ELEPHANTIASIS GRÆCORUM.

DEFINITION.—A chronic infectious disease, due to the invasion of the body by a specific organism, the bacillus lepræ, which produces localized inflammatory disturbances in the skin, mucous membranes, nerves, and viscera, associated with disturbance of sensation, ulceration, and necrosis.

ETIOLOGY.—Leprosy is a disease of wide distribution, sparing neither sex nor social condition. It prevails in early adult life, has never been found in the fœtus, and is extremely rare in infants. The assumed constitutional nature of the disease and the etiological importance of inheritance, especially emphasized by Danielssen, have yielded, since the discovery announced by Hansen, in 1880, of the constant presence of a characteristic bacillus in most of the products of the affection, to the view that leprosy is both infectious and contagious and due to this bacillus. The contagious nature of the disease was strongly advocated by J. C. White in 1882.

The bacillus of leprosy closely resembles in its various characteristics that of tuberculosis. Unlike the latter, it is not inoculable in the lower animals, and the statements of its having been successfully cultivated are still subject to criticism. The bacillus abounds in the diseased corium and subcutaneous tissue, and, according to the report of the India Leprosy Commission, is to be obtained in the fluid from blisters produced over the nodules, in the secretion from the ulcers, and in the saliva when the tongue and larynx are affected with the disease. It has rarely been found in the blood, and never in the urine.

Leprosy arose in Central Asia in remote times, and, according to Hyde, followed the lines of travel to the shores of the Mediterranean, from which it was carried to Norway, Iceland, and Greenland in the north, and to Egypt and other parts of Africa in the south. In the mean time Central Europe became invaded. The disease also travelled east from its place of origin in Asia, and made its appearance in China, in Japan, and in the islands of the Pacific. In North America the valley of the St. Lawrence received the disease from Norway, Iceland, and Greenland, while, according to Graham, New Brunswick was invaded from Normandy. The States bordering upon the great lakes became infected largely from immigrating Scandinavians. The Northern Pacific coast received its supply of cases from China and the Sandwich Islands, while the southernmost States, Mexico, Central and South America, and the West India Islands, were invaded from Southern Europe. According to J. C. White, there are now two hundred and fifty thousand lepers in India, and it is estimated that there are at least two hundred in the United States. Hyde states that there have been recognized in this country, up to 1890, five hundred and sixty cases of leprosy. Of these, one hundred and fifty-eight were found in California, one hundred and twenty in Minnesota, and one hundred in New York. Graham learned that two hundred and fifty lepers have been cared for in New Brunswick since 1815, and that in 1894 there were about thirty cases.

MORBID ANATOMY.—The lesions distinctive of leprosy are the variously distributed tubercles, nodules, and more diffused formations of granulation-tissue, which possess a considerable degree of permanency and therein differ from the granulation-tissue of tuberculosis and syphilis. The structure is chiefly composed of cells of various size, in particular of large endothelioid cells, although leukocytes are also abundant. Within and between the larger cells the bacilli of leprosy are present in enormous numbers, thus strongly contrasting with the distribution in the tissues of the bacilli of tuberculosis. Ulcers and scars accompany the nodules, and the former may lead to a loss of fingers and toes.

SYMPTOMATOLOGY.—Two varieties of leprosy are usually recognized, the anæsthetic and the nodular, tubercular, or tuberous leprosy, although a combination of the two varieties may exist in rare cases, even from the beginning of the disease.

The onset of leprosy is usually very gradual, and a period of years may intervene between exposure to the disease and the outbreak of the symptoms. Prodromal symptoms are usually present, but are of such a general character as to be of little significance. Weakness, dizziness, disturbed digestion, and slight fever are usually mentioned.

In *anæsthetic* leprosy the first distinctive manifestations are spots, macules, which may be preceded by bullæ, few or many, usually first appearing upon the face, hands, and feet, and afterwards elsewhere on the body. They are rounded or irregular in outline, for months remain stationary or recur from time to time, or gradually increase in size to that of the hand. They are of a red color, erythematous, at the outset, but tend to assume a brownish tint. The pigment later disappears, and the pale patches become anæsthetic. This insensibility may have existed from the outset, even in spots free from pigment, but in rare instances is preceded by hyperæsthesia or paræsthesia of the tissue. There may be deep-seated pain as well, and the nerves, at first usually the ulnar and peroneal nerves, become thickened, indurated, and painful, though eventually benumbed from the leprous perineuritis in which the bacilli and granulation-tissue are present: hence anæsthetic leprosy is regarded as a variety of peripheral neuritis. Contraction of the fingers and toes may follow, likewise trophic changes, evidenced by muscular atrophy, the formation of bullæ, ulcers, and necrosis, and a loss of hair and nails and of fingers and toes. When the facial nerves are diseased, ulceration of the cornea may take place and be followed by perforation and blindness. The bacilli are not found in the secondary cutaneous lesions resulting from the neuritis, although they are present in the inflamed nerve-sheaths.

Nodular or *tubercular* leprosy is characterized by the presence of nodules and of a diffused indurated swelling, developing at the outset especially on the face, hands, and feet. The nodules are somewhat sensitive to pressure, and vary in size from that of a pin's head to that of a filbert. They may develop from the macules or bullæ previously mentioned, or may arise in an apparently normal part, and may increase in size and coalesce or become absorbed and recur. Eventually they are likely to soften and give rise to ulcers, superficial or deep, which may heal with deforming scars. When abundant in the skin of the face a leonine aspect is suggested, whence the term leontiasis, of which appearance there are other causes than leprosy. The mucous membrane of the mouth, pharynx, and larynx is a not infrequent seat of the leprous formations, and a hoarse or whispering voice, dyspnœa, and cough may be due to the laryngeal affection. From the conjunctival mucous membrane the nodules may extend into the cornea and be present in the iris, causing blindness. Nodules are also to be found in the lymphatic glands, testis, spleen, and liver. The lungs are almost invariably spared, but Bonome has recently found these organs affected.

DIAGNOSIS.—The diagnosis of leprosy in the early stages is exceedingly difficult. Essential for the recognition of the disease are the development of the characteristic anæsthetic spots and the discovery of the bacilli in the nodules or secretion from the ulcer. Syphilis and cutaneous tuberculosis may be confounded with leprosy, but the macules of syphilis are more transitory and its nodules less permanent and contain no bacilli. The bacilli of cutaneous tuberculosis are less numerous in the lesions, especially in the cells, and are inoculable, thus differing from those of leprosy, which they so closely resemble.

PROGNOSIS.—The course of leprosy, especially of the anæsthetic type, is exceedingly chronic, and the disease may last many years, a period of from four to twenty years having been assigned. The result is usually death from marasmus or an acute intercurrent affection, as pneumonia, or from a complicating tuberculosis or nephritis. Recovery is possible in the case of the early discovery of limited lesions, although apparent cures are often followed by relapses or recurrences. Danielssen reports that he has observed ninety-two cures in forty-five years.

TREATMENT.—The prophylaxis of leprosy is isolation of the leper. There is no specific treatment. Chaulmoogra and gurjun oil have been used both externally and internally with asserted beneficial results, but there is no reason for believing that they are capable of curing the disease. Treatment must be systemic and sustaining.

SYPHILIS.

DEFINITION.—A chronic infectious and highly contagious disease, due to a specific virus of probably bacterial nature, producing a series of disturbances in more or less definite sequence in the skin and in various other parts of the body.

ETIOLOGY.—Syphilis is wholly limited to man, and it has been found impossible to transmit the disease to the lower animals. The virus is transferred directly or indirectly from one person to another, the so-called acquired syphilis, or is communicated from parent to offspring, hereditary syphilis. A person once infected is usually rendered immune from a reinfection, although in rare instances a child who has inherited syphilis may again acquire this disease in adult life. Furthermore, Colles observed that the healthy mother of a child who had inherited syphilis from the father was protected from infection by the child, who, at the same time, is capable of infecting other healthy persons. The virus abounds in the secretion of the initial lesion and of the papules, especially the moist papules in the secondary stage, and is usually considered to be almost wholly absent from the latest products of the disease. Von Zeissl suggests that the degree of contagiousness of the products of syphilis is dependent rather upon the time which has elapsed since infection than upon the nature of the product. On the other hand, it is doubtful whether the virus is present in the pus not due to the lesions characteristic of syphilis. The blood in the earlier stages of syphilis may or may not contain the virus, and it is also probable that physiological secretions do not contain the specific virus unless the surface over which they flow bears a lesion capable of producing this agent. It is, however, generally agreed that the ovum becomes infected by the semen; yet the latter secretion is generally thought to be incapable of directly infecting the mother, although it is considered possible that the latter may be infected from her syphilitic fœtus. The bacterial nature of the virus has long been assumed from its power of indefinite reproduction, and Lustgarten has found bacilli which he regards as characteristic in the secretion from syphilitic ulcers and papules; they have also been found in other pathological products of syphilis, even in the gumma. They somewhat resemble the bacillus of tuberculosis and that of the preputial smegma. Other observers have reported the occurrence of various forms of bacteria in this disease: so that the etiological importance of Lustgarten's bacilli and of the other bacteria is as yet undetermined.

Acquired syphilis is to be found at all periods of life: even the new-born child may become inoculated during the process of parturition. A local lesion of surface, although it is often so slight as not to be recognized, is necessary that the virus may be admitted to the body. The infection generally takes place during sexual intercourse, and the earliest manifestations are usually found upon or in the vicinity of the genitals, but in perverted intercourse may be seen elsewhere, especially upon the lips, tongue, and tonsils. Infection of the latter regions may arise without sexual perversion, since direct and indirect infection of the mouth may otherwise occur, the former by kissing, the latter by the use of infected articles, as pipes, drinking-cups, or other utensils used in common. The act of nursing may prove a source of infection from nurse to infant or from infant to nurse. The physician, surgeon, or nurse may be infected while caring for a syphilitic patient, and the syphilitic virus has been transmitted by the use of infected instruments by surgeon, dentist, or barber. Both infants and operator have become infected during the act of ritual circumcision, and before the use of animal vaccine the syphilitic virus was repeatedly conveyed in vaccination.

MORBID ANATOMY.—The virus of syphilis produces a series of local inflammatory disturbances varying in degree from vascular injection and slight œdema to the formation of masses of dense fibrous tissue. Various terms are applied to these lesions, as sclerosis or induration, papule, tubercle, and gumma. The tissue of which they are composed is a granulation-tissue, which tends towards ulceration, absorption, or induration. Ulceration occurs when the inflammatory tissue lies near the cutaneous or the mucous surface, although epithelial desquamation without ulceration may take place, as in the case of cutaneous papules, and crusts of inspissated secretion and epidermis may adhere to the surface of healing ulcers. The lesion generally regarded as the characteristic of syphilis is the gumma or syphiloma, which may be of a size recognizable only with the microscope or may form a tumor as large as the fist. It occurs singly or in numbers, diffused or circumscribed, usually in various parts of the body and near the surfaces, as well as in the interior, of organs and tissues, and is to be found, as a rule, during the later stages of syphilis. The older and larger gummata are composed of a series of opaque yellow masses of various size and irregular outline, separated from a dense opaque white fibrous tissue by a translucent gray or grayish-red zone. The central cheesy mass is composed of fibrous tissue in which are necrotic cells and fat-granules; the intermediary zone consists of abundant granulation-tissue, and the peripheral fibrous tissue is cicatricial, containing few cells and abundant fibres. The blood-vessels of the gumma often show a cellular hyperplasia of the intima with a corresponding diminution in calibre, frequently resulting in the obliteration of the canal, and leukocytes may abound in the vicinity of the adventitia. The more voluminous the gelatinous granulation-tissue the more active the growth of the gumma. The absorption of the syphilitic fibrous tissue may take place without resulting deformity, while the course of a gummous inflammation tends to produce destruction in its vicinity, and the eventual arrest of the process leaves a cicatricial tissue producing more or less extensive deformity as well in bone as in the soft parts.

The virus of syphilis is a frequent cause of chronic inflammation of the blood-vessels, especially the arteries, and, although the importance of syphilis in the etiology of chronic inflammation of the aorta in early life has long been recognized, Heubner first called attention to a similar process in the small arteries, especially in the brain. This obliterative endarteritis may be associated with a diffuse gummous inflammation, as in gummous leptomeningitis. Amyloid degeneration of the spleen, kidneys, stomach, and intestines finds in syphilis one of its chief causes.

ACQUIRED SYPHILIS.

Symptoms.—In the course of two or three weeks after inoculation, usually of the prepuce or vulva, although sometimes of the interior of the urethra, the initial or primary lesion of syphilis becomes manifest as a papule, an erosion, or an induration. Less often several of these lesions may be present, and a combination of erosion and induration known as the hard or Hunterian chancre is the usual manifestation. The essential characteristic of the initial lesion is the induration or sclerosis, which is manifested as a rounded, flattened nodule, somewhat movable, embedded in the substance of the skin and projecting slightly above its surface. The sore or ulcer is represented by the smooth, shining, and moist surface over the centre of the nodule. This is not to be confounded with the soft chancre, chancroid, or contagious venereal ulcer which follows inoculation in the course of four or five days, spreads rapidly, and usually suppurates within a fortnight, when healing takes place, resulting in the formation of a scar. This lesion is autoinoculable and sometimes contains the syphilitic contagium in addition to the chancroidal virus. The ulcer of syphilis may become infected by various bacteria, and extensive suppuration, pseudo-membranous inflammation, erysipelas, or gangrene arise as complications without modifying the specific effects of the syphilitic virus. The ulceration usually lasts but a few days, but may persist for weeks when a complicating inflammation of the surface exists. The induration continues for some time, usually not disappearing until several weeks after the cutaneous symptoms of syphilis are manifest, and may be present after many years. In the course of a fortnight after the appearance of the induration the lymph-vessels become thickened and the inguinal lymph-glands markedly enlarged, hard, but without pain, forming the indolent bubo. This is to be distinguished from the bubo of the soft chancre, which is of earlier occurrence, painful, of rapid increase in size, and manifesting a tendency to suppurate.

The Secondary Stage.—Within from three to five weeks after the occurrence of the sclerosis or induration, that is, within the course of two months after the infection, the secondary symptoms of syphilis indicative of a general infection become manifest, although exceptionally their development may take place earlier or later. Previous disease in an organ favors the subsequent localization of syphilis in it, and persons subject to naso-pharyngeal catarrh are prone to a syphilitic laryngitis, while syphilitic lesions of bones are of more frequent occurrence in parts especially exposed to injury. The lymph-glands in the various regions of the body become enlarged and indurated, and may remain thus altered for years even in the absence of other manifestations of the disease, and the periosteum, bones, and viscera may become diseased. These secondary symptoms in rare instances occur as a single attack lasting a few months, or progress with such rapidity and severity as to have received the designation of malignant syphilis. Usually, however, recurrent outbreaks take place, separated by an interval of months, in which the disease is regarded as latent, and the period during which recurrences are probable generally extends over a year or two.

Among the earliest secondary symptoms is the eruption or rash appearing upon the skin and the visible mucous membranes, to which the term syphilide is applied. The eruption is free from itching, and may be preceded or accompanied by a fever, represented by disturbance of digestion, muscular pains, headache, and prostration, and by an elevated temperature and quickened pulse. Albuminuria and enlargement of the spleen are associated. This symptomatic fever may also accompany recurrent attacks. The rash or eruption occurs as macules, papules, and pustules, and an important characteristic is the simultaneous occurrence of the several varieties, known as polymorphism. The rash is irregularly rounded, sharply defined, deeply pigmented, often copper-colored, and abounds in certain regions of the body, especially on the forehead, near the edge of the scalp, in the vicinity of the genitals, on the palms of the hands, and on the soles of the feet. The *macular syphilide*, syphilitic erythema, or roseola, appears especially upon the trunk as reddish-brown spots, fading somewhat on pressure, and becoming more conspicuous on the application of heat or cold to the skin. This rash lasts several days, fades during a severe intercurrent febrile attack, and frequently recurs during the latter part of the secondary period. Its presence in the nose is associated with coryza, and when occurring in the throat it gives rise to the syphilitic sore throat.

The *papular syphilide* may accompany or follow the macular rash, and is either sharply defined or has an erythematous base. The papules often occur in large numbers, widely distributed over the body, and present a variety of appearances according to the degree of development and the secondary changes. The miliary or syphilitic papule, syphilitic lichen, is distinguished especially by its size from the more common large papular syphilide. The former is about as large as a pin's head, and is found in the vicinity of the hair-follicles, especially in debilitated persons, and indicates a severe type of disease. The large papular syphilide occurs singly or in clusters, and persists for weeks, when the epidermis desquamates from the surface and absorption gradually and completely takes place. The presence of these papules at the angles of the mouth and in the interdigital folds of the skin is frequently associated with cracks, which readily bleed. In like manner when numerous upon the palms and soles the thickened skin becomes fissured and painful, such localization receiving the term syphilitic psoriasis. When the papules are present in moist places exposed to friction, as the region of the anus and genitals, the axillæ, between the fingers and toes, below the breast, the folds of the navel, the mouth and pharynx, the surface of the papules becomes macerated, perhaps ulcerated, and frequently bleeds. They then receive the term mucous patches, plaques muqueuses, or broad condylomata lesions, which are among the most contagious manifestations of syphilis. They frequently occur in numbers, and when clustered, fissured, and surrounded by inflamed skin, interfere with the function of the part by causing pain, hence producing difficulty in swallowing, nursing, and defecation. When present on the tongue, soft palate, uvula, tonsils, and pharynx, they are likely to form ulcers. Papules becoming mucous patches and associated with catarrh also occur in the larynx.

The term *pustular syphilide* is applied when the papules suppurate, a condition which is present in the severer forms of syphilis. According to the size and secondary changes in the suppurating papules are suggested the resemblances to acne, ecthyma, pemphigus, and variola. Acne is simulated when the pustules are in the vicinity of the hair-follicles, and therefore when they abound in the hairy parts of the body. Pustules enlarged to the size of peas and surrounded by a large dark-red infiltrated circle resemble ecthyma. The suggestion of variola is presented by umbilicated crusts formed by desiccation, while in syphilitic pemphigus the pustules become confluent and suggest a suppurating blister.

After the disappearance of the secondary manifestations of syphilis, especially in women, white patches, leukoderma, with an increased quantity of pigment in the vicinity, often occur upon the neck and elsewhere on the body.

General loss of hair, syphilitic alopecia, accompanies the outbreak of the rash, especially three or four months after the infection, the growth of hair being subsequently restored. The eruption of the papules or pustules in the bed or at the side of the nail, syphilitic onychia and paronychia, occurs soon after the loss of hair, and causes destruction or deformity of the nail.

Among the secondary manifestations of syphilis is periostitis, indicated by severe pain called osteocopic, especially marked at night, and accompanied by localized swelling. The usual result of the inflammation is a thickening and induration of the bone from both a periosteal and a central growth. When the periosteal growth is circumscribed it is called a node or exostosis, and is found especially upon the tibiæ, the clavicles, and the outer surface of the cranium. More rarely a periostitis is purulent, producing a gelatinous pus, which is either evacuated or absorbed; in the former case a superficial caries is probable, in the latter a bone-scar results.

Syphilis is the cause of nearly one-half of all cases of iritis, although iritis occurs in only about five per cent. of cases of syphilis. This affection usually accompanies the first outbreak or first recurrence of the rash, and therefore appears within the first six months of the infection. The milder forms are to be recognized as productive of little functional disturbance, but are associated with congestion at the edge of the cornea and discoloration of the iris. When papules are present in the iris there is severe pain referred to the eyes, aggravated at night, with intolerance of light. Choroiditis is a secondary symptom which sometimes accompanies iritis, but may occur independently of it.

The course of pregnancy is unfavorably affected by syphilis, since abortion or miscarriage is usually occasioned during the earlier manifestations of the disease: the habit of aborting should therefore suggest syphilis as a cause. Pregnancy also tends to prolong the duration of the symptoms of syphilis, although in the later stages of syphilis impregnation may be impossible.

The Tertiary Stage.—The later symptoms of syphilis are manifested by the presence of the gumma or syphiloma and by disease of the bone and viscera. Such manifestations usually begin several years after the infection, and are generally separated from the secondary symptoms by a period of latency, although in rare instances of severe syphilis the gummata may appear within six months of infection and be associated with the secondary lesions. The small gummata in the skin produce the *tubercular syphilide.* This is manifested by the presence of nodules, either few or many, sharply defined, brownish-red, resistant, and slightly projecting above the surface, of the size of a pea or bean, and starting from the deeper layers of the skin. They may be absorbed, but usually as the surface is approached break down and form sharply defined, rounded, and deep ulcers, which increase in size by a similar change in other nodules at the periphery. Their course is chronic, and they may heal at one part and progress at another, thus at times causing extensive cicatrices resembling those produced by burns. They also tend to recur. These ulcers are sometimes surmounted by crusts of inspissated secretion of considerable size with edges lamellated like those of an oyster-shell, when the term rupia is applied to them.

The larger gummata form tumors of the skin and mucous membranes, muscles, bones, and viscera. The gumma of the skin, as distinguished from the tubercular syphilide, begins in the subcutaneous tissue, and gradually increases in size, perhaps to that of a walnut. It eventually adheres to the skin, and becomes either absorbed or necrotic, in the latter case ending in an ulcer and a scar, as does the syphilitic tubercle.

Gummata of the mucous membranes occur as small nodules, gradually increasing in size, and resulting in indurated ulcers which spread laterally and in depth, causing extensive destruction ending in deforming scars or in perforation of bones or cartilage covered with mucous membrane. Such gummata are of more frequent occurrence in the mouth, pharynx, and rectum, but are rare in the stomach and intestine. The nasal septum may be perforated and the root of the nose sink in, and portions of the hard and the soft palate and of the uvula may be destroyed. The soft palate is at times united to the posterior pharyngeal wall, and forms an unyielding diaphragm across the naso-pharynx, through which sometimes a very minute opening is left. The extensive cicatrization of the tongue and pharynx and the perforation of the palate interfere with speech and deglutition, and even with respiration. Gummata of the rectum during the stage of ulceration cause painful defecation, while in the stage of cicatrization intestinal obstruction may result. The leukoplaques or milk spots often found in the mouths of syphilitics during the later stages have no necessary connection with this disease, as they occur in many persons free from venereal taint.

The bones are a favorite seat of gummata, which may develop from the periosteum as a gummous periostitis or from the bone-marrow as a gummous osteomyelitis. The superficial gummata are hard and painful at first, then become soft and elastic, and occasion the progressive destruction of the bone and the formation of sequestra. The overlying skin becomes ulcerated and perforated, and the carious bone is exposed. Both flat and long bones may become thus diseased, the cranial bones in particular being frequently affected, and the resulting gummata may form tumors of the inner and outer surfaces of the bone, leading to perforation of the skull and exposure of the dura mater. The projection of gummata from the bodies of the vertebræ may compress the spinal cord, while absorption of the central gummata of long bones causes one variety of the spina ventosa found in macerated bone. Gummata also form in the joints in both the synovial membrane and the ligaments, while the adjoining tendons and the bursæ, especially the tendo Achillis, are not infrequent seats of the gumma. In *dactylitis syphilitica* there is a chronic inflammation of the fingers and toes, due to a diffuse gummous affection of the subcutaneous tissue, which eventually reaches the periosteum and involves the ligaments.

Gummata occur in the muscles, especially in those of the upper part of the body, as circumscribed, somewhat movable indurations, and usually disappear by absorption. The occurrence of a chronic fibrous myositis independently of the formation of gummata as a late result of syphilis is considered probable by certain writers.

Visceral Syphilis.—The lesions of syphilis are often found in the internal organs. Syphilis of the brain and cord is considered in detail in the section on Diseases of the Nervous System (see pages 528, 592, 618). The occurrence of gummata of the stomach and intestine has been referred to.

Numerous observations have been made of gummata of the lungs, which have been described as being as large as an egg, as few or several, as occurring at any part of the lung, and as closely simulating in appearance cheesy tubercles. They are a late manifestation of syphilis, and are productive of no disturbance to the function of the lungs. In the light of our present knowledge, the occurrence of pulmonary symptoms in acquired syphilis, especially when associated with signs of solidification of the lung, is to be regarded rather as a complication than as a result of this disease.

Syphilis of the heart occurs in the form of gummata or of a diffuse fibrous inflammation, or of both combined. The gumma is to be found within the myocardium, either alone or with other gummata, and may be as large as an apricot. The diffused formation of fibrous tissue may take place in the pericardium, myocardium, or endocardium, in the vicinity of the valves or remote from them. Mraček has recently collected sixty-one cases regarded as of unquestionable cardiac syphilis. The conspicuous symptoms were those attributable to muscular insufficiency, and were characterized by palpitation, a sense of oppression, pain, and dyspnœa, even severe angina pectoris. Semmola calls especial attention to extreme irregularity in the action of the heart. In one-third of the cases collected by Mraček death occurred suddenly, either taking place without warning or being immediately preceded by coma or epileptiform convulsions.

Heubner has called attention to the importance of syphilis in the etiology of endarteritis, especially of the smaller arteries of the brain. Both a periarteritis and an endarteritis may occur in this organ as late lesions of syphilis even if the cerebral gumma or a gummous inflammation of the meninges is absent, and produce various disturbances dependent upon the seat and distribution of the arteries. The affection of the blood-vessels is especially pronounced in those cases of cerebral syphilis in which the symptoms resemble those of general paralysis. It has long been recognized that chronic thickening of the aortic intima previous to the age of forty is principally due to syphilis, and many authors consider that a similar alteration of the arteries elsewhere may be due to the same cause: hence this disease is regarded as of importance in the etiology of aneurisms and disease of the heart and kidneys in which chronic endarteritis is a conspicuous feature. Further attention is given to this subject in the consideration of the affections of the arteries.

Syphilis of the liver is also manifested by the occurrence of gummata and a diffuse fibrous inflammation. The gummata occur particularly in the vicinity of the suspensory ligament, often in groups, and are to be found isolated in other parts of the liver, both near the surface and within the substance of the organ. As a result of the absorption of the gummata the liver is divided by the contraction of the surrounding cicatricial tissue into irregular lobulations, which are often associated with a fibrous perihepatitis, and the lobulated liver is as suggestive of syphilis as the gumma is pathognomonic of this disease. Syphilis of the liver is not characterized by other symptoms than those dependent upon obstruction to the portal circulation, which is usually moderate in degree. Gummata are also to be found in the pancreas and the spleen, and rarely in the kidneys and the suprarenal capsules.

Syphilis of the testis is an important affection as frequently leading to the recognition of the cause of obscure conditions due to visceral syphilis. There are two products, one an interstitial orchitis, the other a gumma, although both may be associated. In interstitial orchitis there is fibrous thickening of the tunica albuginea and the septula of the testis, and, in consequence, on section of the gland an arborescent appearance is presented suggesting that of a deer-horn. The testicle becomes atrophied in consequence of the contraction of fibrous tissue, and a symmetrical or irregular shrinkage results. The gumma is indicated by an enlargement of the testicle, due to the presence of one or several gummata, which increase in size, tending to produce a symmetrical enlargement, syphilitic sarcocele, or syphilitic testicle, with increasing destruction of the tubules, and the tunica vaginalis becomes obliterated, from an associated periorchitis. Both interstitial orchitis and the gumma may be found in one or both testes, progressing slowly, and usually unaccompanied by other symptoms than those caused by the size and weight of the gummous testicle. The gummous differs from the tuberculous orchitis in the usual limitation of the process to the body of the testicle, and in the customary freedom from disease of the epididymis, vas deferens, seminal vesicle, and prostate. It differs from malignant tumors of the testicle in slowness of growth and freedom from pain, and from both malignant and tuberculous testes in showing no tendency to ulceration or to the formation of fungous excrescences. Despite these differences, the syphilitic testicle has often been removed with the diagnosis of sarcoma. Absorption of the gumma may take place with induration, while softening or suppuration rarely occurs. Gummata and interstitial inflammation may occur in the ovaries and in the mammary gland.

HEREDITARY SYPHILIS.

Hereditary syphilis is derived from either father or mother or from both parents, the semen or ovum being capable of conveying the disease; and it has been observed that one of a pair of twins may inherit syphilis while the other remains exempt. A distinction may be drawn between hereditary syphilis and congenital syphilis, the former representing an infection of the semen or ovum, while in congenital syphilis both parents may be free from the disease at the time of conception, yet the mother be subsequently infected, perhaps by a third person, and infection of the fœtus result, this event being the more probable the younger the fœtus. This method of infection has been designated intra-uterine, although Kassowitz concluded that the syphilitic virus does not pass through the vascular walls dividing the fœtal from the maternal blood. As a rule, the syphilitic father infects the wife and has syphilitic offspring, but a syphilitic father may impregnate a healthy mother and may or may not beget a syphilitic child. A healthy father may impregnate a syphilitic mother and a like result follow. The likelihood of the syphilitic father or mother begetting syphilitic offspring apparently depends upon the stage of the disease in the parent at the time of conception. If in either the earlier manifestations of syphilis are present, the child is usually syphilitic. If both parents show the earlier manifestations of syphilis, the child is sure to be syphilitic. If, on the contrary, syphilis is latent in the parents at the time of conception, the offspring may or may not be syphilitic. If there have been no symptoms of syphilis for several years in either parent, or if only tertiary manifestations be present, the children are likely to be free from syphilis. The limit of time between the infection of the parent and the possibility of transmitting syphilis has been somewhat arbitrarily fixed at from four to six years, although cases are reported in which after many years from infection syphilis has been transmitted. It is generally agreed that the tendency to transmit syphilis to the offspring is diminished by antisyphilitic treatment.

MORBID ANATOMY.—In hereditary syphilis, in addition to such circumscribed and diffuse formations of fibrous tissue as occur in acquired syphilis, there is to be found the typical lesion discovered by Wegner near the epiphyses of the long bones and known as syphilitic osteochondritis. It is characterized by an excessive width of the zone of ossification, the edge of which is jagged. The lime salts are irregularly deposited in it, and an extreme brittleness results, in consequence of which the epiphyses may be separated from the shaft. Especial importance is to be attached to the alterations found in the lung, since they frequently prove the immediate cause of death. The white hepatization first described by Virchow, in which the lungs are distended, non-crepitant, of a grayish-white color, and the alveoli filled with epithelium in a state of fatty degeneration, is found in the still-born or in those dying soon after birth. Heller has recently described the frequent occurrence of a fibrous pneumonia due to inherited syphilis and more or less extensively distributed throughout the lüngs. Both the cellular infiltration and the fibrous thickening of the interstitial tissue cause a narrowing of the alveoli, while the lungs are large, red, and dense. The infected portions of the lung are often emphysematous. This fibrous pneumonia occurring in families in which the older children were born prematurely or died of syphilis during or after birth may or may not be combined with other manifestations of syphilis. Gummata are stated to be present in the lungs in one-fourth of the cases of inherited syphilis. Among the visceral lesions of clinical importance to be met with in hereditary syphilis are enlargement of the liver, in which miliary gummata may occur, enlargement of the spleen, often forming a palpable tumor, and fibrous induration of the pancreas. Combined fibrous thickenings and gummata may be found in the several viscera, though more rarely than in acquired syphilis.

SYMPTOMS.—The more recent the infection of the parent at the time of conception, the earlier after birth do the symptoms of hereditary syphilis make their appearance. They are usually more severe if the syphilis is inherited from the mother, while the older children of syphilitic parents commonly suffer more seriously than the younger from the manifestations of the disease. As has already been stated, abortion or miscarriage is the frequent outcome of impregnation soon following infection. In subsequent pregnancies the children may be still-born, or may live for a few minutes after birth, in which case white hepatization of the lungs is often found and ecchymoses are frequent: the so-called hemorrhagic syphilis of the new-born may find an explanation in this affection of the lungs. In the children of later pregnancies the symptoms of syphilis become manifest in the course of days, weeks, or months after delivery, according to the duration and severity of the symptoms in the parent. The longer the period between birth and the development of the symptoms of syphilis the more possible is intra-uterine infection as a cause, and a knowledge of the previous history of the parents becomes important when answering the question whether the syphilitic symptoms in the child at the end of six weeks after delivery are due to inherited syphilis or to infection, either intra-uterine or during birth.

The infants of enfeebled vitality from inherited syphilis are small and emaciated, showing both the cutaneous and the visceral lesions of syphilis. Coryza and a feeble, hoarse cry are often present. Pemphigus is especially frequent, either at birth or within a day or two afterwards, as purulent blisters upon the hands and feet; and this syphilide is of grave import. The characteristic changes of the epiphyseal cartilage are to be found, and death usually occurs, often being the result of pulmonary syphilis or of broncho-pneumonia. Other children may appear well nourished at the time of birth, but in the course of a few weeks, usually not later than three months, according to the stage of syphilis in the parent, the symptoms of hereditary syphilis appear, generally confined to the skin and the mucous membranes. Macules, often combined with papules, abound. The papules near the mouth, anus, and genitals rapidly become mucous patches, while those upon the palms and soles tend to assume the characteristics of psoriasis. Pustules rapidly develop and tend to coalesce, especially upon the hands and feet, producing pemphigus. Snuffles also appear, and may even precede the rash, and the skin in the vicinity of the nostrils becomes macerated and ulcerated, while crusts form and interfere with respiration, especially when nursing. Cutaneous ulcers and gummata are rare early manifestations of inherited syphilis, although the ulcerated gumma of the nasal septum leads to the depression of the root of the nose, with its suggestive deformity. The nutrition of the child becomes impaired, the infant is wrinkled, anæmic, and fretful, and its death is likely to take place within a few months.

The child may recover from the immediate manifestations of hereditary syphilis, but its development is usually stunted, rickets is frequent, and chronic osteitis may cause sclerosis of the bones. As the age of puberty is reached, a fresh outbreak of the disease may arise, and gummata and ulcers frequently appear. Hutchinson regards as indicative of hereditary syphilis the combination of keratitis, producing more or less permanent opacity of the cornea, labyrinthine disease, causing deafness, and a crescentic notching of the upper middle incisor teeth, which are also farther apart at the base than at the cutting edge. Such alterations may be due also to other conditions seriously modifying nutrition, and therefore are not absolutely characteristic of inherited syphilis.

DIAGNOSIS.—The diagnosis of syphilis is not to be made until induration in the region of the primary lesion and in the neighboring lymph-glands has taken place. The recurrent and polymorphic nature of the eruption and its association with indurated glands makes the diagnosis during the secondary symptoms relatively easy, despite the frequent assertion of no known exposure. In the latest stage of cutaneous syphilis the obstinate ulcerations of gummata are to be discriminated from lupus, that is, cutaneous tuberculosis, which they most closely simulate. The gummata are vascular, painful, and rapidly form deep, crater-like ulcers, which are benefited by antisyphilitic treatment and heal with the formation of depressed scars. Cutaneous tuberculosis, on the other hand, usually begins early in life, progresses slowly, and gives but little pain. There is extensive cheesy degeneration; the ulcers are less sharply defined, are not benefited by antisyphilitic treatment, and, when healed, are replaced by superficial scars. The diagnosis of cutaneous tuberculosis is to be made by the histological examination, the discovery of the characteristic bacilli, or the occurrence of positive results from the inoculation of portions of the growth. Visceral syphilis produces disturbances in function of various organs in no way differing from those due to other causes. The history of the patient may aid in the differential diagnosis, and the physical examination may show the induration left from a primary lesion, general enlargement of the lymphatic glands, deep and deforming cicatrices of the skin, pharynx, or rectum, tibial nodes, or a syphilitic testicle.

Visceral syphilis may yet exist, although the physical examination of the surface of the body gives no aid in recognizing the cause of the visceral lesions. The diagnosis of the especial lesion of the organ concerned and the means of forming an opinion as to the cause of the lesion are especially considered in the description of the diseases of the various organs.

PROGNOSIS.—The prognosis of acquired syphilis is never to be definitely forecast. Although spontaneous cures may occur at any stage in its progress, various treatments prove effectual, and the so-called specific treatment often produce almost immediate benefit, there are cases obstinate to all forms of treatment, temporary benefit may be followed by a renewed outbreak, and even without external manifestations visceral lesions may slowly progress. There is no absolute test of the time when recovery from syphilis has taken place. Experienced authorities agree that a period of two to four years must elapse before the probability of recovery is assumed. In general, syphilis pursues a milder course in strong vigorous persons of cleanly habits and in favorable surroundings, while the progress is slow and severe in persons debilitated by insanitary surroundings or by tuberculosis, scrofula, malaria, or alcoholism. The idiosyncrasy of the patient is also of importance, perhaps, to be explained by the relative immunization of certain individuals in virtue of syphilis in a near ancestor. When death results, it is due either to an extensive invasion of the brain or to the localization of the disease in a part especially necessary for the maintenance of life, as important cerebral centres, the heart, or the lungs, or to an extensive destruction of an important organ, like the liver, or to a limited but prohibitory interference with the function of the alimentary canal. Syphilis also acts as one of the conspicuous causes of general amyloid degeneration, the presence of which is necessarily fatal.

The prognosis of hereditary syphilis is extremely grave, since, according to Kassowitz, one-third of those diseased die in the first six months of life. The prognosis is the more favorable the later after birth the manifestations appear.

PROPHYLAXIS.—Although in recorded cases syphilis has been contracted from drinking out of cups attached to public fountains, from accidental contact with individuals infected with the poison, and in numerous other strange methods, even at the communion-table, such accidents are so rare and unforeseeable that they must be considered among the calamities of life against which no prevision can protect. Accidental infection of the practitioner of medicine is unfortunately not infrequent,—liable to occur to any gynæcologist, obstetrician, or surgeon. In the vast majority of cases, however, syphilis is produced by impure coitus, and is to be avoided by a moral life.

Attempts have been made to prevent the development of syphilis by the regulation of prostitution, a practice which, indeed, prevails in a large part of Europe. Without occupying space with any detailed discussion of this matter, we desire to express our conviction that such regulation is, at least so far as concerns the United States of America, not a measure to be favored. We have reached this conclusion not through any moral or philanthropic reasoning, nor have we any sentiment concerning the matter. If syphilis could be banished, or even seriously diminished, by licensing houses of prostitution, we believe that it would be the duty of government to disregard the feelings and rights of individuals as ruthlessly as it disregards the right of animals to live when it sets a bounty upon the heads of wolves. We believe, however, that no measures that can be taken would be of any value. It is notorious that although police espionage and bureaucratic government have reached in Europe their seemingly most perfect development, and although such government has strained itself to the utmost to protect its citizens from syphilis, yet syphilis is most rife in those countries in which the government most assiduously attempts to protect its male citizens from the consequences of impure life. In the United States, where respect for the rights of the individual is carried to an extreme, where the government itself has the looseness and inefficiency which seem to be the inevitable outcome of democracy, we believe that any attempt at such regulation of prostitution would rather increase than diminish the spread of the disease; because it would, in a measure, increase sexual impurity by taking away the fear of consequences, which undoubtedly is a check upon many, and because by the sense of confidence begotten it would diminish the watchfulness of the individual to protect himself from the results of his own acts.

The question of marriage is continually referred to the practitioner by persons suffering from syphilis. The statements of syphilographers as to the time when marriage may be contracted with a fair probability of health to the woman and to the offspring vary. In our opinion four years is the least period which should be allowed to elapse. The number of women whose lives have been wrecked by marriage with syphilitics is appalling. The rule should be absolute that at least two years of continuous treatment and two years of interrupted treatment should pass by before marriage is contracted. Syphilitic lesions which occur after this date are generally believed to be non-contagious and not inheritable.

TREATMENT.—There are only two drugs which are of value in the treatment of syphilis,—namely, mercury and potassium iodide. Of these mercury is especially adapted to the early stages of the disease, iodide to the later. The first question which the physician has to answer is when to begin specific treatment. There are two different ideas held concerning this subject by syphilographers: according to one teaching, specific medication should be begun just so soon as the diagnosis is made out; according to the other, it should not be entered upon until distinct secondary manifestations appear. According to our belief, the first of these teachings is correct; but, on the other hand, mercurials should not be given until the diagnosis is established, as it is a matter of vital importance for the individual to know whether he has or has not syphilis; whereas if mercurials be given before this knowledge is attained certainty may be impossible. We believe specific treatment should be commenced as soon as the diagnosis is made, for various reasons, as important among which may be mentioned the possibilities of permanent local injuries by the specific lesions and the great danger of the accidental infection of other individuals through the presence of the primary sore. Moreover, although in some parts of the world the public knowledge that a man has had syphilis does not sensibly injure his prospects in life, it is not so in the United States. We do not believe that any advantage is gained by waiting. The talk of certain prominent syphilographers about the disease getting "ripe" seems to us a remnant of mediæval fetichism.

Mercury may be administered by the mouth, by inunction, or by hypodermic medication. In the great majority of cases, especially in the beginning of the disease, administration by the mouth is preferred, inunctions being to many unpleasant and often involving the risk of discovery; hypodermic injections are also more or less unpleasant and require some skill in their administration. Both inunctions and hypodermic medication have the great advantage of being less liable to disturb the digestion and produce diarrhœa, and on occasion must be used.

Syphilographers vary greatly in their recommendation of individual preparations of mercury: some prefer mercury with chalk or gray powder, others adhere to blue pill or to calomel, whilst still others affect the red or the green iodide of mercury. It is probable that one of these preparations is as effective as the other, provided it be given in the proportionate dose, although clinical experience shows that sometimes one, sometimes another, suits better the individual case.

Again, two methods of using the mercury exist: one is to make a series of acute attacks, so to speak, of mercurialism: the other is to maintain a steady mild influence. It is very doubtful whether one of these plans has any distinct advantage over the other. We have preferred to use the milder method, to give the mercurial steadily in such dose as shall keep the condition just below that of ptyalism. When the syphilitic processes are active, and especially when they involve a vital organ, mild ptyalism should be produced if it be possible.

The length of time over which mercurialization is to be carried should, in our opinion, be about two years. The mercurial course should always be followed by one of potassium iodide, which should be kept up for many months or a year, the drug being given in such dose as not to disturb the digestion or produce any systemic effect. The object of this procedure is twofold: the prolonged exhibition of mercurials leads to the deposition of mercury in the tissues, whilst the iodide restores this mercury to a soluble salt, with a consequent reabsorption into the blood and elimination. In the second place, the iodide is a very useful remedy in the relief of remaining processes of the specific disease: it should be given in increasing doses until evidences of iodism are induced, and then in half or three-quarter doses continuously for one year.

As a rule, the mercurials and potassium iodide are much better borne by syphilitics than by normal individuals, so that the failure of one of these drugs given in large doses to produce systemic reaction is presumptive, though not positive, evidence of the existence of syphilis, provided the immunity has not been obtained by the gradual use of the remedy. Nevertheless, there are syphilitic persons who will not tolerate mercury, or in whom the iodides in minute doses produce at once constitutional symptoms. In such persons it will usually be found that the small dose of the mercurial or of the iodide accomplishes, so far as the disease is concerned, the same result as in the ordinary individual is produced by the larger dose. The presence of the idiosyncrasy is therefore not an indication for the withdrawal of the drug, but for the reduction of the dose. In giving potassium iodide it is often essential that doses as large as can be borne be administered. In a doubtful case ten grains three times a day may be first given; a few days later, twenty grains; if this be tolerated, forty grains may be administered at once; and if this produce no iodism, three drachms a day may be given in dilute solution, preferably in milk.

The treatment which has thus been detailed is that of a case of syphilis which pursues a favorable course and does not develop any severe local manifestations or derangement of the health or digestion. If such manifestations occur, modification of the treatment is usually necessary. Large or small doses of the mercurials may be given, or large or small doses of the iodides may be required.

It is sometimes advantageous to abandon temporarily the use of mercury, as it seems at times almost to lose its effect upon the system. Under such circumstances it is best to give the patient a course of the iodide and then to return to the mercury. Often, especially in the advanced stages of the disease, a mixed treatment with the mercury and the iodide is advantageous.

When, owing to the involvement of some important organ in a syphilitic lesion, special accidents occur in syphilis, the principle of treatment is to meet such accident as it would be met if it arose from other cause, and at the same time to push actively antispecific medication. If the syphilis dangerously involve any vital organ, mercury should usually be administered in large dose at once, so as to remove the gumma and prevent the secondary effects of pressure or of inflammatory involvement. These principles will be found worked out in some detail in the article on Cerebral Syphilis, to which the reader is referred. The methods of mercurialization by inunction and by hypodermic medication are also described there.

The only preparation of mercury which should be used hypodermically is corrosive sublimate. Very many other simple and complicated preparations have been recommended from time to time, but are of inferior value. The rule in syphilis is that cachexia contra-indicates the use of mercurials; yet we have often seen syphilitic cachexia yield rapidly to the combination of corrosive sublimate with tincture of chloride of iron, and we consider it an axiom that when in syphilitic cachexia there is a very pronounced anæmia such combination should be used.

Mercurialization may be produced by the process of fumigation, either with calomel or with cinnabar. The method is especially useful in cases of syphilitic eruptions. The patient should sit upon a stool or chair, enveloped with a blanket tightly secured around the neck and spread out below in the manner of a tent. Beneath the chair the mercurial is volatilized on an ordinary tin plate with a lamp underneath it. Various patterns of lamps are furnished by instrument-makers, but the simple spirit-lamp set upon a plate, with an ordinary chemical tripod over it, will suffice.

The general tendency of syphilis is to the breaking down of the constitution, so that during antisyphilitic medication it is essential that the bodily health of the patient be maintained by hygienic and other means. According to our experience, syphilitic diseases in the woman are prone to yield less readily than in the man, and especially to be connected with anæmia and exhaustion: so that, whilst very active specific medication is often necessary, great care must be taken, by the use of iron, tonics, and hygienic measures, to maintain the strength of the patient.

In congenital syphilis the best treatment consists in rubbing the abdomen of the child with mercurial ointment and covering it with a flannel smeared with the drug. If it be important not to arouse suspicion, mercury with chalk or other preparation may be given by the mouth; later in the disorder the mixed treatment of mercury and iodide is very useful. The antispecific medication should be kept up at intervals for years, and the child should be especially watched at the period of the second dentition and at puberty for the development of lesions, whose presence will be the signal for immediate active medication.

CHAPTER IV.

DISEASES DUE TO ANIMAL PARASITES.

A VARIETY of animal parasites find their host in man, are nourished at his expense, and either produce little or no disturbance or give rise to grave, if not fatal, disease. In their structure they vary from the simplest forms of animal life to those more highly differentiated. As a rule, they enter the body in food or drink, and either remain permanently in the intestinal canal, or, migrating from this region, are to be found even in the remotest parts of the body; while other parasites infest the skin, are incapable of extensive migration, and produce simply local disturbance. In our consideration of the subject we have closely followed Mosler and Peiper.

PARASITIC PROTOZOA.

Of the lowest form of animal life, the *protozoa*, several varieties occur among human parasites. One of the most important of these is the *Amœba coli* or *dysenteriæ*, which plays an important part in the production of dysentery. (See page 213.) Amœbæ have also been found in the urine, as a rule in connection with hæmaturia, with or without evidences of nephritis. In one case there was hemorrhagic cystitis, and the wall of the bladder was thickened and contained amœbæ in abundance.

The *sporozoa* form a group closely allied to which are the *hæmatozoa*, the description of which is to be found in the article on malaria, page 203. The *Coccidium oviforme*, one of the sporozoa, has proved a cause of death in man, in whom it is rare, although it is of frequent occurrence in rabbits, cats, dogs, and other domesticated animals. According to Leuckart, the amœboid young coccidia enter the epithelium of the intestine and bile-ducts and form capsules in which are developed numerous spores, whence the term sporozoa, which are locally multiplied or transferred elsewhere after the capsules break. Collections of the coccidia are to be found in the epithelium of the intestinal wall and in the liver of rabbits, in the latter forming opaque yellow or white tumors. In man a number of tumors of a similar character were seen by Gubler, and Leuckart recognized coccidia in them. Since then collections of this parasite have been observed not only in the liver, but also in the intestinal mucous membrane, in the myocardium, and in pleuritic exudation, although in general no symptoms have been attributed to their presence. Podwyssozki has reported four cases in which the coccidia were present in the liver, not only in nodules but also in the hepatic cells, and were regarded as the cause of an associated jaundice and cirrhosis. Reference may be made to the presence of hyaline bodies in the disease of the skin called by White keratosis follicularis, in molluscum contagiosum, in Paget's disease of the nipple, and in epidermoid cancer. These bodies have been regarded as psorosperms or sporozoa, and have been considered as the cause of the disease in which they were found, and their presence has been assumed as directly favoring the theory of the parasitic origin of cancer. Although observers are not wholly agreed upon their nature, it is generally conceded that they represent simply a hyaline metamorphosis of epithelial cells. That sporozoa may be of importance in human pathology is evident from the cases described by Rixford and Gilchrist. Nodules had existed in the skin for a period of months or years, the lymph-glands were enlarged, and sporozoa inoculable in rabbits and dogs were obtained from the skin, from lymph-glands, and from tuberculoid nodules in the lungs, spleen, liver, adrenals, and testis.

The *infusoria* form another group of the protozoa important in human pathology. The flagellate infusoria include the *Megastoma entericum* or *Lamblia intestinalis*, the *Cercomonas intestinalis* and *Cercomonas coli hominis*, the *Trichomonas intestinalis* and *Trichomonas vaginalis*. With the exception of the last, these parasites have been recognized in the intestinal contents in a variety of diseases. They have likewise been found in the nose, in the mouth, and, according to Osler, in the vomit, also in gangrenous lung and in pleuritic exudations. Their presence in enormous numbers in the intestinal mucus of children and adults suffering from chronic diarrhœa and dysentery indicates that they may be of pathogenic importance, whether as a cause or as a complication. Dock has recently reported a case of trichomonas in the urine, the parasite being regarded as the cause of the associated inflammation of the bladder and hæmaturia. He suggests that the presence of this parasite should be sought for in hæmaturia, so often in the South attributed to malaria, especially when independent of other symptoms of the latter affection.

Of the ciliated infusoria the *Balantidium coli* has attracted attention from having been found in the stools of dysentery. According to Mosler and Peiper, twenty-eight cases have been observed, most of them occurring in the vicinity of Stockholm, although two of the patients are said to have been infected in the United States. The pathogenic importance of this parasite is still in doubt, but in the reported cases it has been observed that an exacerbation of the diarrhœa was associated with an increase in the number of the infusoria in the stools.

HELMINTHIASIS.

DEFINITION.—The disturbances produced by parasitic worms.

The varieties of verminous parasites found in man are the tape-worms, or cestodes, the flukes, or trematodes, the leeches, or anellides, and the round and thread worms, or nematodes.

TAPE-WORMS.

The cestoid parasites include the several varieties of tænia, or tape-worm, which prove injurious to man by their presence in the intestine, and especially by their occurrence in the larval stage in the various organs and tissues of the body. From the mature worm which lives in the intestine of man or of a lower animal are discharged eggs either free or included within the proglottides or segments. If these eggs are swallowed by man or a suitable lower animal, the envelopes are digested and the embryos set free. The latter penetrate the walls of the blood-vessels and lymphatics, and are then carried to various parts of the body, in which their development into cysts, the cysticerci or echinococci, takes place. These cysts are the larvæ of the tape-worm, and when swallowed become the tape-worm. The tæniæ of most frequent occurrence in man are the *Tænia solium* and the *Tænia saginata.*

Tænia Solium.—The tænia solium, or pork tape-worm, six to nine feet long, has a round head about the size of a pin's head, armed with twenty-six hooklets in a double row, rising from a pigmented base, and provided with four suckers. The narrow neck soon becomes transversely lined, an indication of the formation of segments, which, some three feet from the head, instead of being elongated are square. In the fully developed segments, from the four hundred and fiftieth downward, both the male and the female generative organs are found, and the uterus is readily seen, on pressure of the proglottid between plates of glass, as an arborescent figure with a central trunk, from each side of which eight or ten lateral branches project. From the border of the segment projects a small elevation, the genital opening, out of which the ova, which exist to the number of thousands, may be pressed as an opaque fluid. After the tape-worm has been three or four months in the intestine the mature segments, nine to ten millimetres long and six to seven millimetres wide, may be found in the stools. They are frequently misshapen, from partial or complete fusion, from the presence of lateral buds, or from perforation. From the eggs taken into the stomach of man, swine, sheep, dog, and rat is developed the *Cysticercus cellulosæ* to be found in various parts of the body; in swine the condition thus produced is called *measles.*

The *Tænia solium* lives in the middle of the small intestine, to the wall of which it clings by its hooklets, and may remain alive for several days after the death of its host. Although usually found alone, several may be present, and Kleefeld observed forty-one in the same individual. They may lie outstretched in the intestine, the head uppermost, or may form a complex knot; and at times, through a reversion of peristalsis, segments may be vomited. The *Tænia solium* is very common in Central Germany, in which country raw or insufficiently cooked pork is often eaten, and from one-third to one-half of the patients seeking hospital aid for various purposes harbor the parasite. In regions in which pork is but little eaten, or in which cooking or various methods of its preservation have destroyed the vitality of the eggs, the pork tape-worm is relatively uncommon.

Tænia saginata or **mediocanellata**, the beef tape-worm, is twelve to twenty-four feet long, and has a square, pigmented head, as large as that of a pin, provided with four suckers, but free from hooklets. The neck is short, the mature segments sixteen to twenty millimetres long and four to seven millimetres wide. The uterus has twenty to thirty lateral branches, which are usually dichotomous. The position of the genital opening and the irregularity in the development of the segments are the same as in the case of the *Tænia solium.* The cysticercus of this worm is rarely found in man, but usually develops in the muscles and viscera of cattle, in which it is often overlooked from its small size and its rapid shrinkage when exposed to the air.

The *Tænia saginata* clings to the wall of the small intestine by means of its suckers, and abounds in those countries in which beef is the chief article of animal food. It is, therefore, the common tape-worm of the United States. Its propagation in man is dependent upon the use of raw or insufficiently cooked beef.

The *Tænia elliptica* or *cucumerina* has been found in infants and young children, but abounds in dogs and cats, the embryos being harbored in lice and fleas. The *Tænia nana* has also been repeatedly found in children. There are a few instances of the occurrence of the *Tænia flavopunctata*, and Weinland and Leidy have found it in this country. Rats and mice are the usual hosts, the embryos developing in insects. In the East the *Tænia Madagascariensis* has been found, and Weinland described the presence in a Virginian of the cysticercus of the *Tænia acanthotrias*, although its mature tape-worm has not been found.

The *Bothriocephalus latus*, or fish tape-worm, is fifteen to twenty-seven feet long, and has a club-shaped head, without suckers or hooklets, but provided with two lateral grooves. The proglottides are broad and short. The eggs escape into the intestine from the ripe segments, and are further developed in water. They are swallowed by the pike, perch, salmon, and turbot, in the flesh and viscera of which, according to Braun, the embryos are found, and from which the mature worm has developed in dogs, cats, and man. In regions where improperly cured fish is eaten, especially along the Baltic, in Bavaria, and in Switzerland, this worm abounds. Odier states that in Geneva twenty-five per cent. of the population harbor this parasite.

The *Bothriocephalus cordatus* and the *Bothriocephalus cristatus* may be mentioned as of rare occurrence, while the larval stage of the *Bothriocephalus liguloides* has been observed in China and Japan.

ETIOLOGY.—The tape-worms of man, according to the variety, are derived from raw or insufficiently cooked or preserved beef, pork, and fish.

They are more frequent in men than in women, and abound during the middle third of life, although common among children, and Mensinga found the tape-worm in an infant of ten weeks. Mosler and Peiper state that butchers, innkeepers, waiters, cooks, and housemaids are especially apt to be affected.

SYMPTOMS.—The parasites may be harbored for years, especially by robust individuals, without producing any disturbance, but sensitive persons, particularly women, are likely to suffer various symptoms, notably after the existence of the tape-worm has been discovered. Even before its presence is recognized such persons may be anæmic, easily tired, and subject to digestive derangements. Whatever irregularities of digestion arise after the recognition of the presence of the tape-worm are invariably attributed to its presence. The appetite often becomes feeble or capricious, but more frequently is excessive. Nausea, vomiting, and the regurgitation of gas and a bitter or acid fluid occur. Attacks of colic arise without apparent cause; existing diarrhœa or constipation is often attributed to movements of the worm, which are frequently asserted to be aggravated by certain kinds of food and assuaged by agreeable articles of diet. Women who have borne children have stated that the movements of the tape-worm in the bowel simulate those of the fœtus in the uterus. Numerous disturbances of the nervous system are attributed to the parasite, and are regarded as of a reflex nature. Such are mental and physical sluggishness, often suggesting melancholia and hypochondriasis, while vertigo, fainting, disturbances of sight and hearing, irregular pupils, hiccough, cramps, and convulsions are said to be caused by the tape-worm, and often disappear when the parasite is removed. The importance of the idiosyncrasy of the patient in accounting for the severity of the symptoms is obvious from the facts of their limitation to persons of sensitive nervous temperament, their customary origin after the discovery of the tape-worm, and their presence in persons free from the parasite.

The *Bothriocephalus latus*, in particular, has been frequently found in persons showing a marked degree of anæmia. Palpitation, dyspnœa, loss of flesh and strength, and perhaps fever, may be so severe as to confine the patient to bed, and retinal hemorrhages and dropsy may be present. The resemblance of these symptoms to those of progressive pernicious anæmia is intimate, and in certain cases they are relieved by the expulsion of the parasite, while in others improvement does not follow this result. The question is, therefore, still undecided as to the significance of the *Bothriocephalus* in the production of the associated anæmia.

The discovery of the tape-worm is usually made by the observation of segments in the stools, although they may escape from the bowel at other times than during defecation, and then attract attention by the associated itching near the anus, or by the sensation of a smooth and slippery body upon the skin of the buttocks or thigh. They have escaped through the abdominal wall from intestinal fistulæ, have been voided with the urine in cases of vesico-intestinal fistulæ, and have been vomited. The tape-worm may exist for years (thirty-five years in one instance being reported), and the passage of segments may be observed only at rare intervals, while their evacuation is said to be promoted by a diet containing fruit and salted, pickled, or spiced articles of food.

DIAGNOSIS.—The presence of the tape-worm in the intestine is to be recognized only by the discovery of the segments or of the eggs, and their evacuation may be promoted by the use of a brisk cathartic. The segments of the pork-worm are generally more intimately mixed with the fæces than are those of the beef-worm, which, being more numerous, are more likely to escape at other times than during defecation. The proglottides of the *Bothriocephalus* are usually discharged as a coherent band of considerable length. The difference between the segments of the *Tænia solium* and of the *Tænia saginata* is readily appreciated when they are compressed between glass plates. The former are more transparent, and the uterus has about ten lateral branches, while that of the latter has in the vicinity of eighteen. The segments of the *Bothriocephalus* are short and broad, in the middle of which the uterus forms a rosette.

PROGNOSIS.—Tape-worms are rarely dangerous to their host, the *Tænia saginata*, or beef-worm, being the least harmful. The *Tænia solium* may become dangerous if its mature segments enter the stomach during a reversal of intestinal peristalsis and become digested, since the embryos are then set free and may become cysticerci. The *Bothriocephalus latus* may prove a source of profound anæmia.

TREATMENT.—The chief drugs which are used against the tape-worm are pumpkin seed, the oleoresin of male fern, pomegranate rind and its alkaloids, pelletierine and isopelletierine, kousso and its active principle, tæniin or koussin, turpentine, and thymol. Whatever drug be selected, it is necessary to see that the intestinal canal is as free as may be from contents which should protect the worm. The patient should take a brisk cathartic thirty-six hours before the anthelmintic, be put on milk diet for twenty-four hours, and left entirely without food during the morning of exhibition. We have usually employed pumpkin seed (pepo). Two ounces of it may be made up in an electuary, with sugar and aromatics. Having on Sunday night taken a cathartic and on Monday no food but milk, and none of that after six o'clock in the evening, the patient on Tuesday morning should breakfast on the pumpkin seed confection, with, if desired, a cup of coffee. Three hours subsequently half an ounce to an ounce of castor oil, with two drachms of oil of turpentine, should be taken. If the subject be feeble, the turpentine may be omitted. Purging will usually come on in two or three hours, and at this time about a quart of saturated watery solution of ordinary salt should be thrown into the large intestine, so as to aid in the expulsion of the worm. In a robust, obstinate case one-half to one drachm of the oleoresin of fern may be taken two hours after the ingestion of the pumpkin seed, and followed in two hours by castor oil.

Pomegranate rind is a very efficient vermifuge: bark which is in small thin quills is believed to be more active than the larger pieces. The decoction may be made by boiling two ounces of the bruised drug after maceration for twenty-four hours in two pints of water to one pint. A wineglass of this is to be taken every half-hour until the whole has been taken or violent purging has been produced. If purging do not occur, the last dose should be followed shortly by castor oil. The alkaloids of pomegranate are chiefly used in the form of a tannate: as put on the market by Tanret, their discoverer, each bottle contains one dose, about five grains. The dose of pelletierine tannate as furnished by Merck is set down at from eight to twenty-four grains in an ounce of water, to be followed in an hour by a brisk cathartic. Tæniin is stated by European writers to be very efficient given in doses of twenty to forty grains, followed in two hours by a cathartic.

Of the tænicides just mentioned, pepo is, so far as known, harmless to man. The oleoresin of male fern has in several cases caused death, with symptoms of violent vomiting and purging, attended by failing strength, stupor deepening into coma, motor excitement amounting, it may be, to violent tetanic convulsions, and in the end collapse. A fatal result is said to have been produced by a little over a drachm. Six drachms have in several cases caused death in the adult. Pelletierine, being a nerve paralyzant, is probably capable of taking life, though no cases of serious poisoning by it appear to be on record: we have seen a startling general paralysis follow the use of the French preparation in a feeble woman.

CYSTICERCUS DISEASE.

This affection is due to the presence in the body of the *Cysticercus cellulosæ*, the larval stage of the pork tape-worm, resulting from the entrance of the ova of the *Tænia solium* into the stomach; the cysticercus of the beef tape-worm and that of the fish tape-worm have rarely, if ever, been found in man. These eggs are usually derived from the tape-worm of another host, although it is possible, as has already been stated, that with the entrance of intestinal contents into the stomach the ripe segments of the harbored tape-worm may be admitted and their ova set free. In children, in persons of uncleanly habits, and in the insane, who may be hosts of a tape-worm, it is possible that an auto-infection may take place by the manual transfer of the ova from the anus to the mouth. In most cases, however, they are derived from the tape-worm of another host, Huber stating that in thirty persons only has the combination of cysticercus and tape-worm been observed. The cysticerci occur oftener in men than in women, usually in middle life, although they have been observed at all ages, even, according to Dressel, in the infant a few days old, in which case the ovum must have entered the fœtus. When the embryo, armed with hooklets, is set free by the digestion of its capsule by the fluids of the stomach. it bores into the wall of the alimentary canal and enters the lymphatics or blood-vessels, Leuckart having found them in the portal blood, and is transferred to various parts of the body. As the embryo reaches its resting-place its hooklets are shed, it increases in size, and is transformed into a cyst containing a clear fluid, which is usually as large as a pea, but may become of the size of a pigeon's egg, provided it has room enough in which to grow. An opaque spot on its wall indicates the seat of an inverted portion, at the bottom of which in the course of three months is developed a head with suckers and hooklets, which may be everted by pressure upon the cyst. The cysticercus usually lies in a fibrous capsule formed from the surrounding connective tissue, but in spaces of large size, as cerebral ventricles and the interior of the eye, it may be free, and Zenker observed one lying in a small aneurism at the base of the brain. It may also be free in the large lymph-spaces of the cerebral pia mater, in which it becomes flattened and irregularly lobulated in consequence of the structure of the communicating spaces in which it is contained. Such a cysticercus is called racemose. The cysticercus once lodged usually remains fixed. although when in the eye movements of its head have been observed with the ophthalmoscope, and it is considered that migration is possible provided there is no mechanical obstruction, as in a large space. It may live for years, and one has been under observation in the eye for twenty years. The cysticercus may occur alone or be present in numbers. especially in the muscles and in the subcutaneous tissue. Bonhomme having counted two thousand in the subcutaneous tissue and nine hundred in the muscles of a patient seventy-seven years old. According to Dressel. the organ in which they are most often found is the brain, usually in the membranes and cortex, especially in the fissure of Sylvius. They are also relatively frequent in the eye, Von Graefe having observed eighty cases, and they have been observed in the heart, lungs, liver, kidneys, and bones.

SYMPTOMS.—There may be numerous cysticerci in the body and no resulting disturbance, or a single cysticercus may give rise to the severest symptoms. Of especial importance is the organ in which the parasite lies and the part of the organ it occupies : hence invasion of the brain and cord is more likely to produce disturbance than of the skin and muscles, although in the former there may be but a single cyst and in the latter innumerable cysts may be present. In animals the sudden entrance of large numbers of embryos is associated with fever, prostration, pain, and diarrhœa, and death may soon follow, in which event the heart and muscles have been found speckled with minute cysticerci resembling tubercles. The cerebral cysticerci may cause chronic meningitis and hydrocephalus, and thus may prove a source of headache, dizziness, mental and physical prostration, convulsions or paralyses; while the cysticercus in the eye occasions disturbance of vision. Although numerous cysticerci may be found in the muscles without resulting disturbance, the observation of animals suggests that contraction of the muscles may become difficult and painful; indeed, Osler describes the case of a patient in whom difficult locomotion, soreness and stiffness of the muscles, numbness, and tingling were attributed to cysticerci in the muscles and skin, since they were found in numbers beneath the latter. Cysticerci of the skin are manifested by subcutaneous tumors, either solitary or in large numbers, smooth, round, and elastic, perhaps sensitive, not projecting above the surface, sometimes as large as a walnut. They may produce numbness and pain by pressure upon the peripheral nerves, and have caused a diagnosis of neuritis. Cysticerci of the heart usually give rise to no symptoms, although when seated in the wall palpitation, dyspnœa, angina, and dropsy may result, while pedunculate cysticerci within the cavity of the head may cause valvular obstruction or incompetency, or be torn off and produce embolism. The cysticerci in other organs usually produce little or no disturbance, the larvæ dying and becoming calcified. A bacterial infection of the fibrous capsule may occur and an abscess result.

Diagnosis.—The presence of the cysticercus is recognized by the discovery of the parasite, which takes place when it is seen in the eye or found in a tumor removed from the skin or muscle.

Prognosis and Treatment.—Cysticerci may be inconvenient in the muscles or skin, but are a source of danger only when in the heart and brain. In the eye they are usually productive of local disturbance alone. The only radical treatment is surgical removal.

ECHINOCOCCUS DISEASE. HYDATID DISEASE.

The echinococcus, or hydatid, is the larva of the *Tænia echinococcus*, a tape-worm of the dog, wolf, jackal, and fox, and is rarely, if ever, found in man, although its presence in the eye, presumably directly transferred by a dog's tongue, has been recently recorded. This worm easily escapes notice, as it is only four or five millimetres long, and appears as a small white thread. The head has four suckers and from twenty to thirty booklets in a double row, while there are but three or four joints attached, only the last of which contains productive eggs. When the eggs are swallowed by man or certain of the lower animals, either wild or domesticated, the embryos are set free, make their way into the lymphatics and blood-vessels of the intestinal wall, and by means of the circulation are carried to various parts of the body, in which they become transformed into the echinococcus cyst or hydatid. Their usual seat is the liver, and they are also to be found in the mesenteric glands and the peritoneal cavity; more rarely, if the general circulation is entered, through either the hepatic vein or the thoracic duct, the hydatids are to be found in the lungs, heart, brain, spleen, uro-genital apparatus, muscles, or bones. The disease abounds in countries, as Australia and Iceland, in which dogs are numerous. It is also frequent in various parts of Europe, and Osler has collected evidence of eighty-five cases in the United States and Canada. It prevails especially among those of uncleanly habits who are intimately associated with the dog, as children and shepherds. In Iceland about one-eighth of the population suffer from hydatids, and eight per cent. of the patients are children. The symptoms of the disease usually appear during the middle third of life, the sexes being affected with equal frequency.

MORBID ANATOMY.—After the embryo becomes lodged at any particular part of the body it is gradually transformed into a cyst. One or many cysts may be found, according to the number of embryos admitted into the circulation; they are unilocular or multilocular, the latter variety being found almost exclusively in the liver, although in rare instances it has been seen elsewhere. In the course of four or five months after lodgement the cyst may become as big as a walnut, and it may eventually form a tumor larger than a child's head, lying free within a capsule formed from the fibrous tissue of the body. These cysts are either sterile, acephalocysts, a variety rare in man, or reproductive of successive generations of cysts. The wall of the maternal cyst is lamellated, and from the inner or parenchymatous layer hollow buds, daughter cysts, project into the cavity, and from the interiors of these buds heads, or scolices, provided with suckers and hooklets, arise by the inversion of a portion of the wall. This method of growth is called endogenous, in contradistinction to the production of buds or sprouts from the outer surface of the wall, the exogenous growth which occurs in the echinococci of cattle and swine. The daughter cysts may become detached from the parent cyst and float within its cavity, or when in large numbers so fill the parent cyst as to leave but little room for fluid, while the scolices may be formed by the thousand.

The multilocular echinococcus appears as an irregular mass proceeding from the liver, and may develop into a tumor as large as a child's head. It is traversed by fibrous septa enclosing alveoli in which are collections of transparent gelatinous material, sterile cysts. This variety was formerly regarded as alveolar cancer, but Virchow discovered that the tumor was due to the echinococcus. It is considered probable that the peculiar appearance of the parasite is due to the growth of the cyst in lymph-vessels, blood-vessels, or bile-ducts, along the branches of which it is continued.

The contents of the cyst are a clear, pale-yellow fluid of neutral reaction and a specific gravity of 1005 to 1015, not coagulating with heat. Chlorides are abundant. There may be a trace of sugar, and the presence of succinic acid is shown by the production of a brown color on the addition of a dilute solution of ferric chloride. That the fluid is toxic is evident from the symptoms which follow rupture, and Brieger has found a toxin which rapidly destroys mice. The echinococcus may die either from injury or from acute or chronic inflammation of its capsule, and the presence of bile may kill the liver echinococcus. After its death more or less fluid is absorbed, and the wall becomes corrugated and encloses a grease in which fat-drops, fat-crystals, and hooklets are to be found. On the inner surface of the fibrous capsule of the echinococcus of the liver crystals of hæmatoidin or bilirubin are often found. The shrivelled dead echinococcus may become infiltrated with lime salts and form a concretion in which hooklets may be seen after removal of the lime. The more numerous and the larger the echinococcus cysts the more likely is the occurrence of atrophy of the cells of the organ in which the parasites lie. According to Neisser, the relative frequency of echinococci in the various organs is as follows: liver fifty per cent., kidneys eight and nine-tenths per cent., cranium seven and five-tenths per cent., lungs seven and four-tenths per cent., female genitals and mammæ four and nine-tenths per cent., pelvis four per cent., circulatory apparatus three and two-tenths per cent., spleen three per cent., face, orbit, and mouth two and three-tenths per cent., spinal canal one and nine-tenths per cent. Usually but one organ is affected, although hundreds may be found in the peritoneal cavity, and their presence in numbers is probably due to the simultaneous entrance of many embryos or to their multiple escape from the rupture of productive secondary cysts.

Symptoms.—The invasion of the embryos often causes no known symptoms, probably from the rarity of a manifold infection, which in dogs produces symptoms resembling those of rabies. The echinococcus cyst may give rise to no disturbance, it having been found after death of such size as must have been the result of years of growth, although its presence during life had been wholly unsuspected. When symptoms occur, they are due to the pressure of the cyst upon surrounding parts, to its rupture, or to the suppuration of its capsule, and their severity varies largely in accordance with the size and seat of the cyst and the organ concerned. The large hydatid of the liver may produce but little disturbance, while a small cyst in the brain may prove rapidly fatal, and cysts pressing on the spinal cord cause paralysis, while hydatids of bone have led to spontaneous fracture. When perforation of the capsule takes place, the contents of the cyst may escape into the alimentary canal, into the uro-genital tract, into the bronchi, or through the skin. Perforation may also take place into the serous cavities, with the production of severe disturbances, dependent upon the toxic nature of the fluid, or into the blood-vessels, and cause death from embolism or toxæmia. Suppuration of the fibrous capsule of the cyst results in the formation of abscesses, which are manifested by chills, fever, localized pain, progressive emaciation, debility, and perhaps jaundice. Death from pyæmia or septicæmia is the frequent result.

These general symptoms are associated with the presence of a tumor sometimes larger than a man's head, flat on percussion, and, when tangible, usually sharply defined, rounded, smooth, elastic, fluctuating, and sometimes presenting a thrill suggestive of quivering jelly. This hydatid thrill is of no pathognomonic importance, since it is frequently absent, and since a similar sensation is sometimes obtained from ascitic fluid or from the contents of an ovarian cyst. Especial symptoms due to the presence of the echinococcus in any particular part of the body will be mentioned in the consideration of the regional distribution of the parasite.

Localization of the Echinococcus.

Echinococci of the Nervous System produce symptoms such as are caused by similarly located tumors.

Echinococci of the Heart.—Echinococci of the heart are rare. The cysts project either from the surface of the heart or into the cavities, more frequently of the right side, and either produce no symptoms, or cause sudden death from embolism, either by detachment of the cyst or by its rupture and escape of the contents.

Echinococci of the Lungs and Pleura.—Echinococci of the lungs produce no symptoms until they attain a size sufficient to cause compression of the lung or perforation of a bronchus, when inflammation, gangrene, and empyema may arise. These results also follow perforation of the diaphragm and lung by an echinococcus of the liver or kidney. When the cyst ruptures into the pleural cavity, there are sudden pain and dyspnœa, and sometimes urticaria; pleurisy, perhaps empyema, is the constant outcome. Perforation of the pulmonary vessels has led to embolism and fatal hemorrhage. The growth of the pulmonary echinococcus may be manifested by cough, pain from associated pleurisy, dyspnœa, fever, and emaciation, symptoms suggestive of phthisis. When the echinococcus is in the upper lobe there may be hæmoptysis and signs of consolidation, yet the nutrition of the patient remains undisturbed. The absence of characteristic bacilli becomes important in differential diagnosis, although tuberculosis and echinococcus may coexist. When echinococci develop in the pleural cavity the symptoms resemble those of hydrothorax, and there are displacement of the heart and diaphragm and retraction of the lung corresponding to the size of the cyst.

Echinococcus of the Liver.—When hydatids of the liver are of a size sufficient to produce symptoms, enlargement of the organ results, either local or general, and the cyst may project from the surface as a rounded or pedunculate tumor, or the boundaries of the liver may extend from the second rib to the crest of the ilium. Single echinococci are more often found in the right than in the left lobe. A sensation of weight and pressure in the epigastrium and right hypochondrium may be present, and the upward displacement of the diaphragm may cause dislocation of the heart, compression of the lung, and dyspnœa. Pressure upon the portal vein produces ascites, while, if the hepatic vein or the inferior vena cava is compressed, œdema of the legs results. The larger bile-ducts may become obstructed and jaundice follow. Rupture of the cyst may take place into the pleural, pericardial, or peritoneal cavity, often producing serious, if not fatal, acute inflammation. If the pleural cavity has been previously obliterated and rupture into the lung occurs, pneumonia is likely to follow, perhaps terminating in abscess or gangrene, with expectoration of pus or blood and bilirubin crystals, and the patient may cough for months and even recover after ejecting cysts, scolices, or hooklets. Perforation into the gall-bladder has been followed by symptoms suggestive of gall-stones, and perforation into the stomach or intestine has led to the discharge of cysts through the mouth or anus. Rupture of the cyst into the hepatic vein or the inferior vena cava has caused immediate death by hydatid embolism of the heart and pulmonary artery. Cysts have escaped with the urine when perforation has taken place into the renal pelvis, and hydatids have been discharged through a fistulous opening in the abdominal wall. When the cyst suppurates, the characteristics of an hepatic or a subphrenic abscess are produced, and the abscess, like the hydatid, may be evacuated into the regions above mentioned.

The enlargement of the liver may be suggested by a bulging of the epigastrium or right hypochondrium. The area of hepatic dulness is increased in proportion to the size and situation of the hydatid. The upper line of thoracic dulness is likely to be convex, its highest point in the axillary region, and when the echinococcus cyst is largely developed in the subphrenic region the descent of the liver on inspiration is checked.

The multilocular echinococcus is almost exclusively limited to the liver, and is of very rare occurrence; when sufficiently large, it is likely to present the symptoms of fibrous hepatitis, such as gastro-intestinal hemorrhages, ascites, and perhaps jaundice, while enlargement of the liver and of the spleen is conspicuous.

Echinococcus of the liver is to be diagnosticated by the recognition of the enlargement, usually circumscribed, of this organ, and the determination of its cause by means of the aspirator. For a long time the strength and nutrition of the patient are well preserved: hence amyloid degeneration and cancer are easily excluded, and the persistent jaundice of hypertrophic cirrhosis is lacking. An echinococcus projecting from the upper surface of the liver may simulate a pleuritic exudation, but the highest point of dulness from the latter is in the dorsal and not in the axillary region. Suppurating hydatids may be confounded with simple cyst of the liver, or with a subphrenic abscess, since the symptoms are the same and the physical manifestations may be identical. Dilated gall-bladder, cancer, hydronephrosis, and cystic kidney are excluded by examination of the aspirated fluid.

Echinococcus of the Kidney.—The parasite occurs more often in the left kidney, and its growth tends to produce a cystic tumor sometimes of large size. With the increase in the size of the cyst the kidney becomes atrophied, fatty degeneration of the epithelium with increase of the fibrous tissue occurs, and pigment-granules from extravasated blood are often found in the vicinity of the cyst. Adhesions are likely to form between the peritoneal covering of the cyst and that of the spleen, liver, or intestine, and compensatory hypertrophy of the other kidney is frequently associated. The general health is unaffected, and especial symptoms are usually delayed until perforation of the wall of the cyst, with escape of the contents, takes place. The renal pelvis is oftenest perforated, but the cyst may rupture into the stomach, the intestine, or the peritoneal cavity, or through the diaphragm into the lungs. Perforation into the renal pelvis is followed by the escape in the urine of few or many cysts, perhaps at intervals, during months or years, the passage of the cysts being manifested by attacks of renal colic, and followed by retention of urine, albuminuria, or hæmaturia, and symptoms of pyelitis or cystitis. The physical characteristics of the tumor resemble those of hydronephrosis, and the nature of the contents is to be determined after their withdrawal by means of the aspirator. The parasite may be present in the kidney for thirty years, and, although rupture into the renal pelvis takes place in three-fourths of the cases, spontaneous recovery due to the death of the parasite can occur. The physical examination of the tumor gives evidence of its cystic nature, and its renal origin is determined by its position behind the colon and by its immobility.

Echinococci of the Peritoneum.—The echinococcus may lie free in the peritoneal cavity, but it is more commonly situated in the subperitoneal tissue, especially in the omentum and mesentery and in the wall of the pelvis. Hundreds of cysts may be present, resulting in abdominal tumors of large size. The growth is gradual, and usually without symptoms until the movements of the diaphragm are interfered with, when respiration is disturbed, or the stomach and bowels are compressed or united by adhesions, with corresponding impairment of function. Rupture of the cyst and escape of the fluid into the peritoneal cavity are followed by the results already stated. Childbirth has been delayed and retention of urine produced when echinococci were in the pelvis, while extensive suppuration and death from septicæmia have followed perforation into the intestine or the vagina, although the passage of peritoneal echinococci into the hollow organs is rare. The physical examination of the enlarged abdomen is indicative of the presence of fluid, while the gradual enlargement and absence of symptoms are suggestive of the presence of an ovarian or a parovarian cyst. The pelvis echinococcus rarely produces such large tumors as arise from the ovary or the parovarium. When the cysts are omental or mesenteric the limitation of the tumor at the outset to the upper half of the abdomen may suggest a cyst of the pancreas, but the growth of the latter is usually preceded by striking symptoms, and its presence is frequently associated with disturbance of digestion and discomfort. The characteristics of the aspirated fluid are sufficiently marked to establish the diagnosis.

DIAGNOSIS.—The diagnosis ultimately depends upon the recognition of the tumor, which is of slow growth, usually painless, and generally without disturbance of nutrition. Its physical characteristics have already been described. More important is the determination of the nature of its contents, which are obtained by means of the aspirator. If the removed fluid is free from albumin, and contains sugar and succinic acid, or scolices, hooklets, or lamellated membrane, its echinococcal origin is obvious. Exploratory puncture, however, may prove dangerous by permitting leakage of the contents of the cyst into a serous cavity, in which case urticaria, dyspnœa, collapse, and fever may result. Aspiration should, therefore, be merely preliminary to treatment in case of doubt.

PROGNOSIS.—Although the conditions connected with its growth may promote the death or the evacuation of the parasite, they are more likely to threaten the life of the patient: hence with the establishment of the diagnosis the serious nature of the disease should be made conspicuous.

TREATMENT.—Whenever the cyst becomes a source of discomfort its treatment by aspiration or removal becomes necessary. Aspiration has been frequently followed by complete cure, but is somewhat dangerous, and may prove ineffectual in retarding the growth of the cyst. Of late years cysts and complicating abscesses have been repeatedly opened, evacuated, and drained, with, as a rule, a favorable result.

FLUKES.

Of the trematoid worms dangerous to man those of especial importance are the blood-flukes, lung-flukes, and liver-flukes: the other varieties are of such rare occurrence as to require mention merely by name. Such are the *Distoma lanceolatum*, *Distoma crassum*, *Distoma heterophyes*, *Distoma ophthalmobium*, *Distoma sinense*, *Distoma conjunctum*, and the *Monostoma lentis*.

The *Distoma hæmatobium*, or blood-fluke, was discovered by Bilharz and Griesinger in the portal system and in the recto-vesical plexus. The female is sixteen to eighteen millimetres long, the male is some four millimetres less in length. The eggs are present as small white specks in the liver, in the intestinal wall, and especially in the urinary tract. This fluke prevails in various parts of Africa, particularly in Egypt, in which one-fourth of the native population, notably the poor and the children, are stated to harbor the parasite. It is supposed to live in the water of the Nile, and Europeans who use the filtered water rarely become diseased. The presence of the parasites in the mucous membrane of the bladder and ureters causes a hemorrhagic inflammation of the mucous membrane, within and upon which the eggs are to be found, having escaped from the blood-vessels. Necrotic patches infiltrated with urinary salts are to be seen upon the surface of the membrane, and pyelitis and nephritis may be associated. Rectal and vesical tenesmus, painful micturition, intermittent or persistent hæmaturia increased on exertion, hypogastric tenderness, progressive anæmia, and loss of flesh and strength result.

DIAGNOSIS.—The diagnosis is based upon the discovery of the eggs of the parasite, which are present in large quantities, chiefly in the blood-clots and slime in the sediment of the urine of persons suffering from cystitis and hæmaturia in the regions in which the parasites are found.

The disease, distomiasis, may last for years without serious disturbance, the persons affected even being strong and vigorous, and the death of the parasite may occur with relief to the symptoms, especially after removal to a non-infected region. The severer manifestations arise among those exposed to repeated infection, and the course of the disease is that of an incurable cystitis or pyelitis, death resulting from uræmia or amyloid disease.

The *Distoma pulmonale*, or lung-fluke, according to Baelz and Manson, is frequently found in the bronchi of the natives of China, Japan, Corea, and Formosa. The parasite is probably derived from the drinking-water, and causes a cough with occasional hæmoptysis, the diagnosis being established by the discovery of the eggs in the sputum. The parasite and eggs have been found also in the brain, liver, subperitoneal tissue, and intestinal contents.

The *Distoma hepaticum*, or liver-fluke, is a flattened fluke of elliptical outline, about thirty millimetres in length, its greatest width being twelve millimetres: it is provided with two suckers. This fluke is very common in the bile-ducts of ruminants, but is rarely found in man, in whom it sometimes gives rise to jaundice and dropsy.

In Japan, according to Baelz, the liver-fluke is productive among the natives of serious disease, essentially a chronic inflammation of the bile-ducts; but the parasite has been found both in the subcutaneous tissue and in the liver. The embryos develop in snails, from which they are freed in the stomach of the host, and then wander through the bile-ducts into the liver, where hundreds may be present in the dilated bile-ducts. The liver is enlarged, its fibrous tissue is increased, and the liver-cells are atrophied. There is also enlargement of the spleen. The alterations of the liver are associated with a sense of pressure in the epigastrium, but progress for years with but little pain or general disturbance, and jaundice is usually absent. Eventually diarrhœa, intestinal hemorrhage, and ascites may result and death follow.

The diagnosis is to be made by the discovery of eggs in the dejections.

Medical treatment is only palliative, and consists in meeting symptoms as they arise.

LEECHES.

Of the anellides or leeches there are two varieties, the *Hirudo ceylonica* and the *Hirudo vorax*, which are important as human parasites. The former is a land leech found in Ceylon, the Philippine Islands, Australia, and Chili. It attaches itself to the skin, from which, if adherent, it must be removed with care, since if torn off a portion of the jaw is left and causes chronic suppuration. The latter is a water leech found in Europe and North Africa. The young when swallowed may prove dangerous by causing hemorrhages and chronic disease of the larynx and trachea, which they may enter.

ROUND AND THREAD WORMS.

Of the nematoid worms parasitic in man one of the most common is the *Ascaris lumbricoides*, which somewhat resembles the earth-worm, and is pointed at both ends, of a yellowish color, the female being ten or twelve inches long and the male two to four inches less in length. The eggs are about six-hundredths of a millimetre long, and may be found in large numbers in the fæces, the female, according to Eschricht, annually producing in the vicinity of sixty millions. The embryos develop in warm and moist surroundings in the course of a few weeks or months. According to Grassi, twenty days after the embryos were swallowed by a child examination of the fæces was negative, but in two months numerous eggs were present, and in three months one hundred and forty-three worms were expelled.

The ascaris is the most common verminous parasite of man, the symptoms resulting from its presence receiving the term *ascariasis*. It is to be found at all ages, in both sexes, in all countries, and among all races, and is more frequently observed in children living in the country. The eggs are probably largely swallowed in drinking-water or with contaminated food, especially during summer. This worm lives in the small intestine, and the parasites may be present in large numbers, thousands having been expelled within a short time. They may wander from the small intestine to other parts of the alimentary canal, or through fistulous openings into other parts of the body. They generally survive the death of their host, after which they may migrate from the ileum. They are usually of but little symptomatic importance, but when numerous may produce loss of appetite, nausea, irregular movements of the bowels, and abdominal discomfort. In children, especially nervous children, restlessness, irritability, picking at the nose, grinding of teeth, and disturbed sleep are attributed to the worms, and are frequently relieved after their removal. In rare instances they may form a mass causing a palpable tumor and producing intestinal obstruction. The migration of the worms becomes increased in severe febrile diseases, and in those characterized by frequent movements of the bowels, as typhoid fever, dysentery, and cholera. The wandering worms may enter the common duct and pass into the gall-bladder or into the hepatic ducts ; in the latter case abscesses of the liver may result. Vomiting may follow their appearance in the stomach and the parasites be expelled. According to Davaine, the ascaris has entered the Eustachian tube and the lachrymal canal, and has frequently crawled out of the mouth, nose, or anus of a sleeping child. Dyspnœa, aphonia, immediate death from asphyxia, or a fatal bronchitis has resulted from its entrance into the respiratory tract. When discovered in the peritoneal cavity, a perforation should be sought for, either gastric, duodenal, appendicular, or typhoidal, through which it has passed, it being doubtful whether it can cause perforation of the intestine except a necrosis of the wall already exists. Migration through fistulous tracts between the intestine, pelvis of the kidney, bladder, female genital tract, and skin occurs. Usually the presence of the ascarides is first recognized after their escape from the bowel, although the examination of the fæces may lead to the discovery of the eggs. They are readily removed by appropriate treatment.

TREATMENT.—No special diet or preparation is necessary in the use of remedies against the round worm. The number of vermifuges is quite large: those of the first rank are santonin, spigelia, oil of chenopodium, and turpentine. Of these santonin is probably the most effective. As the object is to bring it in contact with the worm and prevent absorption, the santonate should never be used, nor should the santonin be finely powdered. The official troches of santonin are distinctly inferior to lozenges made of the minutely crystalline, unpowdered drug. The best method of administration is, however, to give the santonin as it naturally occurs, along with calomel (two to six grains), in thick capsules, administered at bedtime, a brisk cathartic being exhibited in the morning if free purgation does not occur on waking. The dose of santonin for a child two years old is one-fourth to one-half grain.

The fluid extract of spigelia and senna, formerly official, was a pleasant and effective vermifuge, especially in the case of children: dose for a child a year old, from forty-four to four hundred and forty minims, repeated every four hours till it purges: to a child a year old, two drops of oil of chenopodium may be given three times a day for two days, and then repeated with a dose of castor oil.

Oxyuris vermicularis.—The female thread-worm or pin-worm is ten to twelve millimetres long, and is about three times the length of the male. The eggs are five-hundredths of a millimetre long, and are produced in abundance, from ten thousand to twelve thousand, according to Leuckart, being present in the female. When the eggs are taken into the stomach the embryos are set free by the digestive fluids, and so rapidly develop in the intestine that in the course of a fortnight the young worm has been found in the stools. These worms abound in the large intestine, from which as the eggs mature the parasite wanders to the rectum, discharges its eggs, and often passes out of the anus, in the vicinity of which eggs may be found. The pin-worm is one of the commonest of the intestinal parasites, and has been found at all ages, even in an infant of five weeks, though most frequently observed in children. The eggs are small enough to be blown about when dry, and are oftenest swallowed in the summer, probably with fruit or vegetables, since soaking in water destroys them. Persons of uncleanly habits may easily infect themselves, since eggs have frequently been found in the vicinity of the anus and beneath the finger-nails. The especial symptom is itching at the anus after the patient goes to bed, due to the movements of the female worm in the rectum. The annoyance is worse immediately after defecation: the persistent discomfort disturbs sleep, and in sensitive children may cause convulsions. The local irritation may serve as a cause of masturbation, and the parasite may wander into the vagina and occasion itching and leucorrhœa. The worm has also been found in the bladder, stomach, œsophagus, and mouth. In nervous persons the suffering is so considerable and sleep so disturbed that the patient may lose flesh and strength and become anæmic.

The diagnosis is easily made by the discovery of the worms in the fæces, in which they may be found in masses entangled in the slime, or they may be seen on eversion of the anus, which is reddened and excoriated, or may be expelled in numbers after the use of an enema.

TREATMENT.—There is no use in the administration of medicines by the mouth for the destruction of seat-worms. They must be reached by injections into the rectum and the lower colon; saturated solution of salt will sometimes do the work, but the most effective and harmless remedy is the decoction of quassia, made by boiling two quarts of water with two ounces of quassia chips to a quart in an earthen vessel. A large salt-water injection is to be given so as to empty the lower colon, and, after it has come away, the whole or a part of the quassia decoction, according to the age of the patient, is to be injected into the upper rectum. If it does not come away in fifteen minutes, saturated salt solution or other irritant injection should be used.

The *Eustrongylus gigas*, of which the female may be three feet long and the male one foot long, found in the renal pelvis and ureters of many animals, has in rare instances been found in the urinary tract of man.

The *Strongylus longivaginatus* has been found in the lungs of a child, and may prove a possible cause of inflammation of the lung.

Ankylostoma duodenale.—This parasite forms a white thread-like worm, the females being some fifteen millimetres in length, the males perhaps one-third shorter, and produces the disease *ankylostomiasis* or *dochmiasis.* The eggs are produced in great abundance, rapidly ripen under favorable conditions, and are six-hundredths of a millimetre long and four-hundredths of a millimetre broad. The embryo escapes, becomes encapsulated, and is set free from the capsule when swallowed by the host. The parasite lives in the upper part of the small intestine, especially between the folds of mucous membrane, to which it clings by hooklets, and which becomes thickened and pigmented from numerous hemorrhages. In fatal cases the lesions are those found in extreme anæmia.

This parasite was recognized by Bilharz and Griesinger as the cause of the severe Egyptian chlorosis, and Wucherer found it to occasion tropical chlorosis in Brazil. Its presence has been recognized in India and Japan, and also in the southern United States. It had been considered to produce the anæmia among workers in brick-yards in Italy, and it proved to be the source of the extreme anæmia affecting the Italians working in the St. Gothard tunnel. It is also recognized as the important factor in the etiology of anæmia among workers in tunnels, mines, and brick-yards elsewhere, the infection probably taking place from the presence of embryos in the drinking-water. The severity of the symptoms depends upon the number of parasites present, and the disturbances begin with the moving about of the worms. Loss of appetite, nausea, vomiting, or abdominal pain may be present. The more serious derangements are dependent upon the progress of the anæmia due to the withdrawal of blood by the parasites. This is sometimes extremely rapid, and is manifested by pallor, debility, vertigo, palpitation, and eventually œdema and loss of flesh. The anæmic symptoms become more extreme, and the patient presents the features of a pernicious anæmia.

The diagnosis is based upon the discovery of the eggs in the fæces, in which they may be found in the course of six weeks after the invasion of the parasite. Although the disease may rapidly prove fatal, it may also be continued for a period of years, for the life of the parasite may be prolonged for five years or more. Recovery may follow its death in case a new infection is prevented, or a condition of more or less permanent invalidism may result from the complicating chronic diarrhœa or dilated heart.

Ankylostoma responds to anthelmintics as do other intestinal parasites. Sufficient preparation should be made to see that the duodenum is free from contents. Among the anthelmintics the oleoresin of male fern is probably the most efficient. It must be given in large doses, and followed after a time by a brisk cathartic, such as castor oil with oil of turpentine. Federici asserts that thymol is especially poisonous to the worm; it must be used in large repeated doses, five grains every two hours, until twenty grains are taken, when a mixture of castor oil and turpentine can be given.

Trichocephalus dispar.—This worm, the whip-worm, is from four to five centimetres long, lives in the cæcum, usually in small numbers, although hundreds have been found, and is of frequent occurrence in many countries, especially in Syria and Egypt. Its pathological importance is still in question.

Filaria medinensis.—This parasite, the Guinea-worm, of which only the female is known, is some two feet long, very elastic, and prevails in Guinea, Egypt, and India. According to Osler, two cases have arisen in the United States. The disease produced by the presence of this parasite in man is called *dracontiasis*. Usually a single parasite is present, but ten or twelve have been found. The embryos are known to enter a small crustacean, in which they undergo further changes, and it is suspected that this intermediate host is swallowed by man. The usual localization of the parasite in the neighborhood of the feet suggests, however, that the worm may invade the skin. As the result of its presence, a circumscribed, red, painful swelling of the skin arises, in which the convoluted worm may be felt. Ulceration and suppuration occur, and the parasite is sometimes discharged, after which the abscess quickly heals. Unsuccessful attempts at removal result in tearing off a portion of the worm, the remainder of which returns to the subcutaneous tissue and causes a continuance of the inflammation, and numerous embryos appear in the pus. The usual method of removing the worm is to roll the projecting end around a piece of wood, as a match, and to wind two or three times daily the slack portion of the worm.

Filaria sanguinis hominis.—This worm, found by Lewis in a blood-clot and by Bancroft in the lymphatics, was first made known by its embryos, which were found in the urine by Wucherer, in Bahia. The mature worm, which lives in the lymph-vessels, especially in those of the uro-genital apparatus, is hair-like, four to eight centimetres long. Its embryos, about one-third of a millimetre long, and of the thickness of the diameter of a red blood-corpuscle, enter the blood in large numbers through the lymphatics. A drop of blood has been found to contain a dozen or more of the rapidly moving embryos. During the day they are to be found with difficulty, although readily seen at night; but this periodicity may be reversed when the host sleeps by day and works by night. According to Manson, the mosquito is the intermediate host, for he has found the embryos in mosquitoes which have taken blood from a patient with filariæ, and considers it probable that the embryos of the latter are set free by the death of the insect after its eggs are deposited in water. The presence of the mature parasite in the lymphatics becomes known by the production of either hæmatochyluria or elephantiasis, although it is to be recognized that these affections may exist independently of the presence of the filaria. In *hæmatochyluria* the urine from time to time is of a milky white or pink color, according to the quantity of blood present, and a cream-like layer forms when the fluid stands for some time. The milk-like appearance is due to the presence of molecular fat-drops, and filarial embryos have been found in the chylous urine, but are more likely to be seen on examination of the blood at night. Intermitting hæmatochyluria may exist for years with little or no general disturbance. At times it may be associated with pain in the region of the kidneys and with painful micturition from the passage of fibrinous clots.

The presence of the mature filaria in the lymph-vessels causes the series of changes occurring in *elephantiasis* and *lymph-scrotum*. The wall of the lymphatic is inflamed, the flow of lymph is obstructed, the smaller lymph-vessels become dilated and varicose, and the fibrous tissue is thickened and œdematous. In lymph-scrotum a chylous fluid may also be present in the tunica vaginalis, and hæmatochyluria may be present or absent. Embryos have been found in the milky fluid escaping from dilated superficial lymphatics, but, according to Manson, are not readily seen in the blood in elephantiasis, from the obstruction to their passage through the lymphatics caused by the presence of the mature worm.

No treatment has been found efficacious in destroying the filaria.

Other filariæ, of less consequence, are the *Filaria Loa*, which has been found in the subconjunctival tissue of persons in West Africa and South America, the *Filaria lentis*, present in cataract, the *Filaria bronchialis*, observed in the bronchial glands, and the *Filaria labialis*, discovered in the lip.

TRICHINOSIS.

Etiology.—The calcified capsule of the *trichina spiralis* was seen by Hilton in 1832 in the muscle of a corpse. The worm was soon after discovered by Paget and described by Owen, and later was found by Leidy in swine: its pathogenic importance in producing the disease *trichinosis* was first definitely recognized by Von Zenker in 1860. In a week after its entrance into the alimentary canal of its host the embryonic trichina matures into a female from two to four millimetres long, or into a somewhat shorter male, and begins to develop embryos, which continue to be produced for nearly six weeks to the number of fifteen hundred from a single female. (Leuckart.) Some of the embryos perforate the wall of the intestine, and entering the subperitoneal tissue are carried along the mesentery towards the muscles in front of the spine, so that they have been found both in the lymph-glands and in the blood-vessels. Others pass though the peritoneum into its cavity, whence they invade the neighboring muscles and wander into other serous cavities. When the embryo reaches the muscle it enters the primitive fibre, in which in a few days it forms a spiral and in the course of a fortnight becomes encapsulated within the thickened sarcolemma. In man, in about six months the deposition of lime salts takes place in the capsule, and the presence of the muscle-trichina becomes evident by a white speck perhaps one millimetre long. In swine, on the contrary, calcification of the capsule is less likely to occur: hence in them the presence of the muscle-trichina is to be recognized only with the microscope. The encapsulated trichina thus calcified may retain its vitality for twenty-five years, and may live for months after the death of the host. When flesh containing the live muscle-trichinæ, of which there may be millions in a single animal, is eaten by man, swine, rats, cats, rabbits, or guinea-pigs, the larvæ are set free.

The disease trichinosis comprises the disturbances resulting from the migration of the embryos, and is found in those countries in which pork, especially insufficiently cooked or preserved, forms an important article of diet: hence it is common in Northern Germany and in other parts of Europe, in North and South America, in India, China, Africa, and Australia. According to Osler, four hundred and fifty-six cases have been recorded in the United States. Epidemics are frequent from the fact that the flesh of the infected hog is often eaten by a number of persons in a limited locality.

Swine are the principal source of infection, and they are frequently invaded by trichinæ, either from eating the infected flesh of other swine or from feeding on rats which have become infected from swine or from the contamination of their food with productive intestinal trichinæ. The trichinæ abound in the diaphragm and tongue, in which they are readily found with the low power of the microscope,—most conveniently when a bit of the muscle is compressed between glass plates. In Prussia, where a careful search for trichinæ is made in slaughtered hogs, according to Eulenberg's figures the proportion of trichinous hogs to those free from trichinæ was 1 in 2160 in 1876, and 1 in 1817 in 1889. According to Billings, of 8773 American swine examined between 1879 and 1881, 1 in 25 was found to contain trichinæ, although in a series of some 2000 last examined the proportion was 1 to 44. The latter ratio was confirmed by Salmon in 1884, who estimated the percentage of American trichinous swine as two and seven-tenths. Despite this large proportion of infected swine in the United States, epidemics of trichinosis are comparatively rare, due in part to the more thorough cooking of the flesh, in part to the destruction of the muscle-trichinæ by salting the pork and smoking the hams and bacon. According to Virchow, the rare cases of trichinosis in Germany asserted to be derived from imported American swine products are not reported with sufficient accuracy to warrant this assertion. In Germany trichinæ are found in man in about one per cent. of all autopsies. In the United States trichinous subjects are occasionally seen in dissecting-rooms, and more rarely at autopsies.

In the early stages of the disease the mucous membrane of the intestine is injected and Peyer's patches and the mesenteric glands may be swollen. The muscles show various shades of color between a reddish-gray and a dark red. After the fifth week minute gray lines are seen, resulting from the granular destruction of the muscle by the trichinæ, and with the microscope the changes characteristic of an acute myositis are to be found. According to Cohnheim, in cases fatal during the second month the liver contains abundant fat, the epithelium of the kidneys is often granular, at times fatty, the heart-muscle is granular, the bronchi contain viscid secretion, hypostatic pneumonia is frequent, and broncho-pneumonic patches are occasionally found. The spleen is but little altered.

Symptoms.—When insufficiently cooked trichinous pork is eaten by man, the symptoms vary in accordance with the number of living trichinæ introduced. When many muscle-trichinæ enter the alimentary canal, the symptoms of an acute gastroenteritis may occur in the course of a few hours or after two or three days, and nausea, vomiting, diarrhœa, abdominal pain, and prostration be present. On the third or fourth day after eating the infected food there are chills, followed by fever; the temperature reaches its maximum of 103° or 104° F. during the second week, the evening temperature being higher than that of the morning. The fever has a remittent or an intermittent type, lasts from two weeks to two months, its course somewhat resembling that of a mild typhoid fever, and is associated with loss of flesh and strength. The symptoms indicative of the entrance of the trichinæ into the muscles begin during the middle of the second week. There is pain referred to the abdominal muscles and the diaphragm. The superficial muscles become resistant, painful, especially on motion, and tender. When the trichinæ are abundant in the respiratory muscles there is dyspnœa, and bronchitis is associated; if the muscles are attacked the patient is confined to the bed, and the position of the trunk and extremities is that of semiflexion. If the muscles of the jaw are invaded, mastication is difficult, and involvement of the tongue and of the muscles of the pharynx and larynx interferes with swallowing. Œdema of the eyelids and sometimes of the glottis, lasting for several days, accompanies the invasion of the respective muscles, and makes its appearance elsewhere at a later period in the disease. Pruritus and desquamation of the epidermis often occur as the œdema subsides. There is profuse sweating, which, with the fever and the muscular pain, has often suggested acute rheumatism. The intelligence of the patient is unaffected, except in the severest cases, but sleep is usually disturbed. In mild cases the symptoms are slight, and there is often no fever, the pains are ill defined, and muscular weakness is more conspicuous than muscular pain. Trichinosis in children runs a milder course than in adults.

Diagnosis.—In sporadic cases the diagnosis is difficult. The continued fever, diarrhœa, and prostration are suggestive of typhoid fever, but the enlarged spleen is lacking, and the muscular pain and rigidity are not features of the latter disease. The fever, pains aggravated on motion, and profuse sweating suggest acute rheumatism, but the joints are not swollen, and the muscles are tender to the touch, while the initial gastro-intestinal disturbance is lacking in acute rheumatism. Acute polymyositis is also simulated by the muscular rigidity, pain, and tenderness, and the œdema of the skin. This affection is of a progressive character and of longer duration, and lacks the acute digestive disturbances. In the severe cases with conspicuous vomiting and diarrhœa, cholera or cholera morbus is suggested, but muscular cramps and rice-water stools are absent. In acute poisoning by meat, fish, or milk-products, the severe gastro-intestinal symptoms of acute trichinosis are simulated, but the profound disturbance of the nervous system caused by these is lacking. Important in the diagnosis of trichinosis is its occurrence among a number of individuals who have eaten hog-products from the same source at or about the same time, and in whom the continued fever, muscular pain, tenderness, and rigidity, œdema, dyspnœa, and wakefulness occur. The diagnosis is assured by the discovery of trichinæ in the flesh served, and has sometimes been determined by their recognition in a bit of muscle removed from a patient for the purpose of diagnosis.

Prognosis.—The mortality in the various epidemics of trichinosis varies from five per cent. to thirty per cent., depending largely upon the number of trichinæ in the infected meat, the method of preparation of the latter, and the quantity taken. Severe diarrhœa at the outset is a favorable sign, since it permits a considerable removal of the trichinæ from the intestine. The more severe and general the symptoms resulting from the invasion of the muscles, as fever, pain, dyspnœa, the worse the outlook, and the longer the duration of the symptoms the more favorable the prognosis as to life, although complete restoration to health may not take place for months or even years.

Treatment.—There is no known method of sensibly affecting the growth of the trichina once lodged in the human muscle: the treatment must be purely symptomatic and sustaining. If it should become known within forty-eight hours that a person has taken infected meat, the alimentary canal should be thoroughly emptied by an immediately acting cathartic, such as castor oil with turpentine (half a fluidounce to a fluidrachm), followed by quarter-grain doses of calomel every two hours until further purgations occur. With the calomel may be used santonin, the oleoresin of male fern, or thymol, which has been especially recommended by some writers.

DISEASES DUE TO ARTHROPODES.

Of the wingless arthropodes a number of arachnids occur as human parasites. The *Pentastoma denticulatum* is the larval form of the *Pentastoma tænioides*, which is found in the nostrils and communicating sinuses of the dog, horse, goat, and other animals, although rarely in man. The embryos are set free from the eggs when taken into the alimentary canal, and either directly or by means of the blood-current reach the nostrils, liver, spleen, kidneys, heart, and lungs, and become encapsulated. In the course of six months the *Pentastoma denticulatum*, four to five millimetres long, is fully developed, and after two months may perforate the cyst wall and be evacuated. In the nostrils catarrh may be produced and the eggs be found in the secretion, while jaundice and digestive disturbance may follow its presence in the liver. Although in Germany the larval form has often been found in man, serious disturbances from its presence rarely occur.

The larger *Pentastoma constrictum* has repeatedly been found in Egypt, and, according to Osler, this parasite has been ejected from the mouth and with the urine in America.

The *Demodex folliculorum*, or acarus of the sebaceous follicles, is found especially in the skin of the face, but is of little pathogenic importance.

The *Sarcoptes hominis*, or *Acarus scabiei*, the itch-insect, is visible as a minute white-yellowish speck. The male is twenty-three-hundredths of a millimetre in length by sixteen-hundredths of a millimetre in breadth, and the female forty-five-hundredths by thirty-five-hundredths of a millimetre. The latter burrows into the epidermis for a distance of three centimetres, lays some fifty eggs during her progress, and dies at the end of three months. Embryos are sufficiently developed from the eggs in the course of a fortnight to form fresh burrows. The itch-insect abounds upon the hands, especially in the interdigital folds, and may thence spread over the body. It is conveyed from one person to another, especially among those of uncleanly habits living in intimate relation, particularly children. The presence of the burrow is indicated by a small blister or papule, which in the course of a few days subsides and is followed by desquamation. The migration of the parasite produces itching, especially severe at night, and the patient seeks for relief by scratching, causing excoriations, hemorrhage, and exudation, and numerous punctate and linear crusts result, principally on the lower abdomen and the thighs, especially in the vicinity of the penis. In consequence of the scratching, vesicles, papules, and pustules are often present. The diagnosis is based upon the localization of the rash and the discovery of the burrows, and sometimes upon the recognition of the parasite.

In the treatment of itch the parts should be thoroughly washed with a strong watery solution of soft soap, and afterwards with simple warm water ; following this, sulphur ointment should be well rubbed into the part, which should then be covered with a thick application of the ointment. This process should be repeated as often as necessary.

Ixodes ricinus and *Ixodes americanus* are ticks which, though frequent among the lower animals, are only occasionally found on man, and produce little or no local disturbance unless, when torn from the skin, the heads remain and cause a local inflammation. The treatment consists in picking off the tick : if the head remains it may be taken out with a needle.

The *Leptus irritans*, or harvest-mite, a small red parasite, attaches itself sometimes in large numbers to the skin, and causes itching and papular or pustular inflammation.

PARASITIC INSECTS.

Of the parasitic insects the pediculus, or louse, is often found in three varieties, the *Pediculus capitis*, the *Pediculus vestimentorum*, and the *Pediculus pubis*. They vary in size, the *Pediculus vestimentorum* being from two to four millimetres long, while the pubic louse is the smallest, and is about one millimetre long. The head-louse lays its eggs among the hairs, to which they become attached and form the oval white specks known as nits. The bites of the insect cause itching, which is relieved by scratching, and a serous or bloody fluid exudes. The hairs are matted together by the drying of the exudation, and when allowed to grow form an offensive tangled mass, the *plica Polonica* of the Polish Jews. The *Pediculus vestimentorum*, though living in the clothing, especially in the seams of those articles lying close to the skin, causes irritation by its bites, which are most numerous between the shoulder-blades, around the waist, and upon the nates; minute hemorrhages result from the bites, and bleeding, excoriations, crusts, pustules, and ulceration are caused by the scratching. The long-continued harboring of the parasite results in a thickened, pigmented, and scarred skin, known as the *vagabond's disease*, and sometimes mistaken for the pigmented skin of Addison's disease. The pubic louse lays its eggs among the hairs of the symphysis pubis, to which they are attached as minute specks. It may also be conveyed to the axillary hairs, the beard, the eyelashes, and the eyebrows. The irritation from its bites leads to scratching, which may occasion an artificial eczema. The occurrence of bluish spots, *taches bleuâtres*, upon the thorax, thighs, and nates, especially in typhoid fever, was attributed by Mourson to the migration of this parasite. Vincent Y. Bowditch has recently recorded his observation of these spots in a number of affections, and almost invariably was able to find the parasite.

TREATMENT.—When a part covered with hair is infested with lice it is usually better to cut the hair off close, as it is almost impossible to kill all the nits or eggs attached to the hair. When the head is affected the scalp should be thickly covered over with sulphur ointment, or coal oil to which has been added one per cent. of carbolic acid may be freely used. When there are but few of the parasites in the head, saturating the hair with coal oil may suffice. In the case of the pubic louse, after removal of the hair the part may be thickly covered with mercurial ointment, which should be removed by thorough washing with soap and water in about twelve hours, and then reapplied. The mercurial ointment is also extremely efficacious against the pediculus capitis, but the amount necessary to be used is so large as greatly to endanger systemic poisoning; we have seen an almost fatal case of salivation result from the free use of the ointment on the head of a vagabond. It is necessary to enforce thorough personal cleanliness to prevent recurrence. Whenever there is great irritation of the skin from these parasites the warm bath containing one-quarter to one-half pound of ordinary washing-soda may be used. A lotion composed of one ounce of alcohol, one ounce of glycerin, two drachms of carbolic acid, and fourteen ounces of water will often give relief. When lice are abundant upon the person it is always essential to destroy them upon the clothing, but in the case of the clothing louse nothing less radical will suffice than leaving the clothing in a sufficiently heated oven, or boiling it thoroughly for some minutes in water.

Cimex lectularius, the bedbug, four or five millimetres long, is of a reddish-brown color, and has an offensive odor. It lives in the joints of wooden bedsteads and in the cracks of the walls and floors especially of old buildings. At night it sucks blood from sleeping man, and produces an urticaria in sensitive persons.

When there is excessive irritation the alkaline bath or the carbolic acid solution (see preceding page) may be used. It may be necessary to take apart furniture infested with bedbugs when it is not possible to reach thoroughly the crevices and joints. If, however, this can be done, the parasite can be destroyed by thorough cleanliness and the free application of a ten per cent. solution of corrosive sublimate. Iron bedsteads are always preferable.

Pulex irritans, the flea, stings the skin, produces a minute hemorrhage, and may cause rose spots, petechiæ, or urticaria. Some persons are so little susceptible as not to be conscious of its presence.

When it is impossible to avoid exposure to fleas, the free use of insect-powder on the inside of the stockings and other articles of underclothing will afford a measure of protection.

Sarcopsylla penetrans, the sand-flea, chigoe, or jigger, is found in South America, in the West Indies, and on the west coast of Africa, and infests man and some of the domesticated animals. It is smaller than the common flea, burrows into the skin, especially between the toes and under the toe-nails, causes intense itching, with the formation of pustules and ulcers, and may lead to the loss of the toes.

The jigger is to be removed with a minute sharp knife or a needle. A certain amount of protection from its attacks is said to be afforded by the free use of aromatic oils upon the feet.

Myiasis.—This term is applied to the disturbances which result from the presence of maggots in various parts of the body. Numerous flies, the common house-fly, the meat-fly, the bluebottle-fly, the horse-fly, the carrion-fly, and the bot-fly, under favoring circumstances, lay their eggs from which are developed the larvæ in the nostrils, cranial sinuses, auditory meatus, and vagina, and on wounded surfaces. The flies attracted by catarrhal conditions of the mucous membranes and neglected wounds are especially likely to invade the body during sleep in the open air, particularly in the tropics. The local inflammation becomes aggravated, the destruction of tissue progresses, and local pain, headache, facial and pharyngeal œdema, and bloody discharges from the nostrils may result.

In open wounds the larvæ are to be picked off mechanically, but in cases of sinuses they are destroyed and removed by means of injections. In either case the affected part should be thoroughly cleansed and treated antiseptically.

Bot-flies, especially in Central America, Africa, and Russia, penetrate the skin and deposit their eggs, from which the larvæ develop and cause boils, which, if numerous, produce weakness, prostration, and general constitutional disturbance. The eggs or larvæ may be swallowed: the latter may live in the stomach perhaps for several days, producing digestive disturbances, especially distress, nausea, and repeated vomiting, and the living larvæ may be found in the vomit. They may also be alive and present in large numbers in the intestine, giving rise to abdominal pain, and are later discharged with the fæces. Under such circumstances the patient should be freely purged by a mixture of one part of oil of turpentine and three parts of castor oil.

CHAPTER V.

POISONING.

ACUTE POISONING.

THE consideration of poisons may seem somewhat out of place in a treatise upon the practice of medicine, but, as poisoning may simulate various diseases, and as it is constantly met with by the physician, it has seemed to us that a brief practical consideration of the subject having a special regard to the clinical diagnosis and treatment may be of service.

A further justification for the present chapter is found in the fact that the treatment of the various conditions produced by poisoning is the same as that of similar conditions produced by what is called disease. The symptoms of disease are in large part the outcome of the presence of poison in the blood, and so far as the treatment of the symptoms themselves is concerned it makes little difference whether the toxic agent is of such nature that the process it sets up is called "disease," or whether it is of such character that the process is called "poisoning." Thus, the narcosis produced by the natural poisons of uræmia is treated in a similar manner to the narcosis produced by a drug. A convulsion caused by the action of a tetanus toxin is to be relieved in the same way as a convulsion caused by strychnine.

For the purpose of diagnosis the various poisons may be divided into narcotics, or those poisons which chiefly affect the cerebrum, causing narcosis; convulsants, those which especially provoke convulsions; paralyzants, those which cause wide-spread general paralysis; cardiants, those which chiefly affect the circulation; and irritants, those which cause violent irritation of the gastro-intestinal tract and commonly also of the kidneys.

It must be remembered that many poisons act in more than one way, so that it is possible they should be classed under two headings. Thus, illuminating gas produces narcosis and also great depression of the circulation; whilst antimony is a violent cardiac depressant and at the same time a gastro-intestinal irritant. In the present chapter the poisoning is considered under the symptom which is the more important. The vital practical thought is that when a toxic substance acts in two different ways it is essential that two sets of remedies be employed to meet the two actions, the judgment of the practitioner being exercised in the individual case of poisoning in the decision as to which of the two sets of remedies shall be most actively pushed.

Narcotics.

The symptoms of narcosis are stupor deepening into coma, which may or may not become complete, may or may not be accompanied by convulsion, and may be quiet or be accompanied by delirium. Drugs of this class kill by acting upon the respiration, so that there is disturbance, often with slowing, of the respiration. Sometimes the respiration may remain normal or may even be more frequent than normal, but becomes extremely shallow. The character of the pulse varies greatly in accordance with the individual poisons.

We shall divide the narcotic poisons into Class 1, those in which there is no delirium; and Class 2 (Delirifacients), those in which the delirium is marked. It should be observed that in Class 1 the pupil is frequently contracted, sometimes normal, sometimes markedly dilated, and that in the deeper stages of the coma the pupillary reflexes are lost, though they may be preserved in the earlier part of the poisoning. In Class 2 the pupil is usually widely dilated and fixed.

The most important members of Class 1 are opium, alcohol, chloral, chloroform, ether, illuminating gas, prussic acid, nitrobenzol, carbolic acid, oil of tansy, and santonin.

Diagnosis.—In accordance with the plan of this chapter, the existence of narcosis constitutes the starting-point in recognizing poisoning of the present class. Narcosis may, however, be the result of disease, and it is not always possible to distinguish, without a history, between a narcosis due to a disease and one due to a poison. Supposing that there is no history either of the taking of a poison or of the coming on of the symptoms during a disease, or, in other words, that there is no account of the way in which the present condition of the patient has developed, if there be high temperature, or highly albuminous urine, or hemiplegia, or strabismus, or unequal pupils, the case is almost certainly one of disease; although we once saw opium poisoning produce inequality of the pupils, and although a patient suffering from chronic kidney disease may have been poisoned, so that albuminuria is only a presumptive and not positive evidence of the existence of a uræmia.

It must be remembered, further, that an apoplexy may come on during a poisoning; indeed, its development is favored by the existence of alcoholism, so that sometimes patients supposed to be suffering only from drunkenness are found by the police dead in the cells where they have been put overnight. An apoplexy, however, which is sufficiently large to produce so complete a general relaxation as to mask the hemiplegia is always associated with an absolutely complete unconsciousness; whereas it is rare in an ordinary poisoning for a patient to be so unconscious as to give no sign of life when shaken or shouted to. If a drunken person cannot be momentarily aroused, apoplexy should be suspected: stertorous breathing under such circumstances should be considered diagnostic of apoplexy. In advanced cases of poisoning by carbolic and prussic acid, illuminating gas, or nitrobenzol, the unconsciousness may be complete.

A malignant malarial attack may develop suddenly and closely resemble a poisoning, but the disturbance of temperature should almost invariably lead to the recognition of the fact that the case is not one of poisoning: unless the patient has been lying out in the cold, a pronouncedly subnormal temperature is very rare in poisoning. High elevation of temperature probably never occurs in toxic narcosis; certainly the coexistence of internal fever with a low external temperature of the body, such as is seen in malignant malarial attacks, does not happen in poisoning.

In attempting recognition of the individual poison in any case the odor of the breath may point to alcohol, chloroform, or ether. Contraction of the pupil would indicate opium, and, unless in the later stages, would be associated with warmth of the surface and a slow, full pulse. Prussic acid can usually be recognized by the violence and rapidity of the symptoms, by the furious convulsions, and by the bloody foam, the result of these convulsions, about the lips of the bloated, livid face; moreover, death almost invariably occurs before the physician reaches the patient. If there be protracted unconsciousness the case is not one of prussic acid poisoning. Nitrobenzol poisoning can be recognized by the peculiar blue color of the whole surface of the body. Illuminating gas may usually be suspected from the surroundings of the patient; the unconsciousness produced by it is extreme, and is accompanied with great depression of the pulse, which is rapid and feeble, and with some fall of the bodily temperature.

For the purposes of treatment it is extremely important to recognize carbolic acid poisoning, because the antidote to this poison is so complete in its action. In carbolic acid poisoning, if the dose has been large the symptoms develop with great suddenness; there are absolute unconsciousness, complete quiet and muscular relaxation, cardiac failure with rapid pulse, and a tendency to subnormal temperature; the odor of the acid may sometimes be made out upon the person; pathognomonic are corrugated white patches upon the lips or in the mouth, marking places where the strong acid has come in contact with the mucous membrane. In slowly developed poisoning from diluted acid, stupor, coma, muscular relaxation, and failing heart, with subnormal temperature, may offer no decisive phenomena unless there has been sufficient time for the secretion of the characteristic brownish or blackish urine.

Santonin poisoning is to be recognized by the chromatopsia which precedes the development of the other symptoms, and by the saffron-colored or even purplish-red urine. According to authority, the yellow urine becomes red on the addition of an alkali. The convulsions are often violent, accompanied by opisthotonos and emprosthotonos, and are of epileptiform type. There is sometimes slight vomiting, but the symptoms of gastro-intestinal irritation are never severe.

Oil of tansy poisoning is almost always the result of an attempt to produce abortion. The symptoms are stupor deepening into coma, epileptiform convulsions and violent gastro-enteritis, with abortion, which is usually attended with much hemorrhage. The stupor and convulsions distinguish this poisoning from poisonings by other ordinary abortifacients.

TREATMENT.—In all forms of poisoning, if there be an antidote to the drug it should be immediately given. Tannic acid is an imperfect antidote to the alkaloids and substances containing them, and should, therefore, be used in opium poisoning. Of the other poisons in the class, carbolic acid is the only ordinary one which has an antidote. This antidote is unique in that it has the capability of not only acting upon the poison in the alimentary canal, but also of following it into the blood and tissues and there neutralizing it. Whether it be late or early in the poisoning, the practitioner should exhibit the antidote freely, both by the mouth and in severe cases hypodermically. Sulphuric acid or the soluble sulphate should be given. Magnesium sulphate may be administered by the mouth, but not hypodermically, at least with any freedom, since when injected directly into the blood it acts as a violent poison. Sodium sulphate may be given hypodermically.

If a poison has been taken by the mouth, after the administration of the antidote the stomach should be washed out : a brisk purgative like croton oil is sometimes useful for the purpose of emptying the alimentary canal.

Unconsciousness is, from a therapeutic point of view, a matter of little importance : the danger in narcosis is from depression of the respiratory centres, so that the indication is to maintain respiration. In certain cases of poisoning, notably opium poisoning, the respiratory centre is depressed more than are the centres of consciousness ; hence it is advisable to keep the patient awake for two purposes : first, that the depression and relaxation of sleep may not be added to the depression and respiratory relaxation directly due to the poison ; second, that automatic respiration may be reinforced by voluntary breathing. We have seen in opium poisoning a patient sitting breathing regularly at the word of command when automatic breathing had practically ceased. It must be remembered in treating such a case that owing to the continuous lack of respiration there is an ever-increasing accumulation of carbonic acid in the blood, so that finally the narcosis is of double origin ; and if by artificial respiration or otherwise the blood can be freed from the accumulated gas the narcosis is greatly lightened, and emetics and other drugs previously powerless may become active for good. It is evidently often very useful to arouse a narcotized patient. For this purpose flagellations and other forms of rough treatment have been much employed. They are, however, unjustifiable. If walking or mild shaking of the patient does not suffice, the dry wire brush should be used with a strong electrical current. This makes an intense irritation of the peripheral nerves without causing any inflammation or structural change.

The drugs which are useful in overcoming respiratory paralysis are the so-called respiratory stimulants,—namely, atropine, strychnine, cocaine, and caffeine. In using these drugs it is essential to apprehend that they are not simple physiological antagonists to the poison, and that they are used for one distinct purpose. Thus, in the employment of atropine in opium poisoning it does not do to use the pupils as a guide to the amount of atropine to be given: opium contracts the pupil centrically, atropine dilates it peripherally,—so that the actions are not, strictly speaking, antagonistic. The results of the administration of the antidotal drug are to be chiefly judged by the action upon the respiration: if the respiration becomes sufficiently more rapid and full the desired result has been reached, and no more of the antidote should be exhibited until respiration begins again to fall. In order to get the necessary quick action from the antidote it should always be given hypodermically. The dose administered should be much larger than that used for ordinary purposes, and should be somewhat proportionate to the amount of the poison taken, if this be known. The remedial action of the antidote in such cases is in obedience to what H. C. Wood has termed the "law of crossed action," a law which may be used for the purposes of antagonism as well as of co-action of medicines. Thus, drug *x* stimulates the heart, the respiratory centres, and the intestinal peristalsis, whilst drug *y* stimulates the respiratory centre and the motor spinal cord, but has no effect upon the heart. It is evident that if these two drugs are given at one time they will reinforce each other at the respiratory centre without acting together in other portions of the body, so that the greatest respiratory effect will be obtained with the least possible disturbance of other functions. We have proved experimentally that if to a dog poisoned with chloral strychnine be given until general convulsions seem imminent, there will be a great increase in the respiratory movement,—an increase, however, which can be still further augmented by atropine or cocaine without precipitating convulsions, as would be done by further doses of strychnine. Hence in narcosis the best effects are to be obtained by using together two or more respiratory stimulants.

In any case of narcosis in which the respiration is failing, artificial respiration should be resorted to. Of the ordinary methods Silvester's is perhaps the most effective. The patient being laid upon his back on a hard table or the floor, the elbows should be forced tightly against the chest so as to compress it, then raised upward and outward (not forward) until the highest point above the head is reached, and then slowly replaced and the process repeated. This should be done about ten times a minute. Of course in any case of existing deep narcosis the tongue should be well drawn out and prevented from slipping back upon the larynx. Very frequently it suffices to draw the whole lower jaw upward and forward by the fingers inserted behind its angle.

Very much more effective than artificial respiration in the treatment of narcosis is forced respiration, in which by means of bellows or other power air is driven into the lungs. For the details of apparatus and method the reader is referred to H. C. Wood's "Therapeutics." In a number of cases of violent narcotic poisoning life has been saved by forced respiration kept up for many hours.

In many cases of narcotic poisoning other symptoms than those of narcotism are sufficiently prominent to need treatment, as heart-failure in carbolic acid, vomiting and purging in poisoning with oil of tansy, etc. The treatment of these symptoms will be found under the head of their respective divisions.

Delirifacients.—The ordinary poisons representing the second class of narcotics—*i.e.*, those which produce delirium with the early stage of the narcosis—are atropine and hyoscyamine and the vegetables which contain them,—such as belladonna, hyoscyamus, datura, or Jamestown ("jimson") weed,—cannabis indica, hyoscine, and cocaine. With the exception of hyoscine, these substances are all respiratory stimulants, and in the early stages of the poisoning increase the rate and force of the respiration. There appears to be no recorded death from cannabis indica, or Indian hemp, nor from hyoscine, though the latter drug is certainly capable of taking life.

DIAGNOSIS.—All these drugs produce dilated pupils, a peculiar talkative delirium, dry mouth, usually nervous excitement and unrest, the whole ending, if the dose has been sufficient, in narcosis of a quiet type, with failure of respiration. The diagnosis between them can be made by noting that atropine and hyoscyamine cause a rapid, hard pulse and much greater excitement than is present with hyoscine, the delirium of which is not accompanied by muscular excitement, is talkative and muttering, and in its milder form simply represents a condition of simple confusion. Moreover, in hyoscine poisoning the pulse is near the norm in rate and force. Cocaine is to be recognized by the intense motor excitement which usually attends its action, and which may bring its poisoning into the convulsant group. When the dose of cocaine has been very large this excitement may be wanting, and collapse with unconsciouness may be the most prominent symptom, so that the poisoning might be placed in the cardiant group.

TREATMENT.—In the early stage, after the use of tannic acid as an antidote and the evacuation of the stomach, if there be great excitement, morphine and even chloral may be used with caution. If the symptoms be due to hyoscine, the treatment should be similar to that of the quiet narcotic group. In the advanced stages, with stupor and quiet narcosis, the treatment becomes that of the quiet narcotic group, with, when there are symptoms of heart-failure, the use of cardiac stimulants.

Convulsants.

Convulsant drugs, that is, drugs the chief symptom of whose poisoning is convulsions, are divided into those which produce cerebral or epileptiform convulsions and those which cause spinal or tetanic convulsions. The epileptiform convulsion is distinguished at once by the loss of consciousness, and by the fact that the convulsion is clonic. The spinal convulsion is without loss of consciousness, with heightened reflexes, whilst the contractions are more or less persistent or tonic.

Drugs which produce epileptiform convulsions often do so in an indirect manner, as by producing anæmia of the brain through cardiac depression (veratrum viride, for instance). They may act directly upon the cerebrum, so as to cause persistent loss of consciousness. (See Narcotics.) The drugs which produce spinal tetanic convulsions are cocaine and strychnine.

DIAGNOSIS.—In the diagnosis of strychnine poisoning it is necessary to differentiate it from tetanus and hysteria. Except in very rare cases, tetanus is at once to be distinguished by the comparative slowness of the symptoms, which extend over hours or days, and especially by the fact that the jaw is primarily locked. The masseter and other chewing muscles are the first to be attacked; whereas in strychnine poisoning they are the last. The hysterical convulsion may simulate that of tetanus; there is usually, however, disorder of consciousness, which may be lost, or, more characteristically, may be so perverted that the patient seems conscious but afterwards has no memory of events which occurred during the convulsion, or else seems unconscious but afterwards has complete memory of such events. The hysterical convulsion is also accompanied by much greater and more varied emotional disturbance than is the strychnic convulsion, though terror in the strychnic convulsion may be extreme. The reflexes, while often exaggerated in the hysterical convulsion, lack the extraordinarily intense activity seen in the strychnic convulsion. The strychnic convulsion is more complete than the hysterical, involving all the muscles of the body, and producing opisthotonos. The hysterical convulsion is usually more or less incomplete, with varieties of postures, emprosthotonos, pleurothotonos, and various apparently purposive attitudes, as that of the cross. In hysteria there is also prolonged rigidity between the convulsive attacks.

We have seen a momentarily mistaken diagnosis made in strychnine poisoning, growing out of the fact that the asphyxia very rapidly developed to the point of unconsciousness. It must be borne in mind that in the toxæmic epileptiform convulsion the unconsciousness precedes the first convulsive movement; while in the strychnic convulsion the convulsive movement always precedes the unconsciousness, though it may do so only for some seconds.

Cocaine poisoning is to be recognized by the coexistence of cerebral excitement, dilated pupil, accelerated pulse, and dry mucous membrane of the mouth, with convulsions which are also more partial and less severe than those of strychnine.

TREATMENT.—In severe strychnine poisoning no attempt should ever be made to wash out the stomach. We have seen a fatal strychnic convulsion provoked by the irritation of the fauces by the stomach-tube. Emetics, hypodermically (apomorphine) or by the mouth, are sometimes helpful, but should be used only in the very beginning of the poisoning. Tannic acid may be given as an antidote.

The drugs to be employed are the anæsthetics, amyl nitrite, potassium bromide, and chloral. Of these the two volatile substances are to be given usually for their immediate action, and are therefore especially to be employed during severe convulsions. When respiration is arrested by the cramp of the respiratory muscles, and no air movement takes place, the amyl nitrite may be given (ten minims) hypodermically, or a clyster of thirty to forty grains of chloral may be administered. The irritation, however, of the rectum by the pipe of the syringe may produce or increase the convulsion. If the patient can swallow, the potassium bromide should be at once administered in very large doses, half an ounce if the strychnine has been in considerable amount, and should be followed or accompanied by the chloral, whose action is much quicker and for the moment more effective than that of the bromide. In accordance with the law of crossed action, no one drug should be relied upon; but Calabar bean is too slow and uncertain in its action for practical use, though its alkaloid, eserine sulphate (one-twentieth of a grain), may be given hypodermically. In prolonged strychnine poisoning alcohol should be used freely, and cardiac stimulants may become necessary.

Paralyzants.

The poisons which cause wide-spread general paralysis may do so by a centric or by a peripheral action. Most of the drugs have other actions besides that of paralyzants, and in many cases the paralytic action is more or less subordinate to other influence. The most important of these drugs are the central motor depressants, chloral, Calabar bean, the nitrites, gelsemium, and the peripheral nerve paralyzants, lobelia, coniine, woorari, pelletierine.

DIAGNOSIS.—The recognition of the motor paralyzing poisons depends upon the existence or non-existence of symptoms other than paralysis. With chloral is unconsciousness; with the nitrites are the flushed face, the cerebral distress and sense of distention, and the violent cardiac action, which in association with the extremely rapid development and fugaciousness of the symptoms are characteristic; with Calabar bean there is the contracted pupil; with lobelia there is violent vomiting without purging, with great nausea, rapid feeble pulse, and general symptoms of collapse, —symptoms resembling very closely those of veratrum viride poisoning;

with gelsemium there are dilatation of the pupil, strabismus and its consequent double vision, dropping of the jaw, and depression of the circulation, shown by rapid, feeble pulse, without much vomiting; with conium the symptoms are purely paralytic, without disturbance of consciousness or circulation,—namely, an ever-increasing weakness associated with dilatation of the pupil and paralytic squint, without dropping of the jaw; with woorari and pelletierine there is simply a progressive general paralysis.

TREATMENT.—To vegetable drugs of this class tannic acid is a more or less imperfect antidote. For the mineral drugs there is no known antidote. Washing out of the stomach should be practised, unless the patient be *in articulo mortis*. Death occurs through failure of respiration. The centric paralyzing drugs act directly upon the respiratory centres, so that the treatment of their poisonings is that for narcotism, except so far as the narcotic treatment is directed towards keeping the patient awake. What was said of the use of respiratory stimulants and artificial respiration under the head of Narcotics holds for drugs of the present class. When the paralysis and the respiratory failure are due to an influence upon the peripheral nerves, centric respiratory stimulants are of very little value, and there are no known drugs which act antagonistically to poisons that affect the nerve-trunks. Artificial or forced respiration is, of course, indicated.

Cardiants.

Drugs may arrest the heart's action in systole or in diastole. Practically, systolic arrest of the heart from a poison is almost never seen; in the rare cases in which it may be threatened from overdoses of digitalis, aconite or some other cardiac depressant should be carefully used. The ordinary poisons which produce arrest of the heart's action in diastole are veratrum viride, aconite, the nitrites, and tartar emetic. Of these drugs, tartar emetic will be spoken of under the heading of Irritants, whilst the nitrites have already been discussed under the heading of Paralyzants.

DIAGNOSIS.—Veratrum viride poisoning is to be recognized by the excessive vomiting and prostration, without either purging, pain, or disturbance of the pupil or of consciousness; there are, in a word, no other symptoms, except progressive weakness, rapid pulse, subnormal temperature, free sweating, and collapse. Aconite poisoning is characterized by the tingling and numbness, which usually appear first in the lips (the point of contact with the drug), then in the fingers and hands, and finally in the legs. Even without this subjective symptom, which must be told by the patient, aconite poison may be recognized by the coexistence of progressive muscular failure and loss of reflexes, with symptoms of collapse, without vomiting, purging, or other phenomenon.

TREATMENT.—In the treatment of the failure of circulation produced by poisons, the patient should be placed upon the back in a horizontal position, or with the head slightly lower than the feet, and in case of sudden and complete failure of the heart should be momentarily inverted. As has been shown experimentally upon dogs by H. C. Wood, such inversion does good, not, as was formerly taught, by supplying blood to the respiratory centres, but by causing a flow of blood from the abdomen into the right heart, with consequent distention of the ventricle and mechanical excitement of the viscus to renewed action. It is evident that a better result is to be obtained by putting the patient back into the nearly horizontal position after the heart has been started, with subsequent inversion for a moment if it be found necessary, than by keeping the patient inverted for a length of time; otherwise the strain on the weakened left heart to force the blood into the abdomen and lower extremities may be too much for it. The sudden assumption of the sitting or erect posture by such a patient may, by causing sudden emptiness of the right auricle and ventricle, lead to immediate cardiac arrest. Absolute quiet must be enforced. If, as is often the case, the temperature falls, it is essential to maintain it, sometimes by immersion in the hot bath or by a device which we have found very useful in various cases of surgical and medical shock or collapse. An ordinary water-bed is about two-thirds filled with water at a temperature of 170° F., and the patient is laid upon a blanket or blankets on the bed; the body is in this way half enveloped in hot water, and must be heated. Such a bed about two-thirds full of water will remain hot from six to ten or even more hours.

The drugs useful in cases of cardiac collapse are digitalis, strychnine, cocaine, and atropine. They should always be used hypodermically in doses proportionate to the amount of poison which has been taken. All that has been said of the value of crossed action in a previous section is equally applicable to drugs of this class. Ether given cautiously by the lungs, in moderate amount, is sometimes useful when the heart has suddenly given out. Alcohol administered by the mouth in concentrated hot solution is frequently of value. The nitrites given very cautiously by inhalation may in sudden cases be serviceable, but it must be remembered that the slightest overdose of the nitrite converts it into a cardiac depressant.

In most cases of collapse vaso-motor depression is a very important part of the condition. Digitalis, strychnine, atropine, and cocaine are all vaso-motor stimulants. They are also respiratory stimulants, and usually there is in the condition under discussion respiratory depression. These drugs are, therefore, much more effective and much more valuable than alcohol or the nitrites. The nitrites are very powerful blood-vessel paralyzants, and must be used with the greatest caution, if at all. Alcohol, even in small dose, probably tends to widen the blood-paths, and certainly has such influence when given in full dose: practical experience is in accord with theory and experiment in showing that in surgical shock or other collapse with vaso-motor paralysis alcohol is of little value.

Irritants.

The number of substances which are capable of producing serious and even fatal gastro-intestinal inflammation is so great that it is not worth while to attempt in this place to give a list of them. Under the head of diagnosis some of the more important will be especially spoken of.

DIAGNOSIS.—The symptoms of gastro-intestinal irritation are vomiting and purging, with pain, tenderness, and secondary collapse. There are certain irritating substances, especially the corrosive chemicals, such as the mineral acids, which produce, when taken internally in concentrated form and large amount, immediate fatal collapse without pronounced local symptoms; after a smaller or less concentrated dose there may be immediate violent pain in the œsophagus and stomach, rapidly followed by collapse without vomiting.

From the symptoms themselves it is not possible to diagnose between the effects of different mineral acids, but usually the source of the poisoning can be recognized by noticing the stains upon the clothing or about the mouth of the victim. Sulphuric acid makes a black stain; nitric acid, a deep yellow stain; nitrohydrochloric and hydrochloric acids, a feeble yellow stain. The holes eaten in linen and other clothing by one of these acids are to be distinguished from holes due to burning by their edges being soft and pulpy, and, if at all recent, yielding an acid reaction with litmus paper.

Among the vegetable acids, citric is scarcely capable of taking life, though it may produce violent vomiting and much pain. Tartaric acid has in a few recorded cases caused death, the primary symptoms being chiefly gastric. Oxalic acid causes violent vomiting and purging (often bloody), with stupor or even deep narcosis, and wide-spread general paralysis, sometimes with epileptiform convulsions, these nervous symptoms occurring early and being out of proportion to the local symptoms because they are not dependent upon the gastro-intestinal inflammation, but are due to a direct action of the poison upon the nerve-centres. After death oxalic acid poisoning can be recognized by the presence of crystalline oxalates in the secreting structure of the kidneys.

Among the mineral poisons, antimony and arsenic produce profuse serous stools, with an association of other symptoms, indistinguishable from the symptoms of true cholera or of cholera nostras. It is rare for acute antimonial poisoning to depart from the type, but in arsenical poisoning stupor and other evidences of centric nerve oppression are not uncommon. There is no way of distinguishing these poisonings from choleraic diseases except by a knowledge of the history of the case or by the recognition of the poison in the vomit, stools, urine, or tissues. In many cases extraneous circumstances may lead the physician to a working diagnosis. Corrosive sublimate in excess produces a very violent poisoning, which is characterized by excessive abdominal pain, with violent vomiting and purging, the stools being small, mucous, bloody, and passed with much straining.

The majority of soluble metallic salts are irritant poisons. Cupric sulphate can often be recognized by the blue color of the first vomit. With plumbic acetate the first vomit is often white and curdy (lead chloride); the stools, which may be loose or hard, are always of an intense black color (lead sulphide). Phosphorus, which may be classed among the irritant poisons, is almost unique among known poisons in not producing distinct symptoms for from six to twelve hours after its ingestion, even although it has been taken in great excess; the symptoms and lesions which it causes so exactly simulate those of acute yellow atrophy that the poisoning can scarcely be distinguished from the natural disease, either during life or upon the post-mortem table, except by chemical examination or a knowledge of the poison having been taken. Phosphorescence in the vomit or stools would be direct evidence of poisoning. According to M. Poulet, phosphorus poisoning can be at once recognized by heating the urine with nitric acid to calcination, when as dryness is reached there is a sudden outburst of flame. In rare cases phosphorus poisoning has differed from the type, with many irregular symptoms.

Among the other irritant, poisonous drugs the only ones which we shall notice are oil of rue and oil of savin, which have frequently, when taken for the purpose of producing abortion, caused violent gastro-enteritis, with vomiting and purging, and abortion attended with much hemorrhage, ending in death, and Spanish fly, or cantharides, the symptoms of whose poisoning are furious gastro-enteritis with intense pain and incessantly repeated small bloody mucous stools, accompanied by inflammation of the genito-urinary tract, as shown by violent pain, strangury, albuminous or suppressed urine, priapism, and finally, perhaps, sloughing of the parts.

TREATMENT.—The antidotes to the mineral and vegetable acids, other than oxalic, are the alkalies and their carbonates and substances containing them, such as soap. For oxalic acid use lime or chalk; for antimony, tannic acid; for arsenic, freshly precipitated ferric hydrate, which may be made by precipitating solution of tersulphate or subsulphate of iron, or tincture of ferric chloride, with an alkali, preferably magnesia (ferri oxidum hydratum cum magnesia, *U.S.*). The antidotes to plumbic acetate are a soluble sulphate or chloride, such as common salt, or an alkali or its carbonate, and soap. To almost all irritant metallic salts the antidote is soap or an alkali; to corrosive sublimate, white of eggs; to phosphorus, cupric sulphate or potassium permanganate.

The drugs to be used in toxic gastro-enteritis are opium,—which in very severe cases threatening immediate collapse may be first given hypodermically, but which it is usual preferably to exhibit by the rectum,—bismuth, and chalk. Demulcent liquids should be used freely, especially when there is great irritation of the kidneys. Leeching at the epigastrium, and sinapisms or turpentine stupes over the whole abdomen, followed by warm, moist applications, are often of great service. Acute toxic nephritis is to be treated in the same manner as the same disease from natural causes.

Among the irritant poisonings may be mentioned the cases in which violent symptoms and even death have been produced by the taking of various articles of food which have undergone changes allied to putrefaction. Among the foods which have produced serious results may be mentioned European mussels, fish, sausage, ham, various fresh meats, canned goods, milk and its products, such as cheese, ice-cream, and custards, or various complicated desserts, like cream-puffs. The nature of the poison in these cases varies to some extent, but usually it is a ptomaine, a nitrogenous base allied to the alkaloids, the result of putrefactive or fermentative changes produced by the presence of bacteria.

The symptoms may develop directly after the ingestion of the poisonous food, or may be delayed for some hours. They usually consist of violent vomiting and purging, accompanied by abdominal uneasiness and sometimes great pain. In rare cases there is constipation. With these abdominal symptoms are often associated nervous disturbances out of proportion to the gastro-intestinal irritation, and evidently directly produced by the poison. These are vertigo, disturbances of vision, dilatation of the pupil, delirium, or stupor, ending, it may be, in coma and death. Sometimes there are violent convulsions. The disturbance of the circulation is usually marked, as shown by rapid, feeble, or irregular heart-action, with a small, thready, rapid pulse. The temperature may be subnormal or febrile. Dryness of the throat, rigors, widely distributed cramps, numbness and tingling in the extremities, and difficulty in swallowing, are not rare phenomena.

The treatment in these cases consists, first, in emptying the alimentary canal; secondly, in treating the symptoms as they arise. Sometimes, especially in children, failure of respiration is an early and very distinct symptom. We have seen life saved by placing such a patient in the hot bath and using artificial respiration until reaction set in, with vomiting and relief of the stomach.

CHRONIC POISONING.

LEAD POISONING.

Colica pictonum, or *subacute lead poisoning*, is especially seen in workers in lead or its compounds, though it may be the result of accidental poisoning in those who do not work in the metal. The symptoms are malaise, followed by violent abdominal colicky pains, which are more or less constant, with exacerbations, and vary in character, being sometimes sharp and sometimes dull, and frequently described as a twisting around the navel. There is usually no appetite, and vomiting and retching are common. The walls of the abdomen are retracted, rigid, knotted; the bowels are obstinately costive; the tongue is contracted and whitish, the appetite absent, and the thirst sometimes excessive. Neuralgic pains in the thorax and in the extremities are of frequent occurrence. In some cases the conjunctiva is distinctly icterode.

Lead colic may yield to treatment or by recurrent attacks may pass into chronic lead poisoning.

Chronic lead poisoning varies so much as almost to baffle concise description. The symptoms can, perhaps, best be studied by arranging the cases in groups; but it must be remembered that not only do these groups shade into one another, but also that there are all kinds of mixed cases,—cases which offer simultaneously or successively symptoms of two or more of these various groups.

The first group contains the great bulk of cases of chronic lead poisoning, at least as seen in this country. The symptoms consist of failure of health, more or less digestive disturbance, and double wrist-drop,—*i.e.*, paralysis of the extensor muscles of each hand. Not rarely the only noticeable symptom is the wrist-drop, the general health seeming to be good. The true nature of such cases can usually be at once recognized by the bilateral character of the extensor-paralysis, cerebral and pressure paralyses being almost invariably unilateral. We have seen, however, bilateral pressure palsy, and also unilateral plumbic wrist-drop, due to a local absorption of lead, in an artisan whose hand was much of the time in a preparation of the metal. The wrist-drop may exist alone, but not rarely there is with it anæsthesia of the affected part, or sometimes of the shoulders or other unparalyzed portion of the body. When the paralysis is complete, the electro-contractility of the muscles is in great part or altogether absent.

The rarer forms of chronic lead poisoning may be divided into the cerebral, the periphero-spinal, and the nutritive.

In the cerebral cases should be included those which are commonly spoken of as *encephalopathia saturnina*, or *saturnine cerebritis*, in which the violent brain-symptoms may develop with great suddenness, or may be preceded by headache, giddiness, sleeplessness, disturbed vision, strabismus, tinnitus aurium, psychical aberration, or other prodromes of cerebral disturbance. Delirium, which is among the chief manifestations of the fully formed condition, may be mild, but is often maniacal; stupor may replace it or alternate with it; and violent epileptiform convulsions, ending in coma, are not infrequent. These convulsions are usually the precursors of death, but recovery may occur.

Without the development of such severe symptoms, headache, loss of memory, giddiness, somnolence, hemianæsthesia, disturbance of the special senses, aphasia, monoplegia, hemiplegia, or multiple cerebral palsies may occur during chronic lead poisoning. Death, preceded by severe cerebral symptoms, may take place without organic lesion; but usually, when focal symptoms have been present, localized alteration of brain structure, secondary to diseases of the cerebral vessels, or to chronic inflammation of the brain or its membranes, can be detected. Sometimes the cerebral symptoms are uræmic; indeed, true plumbic encephalopathy and plumbic uræmia from contracted kidney may coexist. Again, the more serious affection may be masked by a saturnine hysteria, since cases have been reported by Charcot and by Dutilh in which hysterical hemianæsthesia, amaurosis, anosmia, loss of sense of taste, and other cerebral symptoms have been the outcome of a major hysteria due to chronic lead poisoning. Such cases as these probably occur only in individuals of hysterical temperament, and must be extremely rare in persons not of the so-called Latin peoples.

Disturbances of vision are so frequent and so marked in lead poisoning as to deserve special mention. The amblyopia may come on slowly or suddenly; it may be partial or complete; it may coexist with kidney disease or may be entirely independent of the latter; associated with it may be a true optic neuritis or a true optic atrophy, but, on the other hand, it may exist without demonstrable disease of the optic nerves. It is undoubtedly often due to a disease of the optic nerves themselves, but the occurrence of homonymous hemianopsia in some cases seems to demonstrate that the blindness may be of centric origin. Strabismus from paralysis of the external rectus or other ocular muscle is sometimes of saturnine origin.

The second group of cases of chronic lead poisoning comprises those in which the nerve-symptoms apparently originate below the cerebrum. Among these may be mentioned cases such as have been reported by Putnam, by Tissier, by Raymond, and by G. L. Walton, in which the phenomena resemble those of locomotor ataxia, except in the presence of tenderness over the nerve-trunks, preservation of the tendon reflexes, or some other atypical symptoms. We have seen cases in which the symptoms simulated those of an acute poliomyelitis, consisting chiefly of widespread paralyses with rapid wasting of the muscles. These cases usually can be differentiated by the presence of violent neuralgic pains, paralysis of the bladder and rectum, or other atypical symptoms. Again, cases very closely resembling ascending or Landry's paralysis have been reported. Severe intractable chorea has been produced by lead. Disturbances of sensation may occur in lead poisoning; anæsthesias are not very rare, and violent neuralgic pains, probably due to neuritis, may be the chief manifestation. In one case under our care the symptoms were intense general pruritus, with violent neuralgic pains shooting through the rectum and the urethra, coming on at night and producing an insomnia which appeared to be unconquerable. The lesion in most of these motor and sensory cases is probably in the nerve-trunks, and the very rapid pulse seen in some of them may be due to disease of the vagi, since Prevost and Binet have found pronounced degeneration of these nerves.

The third group of cases contains those in which the poison chiefly expends itself upon glandular or visceral organs or in producing wide-spread nutritive changes. It would seem that almost any of the vital structures may undergo degeneration. Potain reports saturnine cirrhosis of the liver; Valence details a plumbic parotitis. Rudolph Maier has found in poisoned animals atrophic degenerations of the intestinal glands and walls; and there can be no doubt that similar alterations sometimes aid in the production of emaciation and anæmia in human plumbism.

Temporary albuminuria may occur in lead poisoning without serious implication of the kidneys; while, on the other hand, fatal nephritis may exist when there is no albumin in the urine. A persistent low specific gravity of the urine in lead poisoning is a symptom of the utmost gravity. Geppert confirms the observation, previously made by Oliver, that in temporary plumbic albuminuria many isolated kidney epithelial cells may often be found in the urinary sediments; and it is evident that a persistence of this condition must end in chronic renal disease. After death, which may be induced by uræmia, the kidneys are found contracted, granular, with excessive development of the fibrous tissue (followed by contraction) and great thickening of the walls of the blood-vessels: these changes are identical with those of contracted kidney produced by gouty and other irritant poisons. As Ellenberger and Hofmeister have shown that the lead is chiefly eliminated by the kidneys, the frequency of plumbic nephritis is easily explained; but it is not readily perceived why it is so frequently associated with an arthralgia whose course and lesions closely simulate those of chronic gout.

There are cases of lead poisoning which do not conform to any of the types as yet given. Acute asthma has been produced by the inhalation of the dust of white lead, whilst chronic saturnine asthma occurs in feeble, narrow-chested people. James J. Putnam asserts that in lead poisoning of children the legs and feet are commonly paralyzed. Pagliano has reported saturnine facial palsy. Upon pregnant women the influence of the poison is very deleterious, and, as was shown by Constantine Paul, it often produces the early death of the fœtus.

In those cases of lead poisoning which pursue a slow course to death, the paralysis involves after a time the extensors of the lower as well as of the upper extremities, epileptic paroxysms occur at intervals, racking pains shoot through the limbs, points of cutaneous anæsthesia appear, and often albuminuria aids in producing the fatal issue. Gradually the patient becomes more and more cachectic, general œdema and the whitened skin betray the increasing anæmia, the paralysis extends from muscle to muscle, locomotion becomes impossible, and, if a convulsion or other accident do not close the scene, death at last takes place from loss of power in the respiratory muscles. Malassez has found that in the anæmia of lead poisoning the red globules are not only diminished in number but also increased in size. After death the metal has been detected in all the soft and hard tissues of the body.

DIAGNOSIS.—The diagnosis of lead poisoning is easy when the characteristic blue line upon the gums where they join the teeth is present, but death may occur without any such mark of plumbism; in which case, if the symptoms be irregular, the nature of the attack can be made positive only by an examination of the urine. Before this is done potassium iodide should be given in moderate doses for four or five days, so as to insure the elimination of lead if it be in the system. The urine should be slightly acidified and put in flint-glass bottles immediately after it is passed, and at least one quart of it should be sent to the chemist.

TREATMENT.—In the treatment of chronic lead poisoning there are three indications: first, to prevent the ingestion of more of the poison; second, to aid in the elimination of that in the system; third, to relieve symptoms and restore lost functions. In lead colic both of the last two indications are met by purgatives, to which opium should be added to relieve pain. It is often necessary to use the most powerful drastics, such as croton oil; but senna, salts, and other of the milder cathartics should always be tried first. *Alum*, it is asserted, acts in some unknown way as a specific in lead colic, and from twenty to sixty grains of it may be given four or five times a day; but our experience is not favorable to its use. In chronic lead poisoning, to fulfil the second indication baths of potassium sulphide should be employed, and potassium iodide (five grains three times a day) be administered internally. Oddo and Silbert state that the elimination of lead through the skin in chronic lead poisoning is important, and is facilitated by injections of pilocarpine. Baths containing six to seven ounces of potassium sulphide should be given in a wooden tub, two or three times a week. The patient during the half-hour of the bath should be from time to time well rubbed with a coarse towel. On coming out he is to be thoroughly washed with warm soapsuds. When severe cerebral symptoms arise, treatment is of little avail, and should be largely expectant. In cases of lead poisoning in which the symptoms resemble those of acute poliomyelitis we have used ascending doses of strychnine with extraordinary results, rapidly deepening paralysis being almost at once controlled. It is essential that the strychnine be pushed to the point of systemic intolerance. It is best to administer it hypodermically at least three times in the twenty-four hours.

The local use of electricity is exceedingly important to restore the lost function of nerve and muscle. When the faradic current elicits a response, it should be employed; but in some cases the continued current retains its power after the induced current has lost all its influence. The rule is always to apply that current which causes contraction with the least pain; if both fail, the continued current should be used, the poles being reversed at intervals of four or five seconds. The electrical séances should be tri-weekly, each lasting about fifteen minutes, and should be persevered in for months.

ARSENICAL POISONING.

The symptoms of chronic arsenical poisoning vary, and may be very obscure. They were summed up by the late Professor Taylor as follows: "dryness and irritation of the throat, irritation of the mucous membranes of the eyes and nostrils, dry cough, languor, headache, loss of appetite, nausea, colicky pains, numbness, cramp, irritability of the bowels, attended with mucous discharges, great prostration of strength, a feverish condition, and wasting of the body." The constitutional troubles most uniformly present in these cases are weakness and emaciation, often accompanied by more decided nervous manifestations than the picture drawn by Taylor would suggest: great depression of spirits and irritability of disposition, sleeplessness, giddiness, tinnitus aurium, failure of memory, cerebral neurasthenia, headache with a feeling of constriction in the forehead, and numbness in the extremities, are probably the most common symptoms, although muscular tremors or stiffness, vertigo, and even convulsions and paralysis, are not extremely rare. Kirchgässer asserts that the most characteristic phenomena are a brown pigment-deposit in the skin of the face, inflammatory affection of the eyelids, and disturbances of sensibility and motion, which affect most frequently the lower extremities, together with scalding during urination.

Diagnosis.—The diagnosis of chronic arsenical poisoning is often very difficult. Sometimes eruptions upon the skin, with laryngo-bronchial catarrh, swollen finger-joints, emaciation, and other disturbances of the general nutrition, constitute the main feature of the case. Peripheral neuritis is almost always due to the presence of some poison, and general emaciation without local disease and with atypical symptoms is usually either toxic or diathetic. In some cases the symptoms have been gastro-intestinal irritation, anæmia, dermatitis, redness of the conjunctiva, puffiness under the eyes, headache, irritation of the upper air-passages, albuminuria with casts and blood, and peripheral neuritis. The mere inability to account for failure of health should put the practitioner on his guard. Extraneous circumstances often are such as to suggest the truth. If green or other colored wall papers are in the bedroom, or if the patient is a chemist or a worker in arsenical compounds, aroused suspicion should lead to a chemical study of the urine.

Treatment.—Recovery almost invariably occurs in chronic arsenical poisoning, if the continuous intaking of the poison be arrested. There are no known means of increasing the elimination of the metal. The symptoms must be met on general principles as they arise. Gastro-intestinal inflammation, peripheral neuritis, and other lesions should be treated in the same way as though produced by more ordinary causes.

CHRONIC ANTIMONIAL POISONING.

Chronic accidental poisoning by antimony or its compounds is practically unknown, but in a number of cases tartar emetic has been given continuously in small but accumulating doses with criminal intent. The symptoms produced have been those of a subacute gastro-enteritis with numerous exacerbations, followed by emaciation and finally death by exhaustion. In such a case suspicion should be aroused by the gastric disturbance being much more severe than that which usually accompanies enteritis from natural causes, and by the unaccountable exacerbations, baffling all medical foresight and treatment. There being no characteristic symptoms, the detection of the true nature of such a case must be based upon chemical examination of the excreta. As the metal is freely eliminated by the kidneys, and as the urine is frequently taken for examination by the physician in a suspicious case, this fluid could be chemically studied and a decision arrived at without alarm to those about the supposed victim.

Chronic antimonial poisoning will almost always get well spontaneously on the cessation of administration. For the relief of the symptoms opium should be freely used. Strychnine and digitalis should be given hypodermically if respiratory and cardiac failure are alarming.

ALCOHOLISM.

Alcohol is an irritant narcotic, which, under certain circumstances, has a tendency to cause numerous degenerative changes in various organs and tissues of the body. The diseases produced by its abuse are in great part elsewhere considered in this volume. At this place the consideration will be confined, first, to the results of an ordinary Alcoholic Debauch ; second, to Delirium Tremens ; third, to Alcoholic Insanity.

ACUTE DEBAUCH.

During an acute debauch the irritant action of the alcohol is felt at its point of entrance and also at its point of escape from the body. There are, therefore, primarily and chiefly, a toxic gastritis and an irritation of the hepatic cells, which may be so intense as to alter their functional activity, and there is also great renal irritation ; hence the icteric conjunctiva, heavily coated tongue, yellowish skin, nausea and vomiting, headache, gastric and hepatic tenderness, and scanty, albuminous urine. The indications for treatment are to get rid of any remaining alcohol, to relieve the congestion of the portal circulation and of the stomach, to purify the blood, to soothe the kidney, and to support the nervous system. To meet the first of these indications, very free sweating may be induced by the Turkish or the vapor bath, or by a hypodermic injection of pilocarpine. In many cases an emetic, tartar emetic if the patient is robust, aided by large draughts of hot water, may be of great service in relieving the stomach and assisting the full dose of calomel in starting biliary secretion. Cream of tartar and other saline diuretics may be early used, assisted by copious draughts of hot water. The food should be liquid, but stimulating and nutritious: milk with lime water, strong broths, and beef-essence, are very useful. The peculiar gastritis caused by alcohol is accompanied by so much relaxation and so much apathy of the mucous membrane that Cayenne pepper and other irritant spices are often very useful additions to the food. Moreover, after acute symptoms have subsided, simple bitters are often of service; especially are the alkaloid hydrastine and its salts of value.

When the gastric symptoms are very severe, leeching and subsequently blistering of the epigastrium may be practised. Ordinarily, no alcohol should be allowed, but bromides, chloral, sulphonal, and opium may be administered when required by the nervous symptoms. Strychnine and tonics should usually be withheld until the gastro-intestinal irritation has been subdued. If, however, there be signs of exhaustion or heart-failure, it may be necessary to give both strychnine and digitalis.

DELIRIUM TREMENS.

DEFINITION.—A peculiar form of confusional insanity produced by the excessive use of alcohol or, in rare cases, of other narcotics, characterized by the existence of tremors and an underlying condition of fear.

ETIOLOGY.—*Mania a potu* often develops after the suspension of a bout of heavy drinking; but the old idea that it is solely due to abstinence from alcohol is not correct, as the symptoms may develop in the midst of a debauch, and cannot be relieved by simply supplying alcohol. The disease is more common in men than in women, probably because drinking is especially a masculine vice.

MORBID ANATOMY.—There is no recognizable lesion in delirium tremens.

SYMPTOMATOLOGY.—In the mildest form of delirium tremens, the "horrors" of old drunkards, the symptoms consist of insomnia, with restless, broken sleep, tremulous hands, extreme depression of spirits, pronounced irresolution, and a weak, confused mental condition, with frightful imaginings and vain alarms.

The symptoms of the fully-formed disorder may appear abruptly, but in most cases are developed gradually out of the "horrors." The insomnia, which is at first partial, becomes absolute, whilst hallucinations of sight or hearing, and more rarely of touch, appear. The emotional depression increases and develops into a state of perpetual fear and terror. The hallucinations are always tinged with sadness or horror: disgusting objects, such as snakes, toads, rats, mice, and other unclean creatures, climb about the bed or over the person; whilst voices of threatening, of reproach, or of foreboding are heard. During the delirium there is unnatural loquacity, and often pronounced restlessness. The delirium may at first be paroxysmal,—worse at night, better in the day,—but finally it becomes constant. The patient may put on an appearance of violence, and may even attack an attendant, always, however, because such attendant is the subject of his delusion, the violence being a battle of despairing defence and not a combat of aggression. Perhaps the most characteristic feature of the delirium is that which is common to most forms of confusional insanity,—namely, the extraordinary versatility of the false ideas. Tremor is usually present from the first; it is irregular, and most frequent in the arms, face, and tongue, but it may attack the whole body, and is always increased by efforts at movement.

In the early attacks of delirium tremens, occurring in very robust people, when all the mucous membranes are irritated, and when there is probably an irritation of the brain and its meninges by excrementitious materials in the blood, there may be a strong and excited pulse, but in the vast majority of cases the disease is plainly asthenic, with loss of muscular power, and with a pulse which is rapid and feeble, or, if it preserve an appearance of strength, very soft and compressible. The temperature is usually from one to three degrees above the norm: a record of 104° F. points strongly to the existence of some complication. Free sweating, scanty, albuminous urine, and complete anorexia are common features of the disease.

The recovery from delirium tremens may be sudden, after a prolonged sleep; or it may be gradual, through a series of restful nights; or the dreams may end in death or in chronic insanity.

In some cases of delirium tremens the symptoms are different from those which have been described. The patient has apparently control of himself, receives his physician with a quiet, gentle courtesy, and answers questions without irritation; but he is evidently preoccupied, occasionally turning his head or casting furtive glances from one side of the apartment to the other; really, during the whole period he is seeing visions and hearing sounds, laboring under the profound apprehension of attack, watching always against the enemies of whose presence he is absolutely convinced.

Complications.—Any of the various diseases of the liver, kidneys, or other organs, commonly produced by the excessive use of alcohol, may complicate delirium tremens. Acute pneumonia is very apt to develop early, especially in drunkards who have suffered exposure. It may come on without cough or pain or other symptoms save increased frequency of breathing and increased elevation of the temperature: hence it is essential for the physician to examine the chest daily in every case of delirium tremens. Not rarely pneumonia precedes the coming on of the delirium tremens, which then must be looked upon as the complication.

DIAGNOSIS.—The peculiar forms of the hallucinations, the underlying emotional condition, and the tremors make the recognition of delirium tremens very easy, even when there is no history of the case.

When pneumonia occurs during a period of delirium tremens the type of the delirium may change, tremors may be lost, and the patient may become so violently aggressive as to lead to a mistaken diagnosis.

PROGNOSIS.—Death is very rare in the first attacks of delirium tremens, but, as most victims of the alcoholic habit do not reform, death is very frequent in recurrent attacks of the disorder. The prognosis, therefore, becomes more serious with each recurring attack, is greatly increased in gravity by the existence of any organic disease, and is very critical when pneumonia develops. It usually is the result of exhaustion or failure of the heart's action, which may be sudden. A temperature of 105° F. is rarely recovered from. When the delirium tremens complicates a severe traumatism it adds enormously to the fatality.

TREATMENT.—In the treatment of delirium tremens the first indication is for restraint to prevent injury by the patient to himself or to others. Freedom in a well-padded room may be allowed, but in the majority of cases such a room is not available, and properly constructed straps securing the person in bed are, in a violent case, much better than restraint by means of nurses, the strap exciting less antagonism than does an attendant, and being more steady and certain in its restraint. Thoroughly padded leather wristlets and anklets may be secured to the bed, and a loose chest strap may be employed in very violent cases.

The second indication is for the support of the system by means of highly nutritious and stimulating food. Milk, strong soups or beef-essence with eggs stirred into them just as they have ceased boiling, and similar liquids, usually constitute the best articles of diet. As the digestion is in these cases often deranged, it is essential for the practitioner to remember that the food which nourishes is not that which enters the stomach, but that which is digested: so that the effort should be by frequently repeated small portions of nutriment to get as much material worked up as possible. Often partially predigested food may be used with advantage. Again, the mucous membrane, which has been accustomed to the local effects of alcohol, is often simply beneficially stimulated by amounts of red pepper and other spices which would produce gastritis in a normal stomach: hence, even though the stomach of the drunkard is inflamed, highly seasoned food is usually of great service. The limit of the amount of food given in these cases should be that of possible digestion. When the stomach will not take food freely, rectal feeding should be employed.

In the medical treatment of delirium tremens the first indication is in most cases to relieve abdominal engorgement and to remove effete materials from the system. Three grains of ipecacuanha may be exhibited in pill form every fifteen minutes until free vomiting is produced, and, even if the patient is suffering from excessive nausea and vomiting, this practice is often of service. On the other hand, when great feebleness exists the use of such an emetic may be improper, and in rare, markedly sthenic cases veratrum viride given in drop doses of the fluid extract until it vomits may be advantageously substituted for the ipecacuanha. After the ipecacuanha has acted, a grain of calomel may be given every hour until free purgation is induced, and not rarely an effervescent mixture containing potassium citrate is very serviceable in acting as a depurant through the kidneys. Again, profuse sweating produced by pilocarpine or the hot bath may sometimes be useful.

The second indication for medical treatment is to quiet nervous excitement. For this the bromides and hyoscine hydrobromate must be relied upon. These drugs should be given steadily day and night at regular intervals, the hyoscine being withdrawn if found in any way to disagree with the patient.

The third indication is to produce sleep. For this purpose various hypnotics have been used. Sulphonal may be employed; paraldehyde has been exhibited; but the combination of chloral and morphine sulphate far exceeds in efficiency and general applicability all other hypnotics. The chloral (fifteen to twenty grains) and morphine (grain one-quarter to one-third) may be exhibited at bedtime, and repeated in half-doses at intervals of an hour, *pro re nata*, care being exercised not to overdo the exhibition of narcotics.

The fourth indication is to support the system by means of stimulants. One of the most important questions to be decided is as to the necessity of using alcoholic drinks. In many cases of delirium tremens alcohol does harm rather than good, and in the majority of cases its use is not essential. The moral reasons against its employment are very strong, and therefore commonly it is not wise to give it. On the other hand, in feeble subjects or in old alcoholics the exhibition of alcohol in some form or other may be necessary to the saving of life. In many cases of delirium tremens strychnine is serviceable as a stimulant, but when there is any fear of cardiac failure digitalis is the most reliable of all remedies. It must be given in very large doses, and usually enormous amounts are well borne. Various clinicians have claimed very good results from the exhibition of half-ounce doses of the tincture, but we think that a safer method is to give from ten to twenty minims at intervals of from two to four hours, watching closely the effect, and withdrawing the remedy as soon as any evidence of the digitalis pulse can be perceived.

Congestion of the lungs occurring in delirium tremens may be beneficially treated by ergot in large doses (thirty grains of the extract). It should be at once the signal for free stimulation, and for the exhibition of large doses of digitalis. Camphor is used to a considerable extent on the Continent of Europe as a stimulant. Musk certainly has a distinct but transitory power; it should be given in large doses (preferably fifteen grains in two ounces of emulsion with twenty minims of laudanum by the rectum) every six hours. Free counter-irritation by means of poultices containing mustard or by means of turpentine stupes is also serviceable. After from twenty-four to thirty-six hours the ergot should be withdrawn, and five minims of turpentine given in emulsion every two hours.

ALCOHOLIC INSANITY.

Closely allied in symptoms and probably also in basal cerebral condition to delirium tremens is the chronic alcoholic mental aberration to which the term alcoholic insanity is usually applied. Under the continuing influence of alcohol the brain performs its functions slowly and imperfectly. The mental movements become sluggish, the memory is impaired, the power of fixing the attention is diminished, and the control of the will is almost abolished. Usually with this condition there is a tendency to emotional depression, and often a peculiar suspiciousness which is the ground-work for delusions. In such a case, if the drinking habit be maintained, delirium tremens may result; and this delirium tremens may be recovered from, or may more rarely end in a chronic mental aberration,—an alcoholic insanity. In another set of cases the alcoholic insanity is gradually developed out of the condition described above without the production of a distinct delirium tremens; but even in this form of insanity the symptoms often resemble those of delirium tremens.

The hallucinations are very numerous, constantly changing, full of terror and disgust, as in delirium tremens, but are less acute and give rise to less excitement; in most cases they take the form of delusions of persecution: voices of reproach or of threatening, mocking faces, unclean beasts, tormenting devils,—these and similar hallucinations may drive the victim into a profound melancholy, ending in suicide. Very often, however, the mental condition in chronic alcoholic insanity is less violent, the subject simply being full of delusions of persecution which in a very large proportion of the cases have a sexual coloring or relate to poisoning. Almost universally the mind of the husband continually runs upon the sexual relations of his wife, until there is a fixed, overpowering delusion that she is unfaithful. This delusion may in turn lead to an outburst of uncontrollable jealousy and rage, so that wife-murder is not a rare result of alcoholic mania. Usually in alcoholic mania there is a substratum of fear, which in itself may lead to violence, but in some cases the subject is aggressive. It is in such instances that the marked relation between the presence of alcohol in the blood and the insane outburst can be seen. The maniacal drunkard may be apparently rational, certainly quiet and peaceable, when not under the influence of alcohol; but by a moderate dose of the poison he may be converted into a wild beast as murderously aggressive as a tiger. This, too, may happen when the man is capable of walking straight and of talking rationally on general subjects. In many such cases honest testimony has been given that the subject was neither drunk nor crazy when he committed the crime, although in fact he was in a condition of violent alcoholic insanity.

The form of alcoholic insanity described in the preceding paragraph is the ordinary one,—alcoholic lypemania with delusions of persecution. It is asserted that there is also an alcoholic megalomania, in which there are expansive delusions or hallucinations of sight and hearing, which in most instances relate to God and a future state. In an ecstatic exaltation the patient is enrapt by visions of supernatural beings, or basks in the presence of the Deity; ministering bands of angels speak words of comfort to him, or, it may be, the voice of God himself is heard in command or instruction.

PROGNOSIS.—Alcoholic insanity usually gets well if the symptoms are those of the ordinary type as given above, provided there be no renal disease or other serious complication. It should, however, be remembered that the habitual use of alcohol may aid in the development of an ordinary insanity, which, having other etiological roots, cannot be properly called alcoholic insanity, and may readily be incurable. A true alcoholic insanity which has lasted for a length of time under the habitual excessive use of alcohol, or which has followed numerous attacks of delirium tremens, may be permanent.

TREATMENT.—The treatment of alcoholic insanity consists primarily in the absolute withdrawal of the alcohol. There may be cases in which it is not safe to take away the narcotic at once, but we cannot remember to have ever seen one. Restraint should always be enforced, by isolation in an institution unless the patient's pecuniary circumstances are such that private isolation and abundance of nurses are possible. Such restraint is necessary for the purpose of enforcing the abstinence from liquor, but it is still more essential because the person suffering from alcoholic insanity is always a dangerous lunatic.

Hyoscine hydrobromate, the bromides, opium, or chloral, must be used judiciously to secure quiet and sleep. Blisters to the back of the neck are sometimes valuable. Hot baths and hot packs are often of service in allaying the general irritation. Aconite, digitalis, and other arterial sedatives or stimulants are to be used in accordance with the needs of the individual case. It is necessary to pay very careful attention to the digestive organs, as chronic gastritis and hepatic congestion are ordinary complications. As much simple nutritious food should be given as can be digested, the tendency of the disorder being distinctly asthenic. Quinine rarely does good, and is apt to irritate the stomach. Strychnine is often of great service, especially in the more chronic cases.

OPIUMISM.

The excessive habitual use of opium produces general failure of health, usually with derangement of digestion, and nervous symptoms which resemble those of neurasthenia to some extent. There are no characteristic phenomena, but when in any case of apparently causeless failure of health the patient at times is dreamy, with an indolent, quiet, *dolce far niente* manner, the practitioner's suspicions should be aroused. Not rarely great suspiciousness exists, and delusions of persecution often occur. Very frequently the delusions take a sexual tinge, and the belief in unfaithfulness on the part of a marital partner is not uncommon. On the other hand, in many cases there are no delusionary tendencies, even in the last stages of opium poisoning. The peculiarity of the beliefs is their unfixedness, so that the subject may seem one day entirely free from any disorder of mental action, and the next day may have a distinct delusion.

Infants, born of a mother who is a heavy opium-eater, often appear healthy at the time of birth, but suddenly, without apparent cause, within two or three days pass into a condition of collapse, ending in death. There can be little doubt that in such cases the cause of the collapse is the sudden withdrawal of the opium to which the child had become accustomed whilst in the uterus, and it would seem, therefore, a rational procedure cautiously to give opium to such a child for a time after its birth.

TREATMENT.—No confidence can be placed in the statements of the opium-eater, and it is essential for cure that such person be in a hospital or be confined to an apartment under the care of an absolutely reliable nurse, so that the orders of the physician can be strictly enforced. The basis of the treatment must consist in the withdrawal of the narcotic, and there are three ways in which this can be effected. First, the opium may be suddenly taken away; secondly, it may be taken away rapidly, but not suddenly; thirdly, it may be withdrawn very gradually. The first of these methods is undoubtedly in most cases efficient, but is often attended with grave danger of collapse, and has no distinct advantages over the plan of rapid withdrawal. The time required for the very gradual withdrawal of the remedy is too great for practical purposes, and the sufferings of the patient are too long drawn out. Unless the daily dose has been extraordinary or the patient is in a very feeble condition, it is safe to withdraw the narcotic completely in from seven to twelve days. An excellent plan is to direct that a solution of morphine or opium be prepared, and that whenever a dose is taken out an equivalent amount of water be added. In a successful case of a woman who took three pints of paregoric daily, we had prepared a gallon of paregoric and also a supply of paregoric without opium in it. Whenever a dose of the true paregoric was taken out the demijohn was filled up with the pseudo-paregoric. The chief symptoms that follow the rapid withdrawal are excessive malaise, insomnia, complete loss of appetite, vomiting, diarrhœa, and great feebleness. Very rarely these symptoms become so uncontrollable as to warrant alarm for the safety of the patient. Much may be done by proper feeding. The food should consist of highly nutritious, stimulating, and easily digested articles, and in severe cases should be liquid, such as milk, rich soups, etc. When the circulation fails, alcohol may be used, and much relief may be afforded by massage, and often by simple rubbing of the patient. General electrical stimulation and faradization of the muscles are often useful, not only by their effect upon the circulation, but also by distracting the attention of the patient from his sufferings. The use of the alkaloid cocaine as a stimulant has been recommended. We have seen apparently very good results from the free internal administration of the fluid extract of coca, but the use of hypodermic injections of cocaine is scarcely justifiable, on account of the danger of setting up the cocaine habit. If gastro-intestinal irritation exists, bismuth may be administered freely. The diarrhœa is usually controllable by mild astringents, especially if combined with sulphuric acid. If the bodily temperature falls, it must be maintained by external warmth. Potassium bromide, ammonium valerianate, Hoffmann's anodyne, and other similar feeble nerve-sedatives may be employed and give some comfort. Moral support and stimulation are essential, and any device which aids in passing the time of suffering is beneficial.

COCAINISM.

In America the third in importance of the narcotic habits is that which is known as "cocainism." In the symptoms there is nothing which is characteristic. Failure of health, indigestion, disturbances of circulation, with rapid pulse and sometimes shortness of breath, a pasty yellowish or even bronze skin, and various nervous disturbances, may be the results of the excessive use of the alkaloid. According to Mattison, hallucinations and delusions, with homicidal mania, are frequent, and a peculiar pathognomonic symptom is the impression that there is some foreign body under the skin, especially about the finger-tips, which leads the subject to dig continually at himself with some instrument.

In every case of abuse of cocaine which we have seen the subject has been addicted to other narcotic habits, and we believe that pure cocainism is very rare in the United States. According to our observations, it is entirely safe to withdraw the drug at once; at least we have so done in persons who were taking as high as fifteen grains of cocaine hydrochlorate a day, without the production of any severe symptoms. The general treatment should be very much like that for the morphine habit. The symptoms must be met as they arise, and the patient supported by all possible means.

SECTION II.

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

GENERAL SYMPTOMATOLOGY.

THOSE disturbances of the functions of the nerve-centres or of the peripheral nerves which constitute the symptoms of disease are best outlined under the headings of Disturbances of Motion, voluntary, involuntary, and reflex; of Coördination; of Sensation; of Vaso-motor and Trophic Functions; and of Intellection, including personal character and emotions.

DISTURBANCES OF MOTION.

Paralysis, or true loss of motor power, must be distinguished from the loss of motion due to local disease not connected with the nervous system. This pseudo-paralysis can usually be recognized by the fact that passive motion or local pressure gives pain. When, however, contractures exist, or when peripheral nerves are diseased, there may be a true paralysis although there is soreness to pressure and passive movements are painful.

An entire loss of power is known as a *complete paralysis:* a partial loss, as an *incomplete paralysis*, or a *paresis.* A *general paralysis* is a loss of power in the whole body below the head: it is never absolutely complete, since death from loss of power in the respiratory muscles must result before such condition is reached. The loss of power may, however, be complete in the arms and legs. *Hemiplegia* is paralysis of one lateral half of the body; but, as it is very rare for the respiratory muscles to be paralyzed, this term is universally used not only when the face, arm, and leg are paralyzed, but also when only the arm and leg are affected. Hemiplegia is almost invariably of brain origin. A *cross paralysis* is one in which one side of the face and the opposite side of the body are affected. *Paraplegia* is paralysis of the lower transverse half of the body, and is almost always due to disease of the spinal cord. *Monoplegia*—that is, paralysis of one part—may be *facial*, *brachial*, or *crural.* A *local paralysis* is a palsy of a single muscle or muscle-group. A *multiple paralysis* is an association of local paralyses,—*i.e.*, a paralysis of groups of muscles more or less scattered and having no direct connection. Monoplegia, local paralysis, and multiple paralysis may be due to lesions of the brain, the spinal cord, or the nerve-trunk. The brain lesion in such a case is almost invariably in the cortex, while the spinal lesion is in the ganglionic cells of the anterior cornua. The loss of movement which constitutes a paralysis can usually be seen at once; in paralysis of the face the mouth is drawn away from the affected side unless contractures have taken place in the paralyzed muscles, and in lingual paralysis the protruded tongue is thrust towards the paralyzed side. In paresis of the forearm it is customary to note the exact power of grasp by means of the dynamometer. Various apparatuses have been devised for testing loss of power in the leg, but in practice a sufficiently accurate judgment can be made by noting the extent of forced movements, the amount of endurance in walking or in standing on one leg, the ability to rise out of a chair, etc.

Convulsions.—Three types of convulsions are recognized: the epileptiform, or cerebral, in which consciousness is completely lost; the hysterical, in which consciousness is disturbed; and the tetanic, or spinal, in which consciousness is normal and reflex activity is grossly exaggerated. These varieties of convulsions grade one into the other. A detailed discussion of convulsions will be found in various articles, especially in those on Epilepsy and Hysteria.

Automatic Movements.—Automatism is the condition which sometimes occurs in epilepsy, the hypnotic state, etc., in which the subject performs seemingly voluntary purposive acts without clear consciousness or after-memory. A purposive act,* involving a more or less elaborate series of movements, performed as the result of an entirely irresistible impulse, is spoken of as *automatic*, although there may be complete consciousness during the whole time. Automatic acts must be clearly separated from choreic movements.

Reflexes.—For the performance of a reflex action there must be a complete arc composed of afferent nerve with its root, motor ganglion-cell, efferent nerve with its root, and muscle. Any disturbance of this arc may register itself in disturbance of reflex activity. A *deep reflex* is one elicited by irritation of a deep structure; a *superficial reflex* is brought about by a superficial irritation.

Of the *deep reflexes* the most important are the *patella reflex* and the *ankle reflex*. The patella reflex (*Westphal's symptom, knee-jerk*) is the movement which is elicited by striking the patellar tendon. In testing for it, it is better to have the leg hanging loosely over a chair or a cane, and to strike a sharp quick blow with the tips of the bent fingers or with a small rubber hammer prepared for the purpose; or the blow may be delivered with the foot placed upon the ground and the contraction of the rectus femoris be felt with the hand. Marie asserts that in many cases there is a simultaneous contraction of the adductors of the opposite thigh, and that in disease this may persist when the knee-jerk is lost. Any voluntary movement, such as clenching the hands, straining, or even winking, made at the time of the delivery of the blow, increases (technically, *reinforces*) the contraction. The knee-jerk is probably absent in about two per cent. of normal individuals; according to our experience it is quite frequently absent in the Latin race living in the tropics. It may also be absent as the result of disease not connected with the nervous system; any affection, such as emphysema, which produces defective aeration or oxidation of the blood, has a tendency to destroy it. In Dr. Russell's experiments upon the dog, asphyxia produced a primary increase followed by a loss of the knee-jerk. It may also disappear as the result of various diseases which lower the muscular tone, such as diabetes, also after diphtheria, even at a time when there is no indication of peripheral neuritis. It is, however, often exaggerated when the muscular system and the general vitality are much enfeebled, as in phthisis, and, it is asserted, even in acute disease such as typhoid fever. It may be increased or decreased by disease in or about the cerebellum, but lesions of the nervous system affecting it are usually situated either in the spinal cord or in the nerve-trunks.

---

* A purposive act, as the term is used in this work, is an act which is apparently but not really performed for a purpose.

Clonus is a to-and-fro movement produced by sudden stretching of a tendon, with or without a blow upon the stretched tendon. It is never present in normal individuals; it is very rarely producible under any circumstances in the elbow, wrist, and jaw, but may often be elicited in the ankle during spinal disease or hysteria. Ankle-clonus is best obtained by suddenly and forcibly flexing the foot with one hand placed upon the ball whilst the leg is held by the other hand. In doubtful cases a sharp quick blow may be struck upon the stretched tendon. In some persons a *biceps* or *elbow jerk* may be elicited by allowing the half-flexed arm to rest supinely in one hand and tapping the biceps or flexor tendon of the arm with the fingers or the hammer. A still rarer reflex is the jaw or chin reflex, which may sometimes be obtained by allowing the jaw to hang passively or gently supported in one hand, whilst a quick blow is struck upon the chin from above downward.

Paradoxical contractions are produced by suddenly relaxing the muscle, as happens to the anterior muscles of the leg when the foot is forcibly flexed by another person: they are probably never present in normal individuals, and are usually neurasthenic or hysterical in origin.

A *spasm* is an involuntary non-permanent contraction of the muscles, which must be separated from the *contracture*, which is a permanent shortening or contraction of the muscles. A spasm may be clonic or tonic. A contracture may be due to the disease of the muscles affected or of the nerve apparatus to which the muscle is tributary; or it may be the outcome of a long-continued lack of power in an antagonistic muscle.

A *tremor* is a to-and-fro vibratile movement, caused by more or less rhythmical contractions of antagonistic muscles. Tremors are of two kinds,—those which occur whether the subject be at rest or in motion (*persistent tremors*), and those which cease when there is absolute rest (*intention tremors*). A persistent tremor may, however, cease during sleep. Persistent tremors may be due to old age, to various poisons, such as alcohol, tobacco, mercury, etc., to general paralysis, or to paralysis agitans. The intention tremor is usually the outcome of a multiple cerebro-spinal sclerosis, although some toxic tremors, especially those of mercury, may simulate an intention tremor.

Choreic movements are irregular movements due to independent contractions of single or associated groups of muscles; they may closely simulate purposive movements, and probably in some cases are exaggerations of such movements, but they never form a complicated series of apparently purposive actions. They vary in intensity from a slight restlessness or a slight movement of the fingers or toes to the most severe constantly recurring motions. They may be confined to one or more groups of muscles (*local chorea*), or they may affect the entire muscular system (*general chorea*). Usually they are not regular, rhythmical, or consentaneous; in certain cases, however, movements more or less resembling tremors, but slower and more extensive, occur, and are known as *rhythmical chorea*. Choreic movements may be produced by lesions affecting any of the ganglionic cells in the cerebral or pyramidal tract, and are, therefore, no more uniform in their significance than is paralysis. *Generalized* choreic movements (*chorea* of many authors) may be caused by St. Vitus's dance, Huntingdon's disease, local irritation, organic disease of the nerve-centres, pregnancy, old age, and hysteria.

Hemichorea is a disordered, irregular, persistent movement of one side of the body, not ceasing during rest, exaggerated by voluntary movements or by concentration of the attention. It is distinguished from tremors by its irregularity and by the extent of the movements. A form of hemichorea is the so-called *athetosis* (ἀθετός, without fixed position), which is characterized by an incessant movement of the fingers and toes, and by the impossibility of maintaining these parts in any position in which they have been placed.

DISTURBANCES OF COÖRDINATION.

Coördination may be disturbed in the whole organization or in the arms or the legs separately. The first test of coördination is the so-called *station test*, in which the individual is placed strictly erect, with the heels and toes of the two feet closely approximated, when, if coördination be imperfect, the swaying of the body will be beyond the norm, and movement may become necessary. The test will be more delicate if the patient be required to stand on one foot. The patient should be further required to walk a chalk-line, to walk backward, to turn suddenly, then with the eyes shut to stand and to walk backward and forward. Care is sometimes necessary to avoid mistaking the awkwardness that arises from muscular weakness or stiffness, or from the vertigo of cerebral disease, for true loss of coördination. The peculiar form of disorder of coördination known as *titubation* is described under Cerebellar Disease.

Disturbance of coördination in the arms produces the loss of power of executing delicate movements. The usual test is for the patient to close the eyes, clench the hands with extension of the index fingers, and then, opening the arms widely, rapidly bring the index fingers together, first with the eyes open, then with the eyes shut. If coördination be imperfect, the points of the fingers will not come in contact.

DISTURBANCES OF SENSATION.

In the study of sensation, *algesia*, or the power of feeling pain, must be distinguished from *sensibility*, which itself is naturally divided into the sense of *touch*, or the power of recognizing contacts; *electrical sensibility*, or the power of recognizing electrical currents; *thermic sensibility*, or the power of recognizing the temperature of bodies; *pressure-sense*, or the power of recognizing weights; and *muscular sense*, or the power of estimating muscular movements. An æsthesiometer is used for the testing of the sense of touch; it may consist of ordinary compasses with blunted points, furnished with a graduated scale, or of a pair of blunt points one of which slides upon a graduated bar. When applied evenly and simultaneously to the surface the two points may be felt as two or as a single point, according as they are more or less widely separated. The sensibility varies not only in different parts of the skin, but also in the same portions of the skin in different individuals; usually, therefore, it is better to compare the affected part with the opposite side of the body: any wide deviation, however, from the following scale may be regarded as abnormal: the upper surface of the tongue, 1.18 mm.; the tips of the fingers, 2.25 mm.; the side of the first phalanx, 16 mm.; the back of the hand, 3.1 mm.; the upper arm and the thigh, 3.7 mm.

Thermo-æsthesiometers are used for testing the thermic sensibility, but a series of test-tubes filled with water of different temperatures will afford all the necessary apparatus for the purpose. The most accurate temperature range lies between 81° and 86° F., then between 91° and 102° F., and, lastly, between 57° and 80° F.; above or below these limits the sense is lost in the sensation of pain. According to Nothnagel, the smallest perceptible differences of temperature are the following: on the breast, 0.72° F.; on the back, 1.62° F.; on the back of the hand, 0.54° F.; on the palm of the hand, 0.72° F.; on the arm, 0.36° F.; on the back of the foot, 0.72° F.; on the lower extremities, 0.90° to 1.08° F.; on the cheek, 0.36° to 0.72° F.; on the temples, 0.54° to 0.72° F. Few normal individuals, however, recognize temperature so accurately. Pressure-sense is tested by placing graduated weights upon the hand, foot, etc., whilst lying upon a firm hard surface. Various apparatuses have been devised, but it is possible to make a regular series of weights which will suffice by partially filling shot-gun cartridges with shot. The muscular sense may be tested by noting the power of the patient to recognize the amount of various weights when lifted; it is usually better to test one hand against the other than to rely upon the accuracy of the patient's statements as to the amount of the weight lifted.

Loss of sensibility is spoken of as *anæsthesia;* excessive sensibility, as *hyperæsthesia.* Hyperæsthesia, as the term is commonly used, must be distinguished from simple tenderness; it is a condition of excited functional activity of the nerves of the skin or other parts with which contact can be made, not dependent upon any local disease. *Paræsthesia* is the term applied to all forms of abnormal sensation which are not actual pain, such as formication, or the feeling of the crawling of insects, the sensation of running water upon the skin, etc.

VASO-MOTOR AND TROPHIC DISTURBANCES.

Vaso-motor disturbances produce alteration in the color of the affected part, and usually in the temperature. Trophic alterations cause changes of form and structure which are readily recognized by the eye, so that no further discussion of them here seems necessary.

DISTURBANCES OF INTELLECTION.

In the outline sketch of mental diseases such as is alone possible in the present volume, the human intellectual faculties may be separated into the Will, the Intellectual Faculties proper,—such as Memory, Reason, Imagination,—the Emotions,—such as Fear, Anger,—and finally Character. For our present purposes the alterations of these functions produced by disease may be studied under the headings of Excitation, or increased functional activity; Depression, or lowered functional activity; and Perversion, or perverted functional activity.

Will.—The will is the inhibitory function of the brain. It arrests or dismisses thought and intellectual activity in general; it checks or keeps down emotions. Man can control a passion by the will, but he cannot directly will himself into a passion. He can stimulate, if he desire, an emotion by bringing before the mind thoughts which act as a stimulant to this emotion. Abnormal excitation (*hyperbulia*) of the will—*i.e.*, of the inhibitory brain function—is a somewhat rare phenomenon, difficult of recognition. The excessive obstinacy and self-assertion often seen in mental disorder are usually rather the outcome of a weakened will than of an overpowering egoism, the person being obstinate or aggressive because his will is enslaved by a lower intellectual or emotional nerve-centre. Thus, in melancholia inflexible obstinacy may result from the despotism of a depressive emotion, or a dominant idea may absolutely rule the individual. In hysteria the will is probably always feeble, but the persistence and apparent wilfulness of hysterical subjects are proverbial. Again, in chronic alcoholism the extraordinary persistency of the subject in the pursuit of alcohol is the outcome of a weakened will which is dominated by an appetite.

Abnormal weakness of the will (*abulia*) may be the result of acute illness, starvation, intoxication, age, chronic disease, or any other influence which lowers the nutrition of the brain-cortex. Concerning perversion of the will we have no knowledge.

Intellectual Faculties.—Absolute increase of mental power is very rare, and is never present in any advanced stage of disease. Its subject has a passion for intellectual labor, with an abnormal power of accomplishing: without fatigue and without pain the attention is kept concentrated for hours, both the quality and the quantity of the work being beyond that which is normal to the individual. Mental exhilaration is probably always associated with acute hyperæmia, and usually with pronounced insomnia. It may come on as the result of protracted mental labor, especially when stimulants have been employed, and may be the prodrome of severe mental disease, such as acute encephalitis or paretic dementia. It is always a very dangerous condition, and should be the signal for immediate cessation of mental effort and for careful medical treatment.

Failure of the mental powers (*amentia* when complete, *dementia* when partial) is a very common result of brain-disease. In its earliest beginnings mental failure usually reveals itself in loss of memory and of the power of fixing the attention. Fixation of the attention depends upon the exertion of the inhibitive power of the will in repressing distracting thoughts and shutting out new perceptions. Persistence in it is a large feature in all severe intellectual work, and when the brain is becoming exhausted, mental toil grows more and more irksome, not simply nor even chiefly because the reasoning faculties labor with difficulty, but because the will is unable to shut out the influences of distracting thoughts, emotions, or perceptions. Failure of memory and failure of the power of attention may coexist, and, if of severe type, in their coexistence usually indicate organic brain-disease. Failure of memory, when very pronounced, even if it exist by itself, usually depends upon incipient brain-disease, but the loss of the power of fixing the attention, when it exists alone or is accompanied by only a slight failure of memory, is commonly due to cerebral asthenia.

Incoherence may be due to mental excitement or mental depression, constituting the so-called acute and passive incoherence. In acute incoherence a heightened or irregular cerebral activity manifests itself in an excessive rapidity of intellectual acts lacking connected sequence. Before one idea is fully conceived, much less expressed in words, another rushes into existence and demands utterance, so that a confusion of talk results. Passive incoherence is, on the other hand, due to a lack of mental power which prevents the completion of the mental act, or, at least, its expression in words. In the acute incoherence of the raving maniac the rapid utterances are but the hints of the infinite crowd of jostling ideas; whilst the slow, confused, disconnected, hesitating words of the demented are evidently the outcome of a failure to conceive. It must, however, be remembered that in many cases mental excitement coexists with failing power, so that a mixed type of incoherence is produced.

Under the heading of Perversions of the Intellectual Powers we shall discuss certain phenomena seen in mental disease which can hardly be said to be due to exaltation or depression of the mental functions, although they are often associated with a true loss of mental power.

A sensation which is produced by an external object is spoken of as *objective.* A sensation which is the result of some change in the sense organs and is independent of external objects is *subjective.* An external object may give rise to such a distorted, misleading perception that the subject really says or feels or hears that which has no existence, as in mirage. Such a distorted perception is known as a *false perception*, or sometimes as an *illusion.* If, however, a perception be entirely subjective,—*i.e.*, if it be the conscious recognition of a sensation which is not due to any impulse received by the perceptive apparatus from without, but arises within the perceptive apparatus itself,—it is an *hallucination.* In nature illusions and hallucinations grade into one another, there being no sharp line, on the one hand, between pronounced illusions and slight distortions of objective sensations, nor yet, on the other hand, between hallucinations and illusions which have been provoked by the slightest and most indefinite of external stimuli. An hallucination has no definite diagnostic import. It may be due to the direct or indirect action of a poison, to cerebral exhaustion, or to peculiar conditions of the nerve-centres produced by various agencies. An hallucination does not necessarily depend upon or have connection with intellectual unsoundness. It is, however, often associated with such unsoundness, because the condition of the sensory brain-tract which produces it is apt to accompany a similar condition of the higher or intellectual centres. It often affords a test of the condition of the upper brain-centres. If the judgment fails to correct the testimony of the disordered sense by impressions derived from other senses, the subject is of unsound mind. If an individual, for example, cannot be made to believe that the vision which he sees or the false voice which he hears has no existence, then is his judgment dethroned. It is not the seeing of the vision, but the loss of the power of weighing evidence, which is at once the outcome and the proof of the intellectual degradation. Under the circumstances just spoken of, the hallucination gives rise to a *delusion*, a term which as used by the alienist is a synonyme for *insane delusion.* A delusion may be defined to be a faulty belief concerning a subject capable of demonstration out of which the person cannot be reasoned by methods which should be adequate. An insane hallucination is a false fixed perception ; an insane delusion is a false fixed belief. The existence of a delusion is commonly considered proof of insanity ; but, whether it be an hallucination or a delusion that is in question, the real proof of mental unsoundness is the failure of the judgment to correct the disordered perception or belief. In either case the essence of the insane mental state is the loss of power to receive and weigh adequate evidence. The nature of delusions varies so indefinitely as almost to defy classification, but the most important forms of delusions are, first, expansive delusions ; second, hypochondriacal delusions ; third, delusions of persecution.

An *expansive delusion* is an exaggeration of greatness or goodness or power ; it usually concerns the personality of the individual. Thus, a man having a delusion of grandeur believes that his prowess is irresistible, his wealth incalculable, or his future prospects unbounded. *Hypochondriacal delusions* relate to disease of the person, and of all delusions are those in which the gradations between the sane and the insane belief are most subtile. Almost every chronic invalid exaggerates his symptoms, and even the man who clearly has hypochondriacal delusions often has some physical basis for his beliefs. *Delusions of persecution* are those in which the patient believes that he is the object of the active antipathy of his fellows. They always constitute an element of danger, their subject being impelled by motives of revenge or of fear to kill the persecutors. They are especially dangerous when the delusion attaches itself to one or several individuals and is not generalized.

When the subject of a delusion reasons about and defends more or less logically his delusion, such delusion is said to be *systematized.* Thus, if a person who has a delusion that his soul is lost simply reasserts his belief under opposition, and assigns no reason for it, his delusion is unsystematized ; but if he affirms that he has committed the unpardonable sin, and quotes Scripture to show that his doom is, under the circumstances, the proper one, then is his delusion systematized. Great diagnostic value is attached by some writers to the distinction between systematized and unsystematized delusions. There is, however, in fact, every gradation between the most thoroughly systematized delusion and that which is most completely isolated ; so that in this as in all other respects the two great groups of insanities hereafter to be spoken of pass insensibly into each other.

An *imperative conception* is a general idea which, usually without obvious cause, arises in the brain of a person and dominates his action, although its falsity is recognized by the individual. The imperative conception may give rise to the *morbid impulse,*—that is, a dominating desire to do a certain act. Frequently, however, the morbid impulse exists without its being possible to discover any imperative conception. The act which results from the morbid impulse is spoken of as an *imperative act*. It is clear that the imperative conception is not necessarily a delusion, but when the patient fails to recognize the untruthfulness of the imperative conception the conception becomes a delusion. To illustrate what is meant by an imperative conception, morbid fears, as among the most frequent forms of this disordered mentality, may be selected. These morbid fears may be an exaggeration of a normal feeling. Thus, an exaggeration of the natural dislike for filth gives rise to *mysophobia*, or the fear of contamination, but the overwhelming horror of walking under an open sky (a common form of these fears) seems to be based upon no natural feeling.

If the sufferer from a morbid fear clearly perceives that his fear is not natural and has no basis in fact, there is no delusion, although the morbid fear may dominate the acts of the individual. Thus, a mysophobic will refuse to open a door, to pick up any object, to handle any money except it be new, to shake hands, etc., all simply through a groundless fear of contamination (the true nature of which he recognizes), or a person having the fear of leaving things crooked will spend hours in picking up and laying down an object; here a distinct morbid impulse grows out of a morbid fear.*

By the use of the word *mania* as a suffix, numerous names have been formed which are sometimes incorrectly used as denoting the morbid impulse. It must also be clearly borne in mind that these so-called manias are not distinct diseases. In *pyromania* the morbid impulse is to start conflagration; in *kleptomania*, to steal; in *arithromania*, to be perpetually making calculations. By a similar fallacious nomenclature, morbid desires or exaggeration or perversions of natural appetites give rise to a number of so-called manias. Thus, the condition of uncontrollable sexual excitement is known in the female as *nymphomania*, in the male as *satyriasis*. *Erotomania* is a condition in which there is the appearance but not the reality of sexual excitement; the subject of it conceives (or believes that he has conceived) a strong attachment for some person of the opposite sex whom, probably, he has never seen, and lives in an attitude of perpetual worship of his inamorata. Whilst satyriasis leads to sexual excess and to rape, erotomania is a platonic affection, which involves rather the higher conceptive sphere than the lower nerve-centres, and leads to sexual abstinence.

---

* To many of these morbid fears names have been given by systematic writers. The fears, however, vary so greatly that it seems impossible to have any accurate system of classification or nomenclature. The following list, taken from Dr. Beard, portrays very well the absurdity of the attempt, and at the same time suggests some of the common forms of morbid fears:

Astraphobia, fear of lightning; Topophobia, fear of places (a generic term, with these subdivisions: Agoraphobia, fear of open places; Claustrophobia, fear of narrow, closed places); Anthrophobia, fear of man,—a generic term, including fear of society; Gynæphobia, fear of woman; Monophobia, fear of being alone; Pathophobia, fear of disease,—usually called hypochondriasis; Pantaphobia, fear of everything; Phobophobia, fear of being afraid; Mysophobia, fear of contamination.

Both imperative conceptions and morbid impulses are undoubtedly frequent in the insane, but they may exist in persons whose intellectual actions in other respects are within the limit of sanity, and in whom the judgment is not dominated by the conception, although the conception and its consequent morbid impulse may cause the person to perform actions which are against his judgment. To himself such a subject seems possessed by a demon whom he must obey. A very intelligent patient once aptly compared the controlling force of the impulse to the *besoin de respirer:* for a time it could be restrained, but finally it must be obeyed. Such persons may be successful business men and useful citizens, and it is evident that they should not be considered legally insane or be put under restraint unless the morbid impulse is of such character that it endangers the safety of others.

The relations of morbid conceptions and impulses to legal responsibility may involve questions of great practical difficulty. The victim of a morbid impulse cannot properly urge such impulse as a legal excuse for crime unless such crime has been committed in immediate obedience to the impulse. Thus, a person who had the morbid impulse to kill could not plead the existence of such an impulse as a defence for theft. Further, when the act has been committed because the actor has been forced to do it by a morbid impulse which he could not possibly control, the actor remains morally blameless; but who can tell in any concrete case whether the impulse was resisted to the uttermost? Moreover, the needs of society and the ease with which such impulses may be alleged or counterfeited very properly give us pause in attempting by them to excuse a criminal act; whilst the claims of humanity may draw towards mercy for the alleged criminal, the interests of general human life, the good of the great mass, may well urge the execution of the laws. Certainly, under all circumstances the clearest possible proof should be required that the impulse had existed and had been recognized by others than the sufferer himself on occasions previous to the commission of the crime; and, further, it should be made probable that the impulse was really morbid and irresistible at the time of the crime.

An intellectual attribute which underlies all intellectual work and carefully considered action is confidence in one's own mental processes. When this confidence is naturally slight, the individual is timid and indecisive; when it is excessive, he is bold and prompt in action. In certain diseased conditions this confidence may be entirely lost, giving rise to the so-called *delirium of doubt.* This may occur without any other demonstrable disturbance of the mental faculties, and reveal itself in action, the patient continually repeating the act because he or she is uncertain that it has been completed. Thus, in the case of a mother under our observation, a large portion of the night would be passed in changing the diaper of an unfortunate babe, because the woman was never certain that her memory of having changed the diaper was correct or that her present perception of the dryness of the diaper was accurate.

Emotion.—Human emotion may be depressed or exalted by disease; but we find that, owing to inaccurate thinking, there is in literature a complete departure from the proper use of the words "exaltation" and "depression" in regard to emotional condition. In advanced cerebral disease there may be a condition of true emotional enfeeblement, so that circumstances which would have affected most vividly the individual in his normal condition fail to elicit response. This mental condition should logically be known as emotional depression, but it is the *apathy* of writers. It is to be clearly distinguished from that condition which is produced by the excitement of the depressive emotions, such as grief,—a condition which, however, is the emotional depression of authors. A person suffering from melancholia is not in a condition of emotional depression in the correct sense of the term, but in one of emotional excitement,—*i.e.*, excitement of a depressive emotion. It is true that excitement of a depressive emotion is frequently associated with a general depression of the nervous system; but this is not always the case, and the victim of melancholia agitata is in a state of general nervous erethism or excitement. The use of the terms is, however, so fixed in language that it is impossible to escape from them without running the risk of confusion, and therefore we shall in the present volume use the terms "emotional depression" and "emotional excitement" in their ordinary meaning,—*i.e.*, as respectively representing excitement of the depressive emotions and excitement of the emotions of exaltation.

It is often necessary to distinguish between a true apathy or lack of emotional power and the pseudo-apathy in which the patient is rendered oblivious to external influences because he is overwhelmed by a fixed idea or by an internal emotional excitement. Thus, a man who believes that he is to be devoured by the flames of hell may be dumb through fear and despair, or, as the German alienists say, "thunderstruck."

Human character is the result of the balance between the will, the intellectual attributes, and the emotional forces of the individual, so that any disturbance of one of these correlated factors must produce a corresponding change in the character of the individual. Character is, therefore, always seriously implicated in mental affections, and intellectual or emotional disturbances so subtile as not to be readily perceived sometimes register themselves plainly on the dial-plate of character. Hence alterations of character are always significant, and are not infrequently the first evidences of serious disease. In studying any case of alleged but doubtful insanity, it is therefore of the greatest importance to contrast, if possible, the individual as he now is with himself as he was when in full, undisputed health.

CHAPTER II.

FUNCTIONAL NERVOUS DISEASES.

NERVOUS diseases may be divided into the organic and the functional, although, strictly speaking, there is no such thing as a functional disease, all diseases being, without doubt, attended with alteration of structure; but as there are numerous nervous diseases whose pathological basis is too delicate to be detected by our methods of examination, and as there are other nervous diseases not yet sufficiently studied for a conclusion to be reached, it is convenient to group together in a chapter like the present all nervous diseases whose pathology is not established. Again, in the present imperfect state of our knowledge it seems necessary to have articles upon certain symptoms, such as headache, vertigo, etc., because in daily practice these symptoms seem to patients to be distinct diseases, and require special knowledge and skill on the part of the practitioner. In the present chapter are therefore included Symptomatic Conditions, and Diseases of Unknown Pathology.

INSANITY.

DEFINITION.—Insanity is a condition of mental aberration sufficiently intense to overthrow the normal relations of the individual to his own thoughts and actions, so that he is no longer able to control them through the will; this condition being independent of known structural alteration of the brain.*

GENERAL CONSIDERATIONS.

The definition of insanity which has just been given is necessary rather for medico-legal purposes than for the immediate needs of the medical practitioner. Insanity is not a distinct disease, but an abnormal state, varying indefinitely in its intensity and separated by no tangible line from sanity. Its manifestations are simply alterations, exaggerations, or perversions of the normal faculties, and therefore offer nothing that is absolutely distinctive. Emotional depression deepens into a pronounced melancholia, emotional exaltation lifts itself into the highest mania, by insensible gradations, and who shall say where the dividing line is between the state in which the man is master of the mood and that in which the mood is master of the man? The insane morbid impulse is but an exaggeration of that which bids a man standing on the verge of some great height to plunge headlong, or which, spreading from breast to breast, fills a mob with reckless rage or scatters it in apparently causeless panic.

---

* Insanity as here defined does not include cases of mental aberration which are commonly known in the court-room as insanities, but in which there is a distinct organic disease; in other words, it does not include the so-called *Organic* or *Complicating Insanities.*

Insanity being a symptomatic condition and not a disease, it is incorrect to consider its different forms as distinct diseases ; but for the purposes of discussion it is necessary to associate cases in symptom-groups to which names are given. The naming of these symptom-groups has a distinct tendency to lead to the delusion that they are diseases ; hence melancholia, mania, etc., are continually written about as though they were of equal rank with typhoid fever or scarlatina, whereas they are simply parallel groups to diarrhœa, paralysis, or dropsy. That they are not distinct diseases is shown by the facts—first, that similar mental symptoms may be produced by various organic brain-diseases, and that one organic brain-disease will cause, or may cause, antagonistic forms of insanity ; thus, in paretic dementia, now there may be a maniacal condition, now a melancholic one ; second, that not only does every grade of case exist in nature, so that acute mania grades into acute melancholia without distinct line of demarcation, and cases not infrequently occur which may with equal propriety be referred to one or the other of these so-called diseases, but also in a single attack of insanity the form may change without appreciable cause, so that the patient to-day has mania, to-morrow melancholia.

The insanities included in the definition given above are divided into, first, Constitutional Insanities ; second, Pure Insanities.

CONSTITUTIONAL INSANITIES.

Definition.—Insanities which are produced by constitutional vice or by poisons acting on the whole organism.

The most important of the constitutional insanities are the toxæmic, the lithæmic, the epileptic, and the hysteric.

The *toxæmic insanities* are those produced by poisons not formed within the body. The only one of sufficient importance to require discussion in this volume is that caused by alcohol. For an account of it see the article on Alcoholism. For a discussion of epileptic insanity and of hysterical insanity, see the articles on Epilepsy and on Hysteria.

It is well known that lithæmia as well as a paroxysm of acute gout is often accompanied by a marked depression of spirits and a peculiar irritability which may be beyond the control of the sufferer. In some cases the mental symptoms amount to an insanity. Moreover, hallucinations, delusions, loss of mental power, indeed, almost any conceivable manifestation of a disordered mentality, may be directly or indirectly produced by gout. The true character of such attacks is to be made out by recognizing the presence of a profound gouty condition, whilst the treatment of the case is that for gout, with the superaddition of such narcotics (chloral, hyoscine, opium, etc.) as may be necessary to keep in check the mental disorder and to secure sleep if there be insomnia. In other words, the general management of the case is that of the form of insanity simulated, with the addition of a very active anti-gout treatment.

PURE INSANITIES.

DEFINITION.—Insanities which are not dependent upon diathetic conditions or upon poisons.

For the purpose of study, these insanities may be divided into two groups:

Functional Insanities comprise those forms or types which are liable to occur in individuals who have no distinct original mental warp.

Neuropathic Insanities are the outgrowths of original vice of brain construction; such vice usually shows its presence early in life in the character and mental acts of the individual.

It must always be remembered, however, that here, as everywhere in insanity, our divisions are largely arbitrary, and that in nature cases everywhere grade into one another. The varieties of the two insanities are as follows:

Functional Insanities.—Melancholia; Mania; Confusional Insanity; Terminal Dementia.

Neuropathic Insanities.—Constitutional Affective Insanity; Moral Insanity; Paranoia (Monomania); Periodic Insanity.

MELANCHOLIA.

DEFINITION.—An acute or chronic functional insanity characterized by the dominance of the depressive emotions.

SYMPTOMATOLOGY.—Melancholia usually comes on gradually, with insomnia, depression of spirits, malaise, and progressive neurasthenia. The characteristic symptom of the disorder is a psychical anguish or depression, not dependent upon extraneous causes, and so severe as to dominate the whole life, so that every perception is painful (*psychical dysthesia*), or all perceptions are flattened down by the absence of desire (*psychical anæsthesia*), or both mental acts and perceptions cause intense disgust (*psychical hyperæsthesia*). In the milder forms of the disorder there are no loss of the reasoning power and no delusions, the chief symptoms being the great depression and apathy which are produced by the subject being absorbed in his own distress and being paralyzed by the psychical pain which attends effort. In severer cases there are wringing of the hands and perpetual moaning and lamentations. There may still be no intellectual aberration, the patient when aroused talking well and reasoning well; but after a time delusions develop which in typical cases are unsystematized, and which may exist with or without hallucinations. Both hallucinations and delusions are always of the depressive type; the patient hears voices, but they are voices of reproach, of misleading, of threatenings. Hallucinations of sight sometimes occur, but are less common than hallucinations of hearing; they are always evil, demons of sorrow or woe, perchance lost spirits or avenging angels. Hallucinations of touch are rare, and those of smell are still more uncommon; if they occur, the touch gives pain or disgust, the odor is a sulphurous vapor or a horridly fetid exhalation.

In mild melancholia there may be no distinct sensory disturbances, but headache may exist, although more frequently the complaint is of emptiness, of pressure, or of indescribable distress in the head. In the height of the disease paræsthesia, irregular anæsthesia, and hyperæsthesia are often present. The most intense suffering is from a peculiar distress, referred to the upper chest (*præcordial anguish*), which seems to be an exaggeration of the cardiac distress sometimes produced in normal life by sudden and overpowering sorrow. Usually occurring in morning paroxysms, præcordial anguish may happen at any time and continue for many hours. The attacks are habitually abrupt, and the agony with its accompanying terror may so dominate the consciousness that it is obscured or lost in a wild delirium, in which, with a blind disregard of himself and others, the patient convulsively attacks and destroys all within reach (*raptus melancholicus*), stripping himself naked, breaking, smashing, cutting, tearing both persons and things, and perchance disembowelling himself, twisting off his own genitalia, or in some other way committing suicide. During a paroxysm the respiration is rapid and superficial, the heart's action irregular and feeble, the skin cool and white, and not rarely the paroxysm ends abruptly with a profuse sweat.

Insomnia is a common symptom, and what sleep the patient gets is broken, unrefreshing, disturbed by horrible dreams. The general nutrition is lowered, the breath foul, the appetite wanting, the urine scanty and heavily loaded with urates, oxalates, and phosphates, the muscular power greatly lowered. In severe cases there are progressive emaciation, subnormal temperature, dry cold skin, and cyanotic extremities.

Simple melancholia has been divided by writers into various forms, according to the character of the delusions. Thus, there are *Melancholia Religiosa*, in which the delusion is of personal damnation; *Melancholia Hypochondriaca*, with hypochondriacal delusions (a form which shades by insensible degrees into hypochondriasis); and *Melancholia Attonita*, a very severe form, in which the patient passes the time in a condition of partial or complete stupor, almost motionless and emotionless. In extreme cases cerebral action would seem to be completely abolished, but that an occasional anxious look, the wrinkling of the forehead, or other slight muscular contraction shows that there is still power of thought. In this form of melancholia rigidity, muscular contractures, and even catalepsy may be present. Sensation may be normal, but there is usually either anæsthesia or hyperæsthesia.

Melancholia Agitata is a severe melancholia, accompanied by a great excitement which may rise to a complete frenzy, differing from that seen in acute mania in being founded upon intense fear and terror.

Acute, subacute, and chronic melancholia are constantly spoken of, but the terms are arbitrary, there being no distinct forms of the disorder.

PROGNOSIS.—In melancholia the probabilities of cure are in direct proportion to the lightness of the symptoms. About sixty per cent. of all the cases get well, usually in periods of from three to twelve months,—sometimes after many years. A small proportion of the cases end in dementia, and death may take place from complications or even from pure exhaustion.

TREATMENT.—In the treatment of melancholia the first indication is for absolute bodily and mental rest. When there is marked exhaustion, without much restlessness, the so-called "rest-cure," in a more or less modified form, may be employed; but, unless the exhaustion be very extreme, some out-door life and exercise should be insisted upon. It should always be remembered that attempting to argue a patient out of his delusions and assertions or to combat his schemes is to excite and not to calm. The second indication is to restore as far as possible bodily health by meeting any concomitant disease, and by sustaining the bodily power by means of properly regulated tonics and nutritious diet. Overfeeding should be practised as the rule; almost invariably the patient should be given as much food as the digestive organs will take. The third indication is to suppress nervous excitement and induce sleep. Very commonly, hyoscine hydrobromate (one-sixtieth to one-hundred-and-twentieth grain) may be given regularly at intervals of from eight to ten hours, whilst in some instances the similar administration of extract of opium will act most happily. At night sulphonal, chloralamide, chloral, and other narcotics should be used to procure sleep. It is essential that these narcotics be given alternately, so as to avoid the danger of chronic poisoning by any one of them, and also to prevent, as far as may be, the system from becoming habituated to one narcotic. Alcoholic drinks are often of service, but in a long-continued case great moral danger attends their use. Prolonged hot baths or hot packs are often very effective in quieting agitation and procuring sleep. In all cases of melancholia there should be the greatest watchfulness against sudden suicidal or homicidal outbreaks.

MANIA.

DEFINITION.—A functional insanity in which there is great emotional exaltation dominating the individual.

PATHOLOGY.—The relations of mania with acute periencephalitis are still matters of grave doubt. They will be discussed under the heading of acute periencephalitis.

SYMPTOMATOLOGY.—Acute mania may be developed suddenly or be preceded by a prodromic stage of emotional depression lasting from a few days to several months, during which time the symptoms resemble those of a mild melancholia. When the maniacal stage is reached, emotional excitement develops rapidly, delusions and hallucinations appear, and in bad cases the patient raves incessantly, shouting out a perpetual stream of incoherent threatenings, revilings, obscenities, and blasphemies. There are pronounced loss of sensibility, almost complete insomnia, great sexual excitement, incessant activity, the patient rushing about his apartment, struggling with his attendants or mechanical restraints, destroying clothing, bedding, etc., smearing his excrement over his person, and passing whole days and nights in a fury. The maniac has great muscular strength and endurance, and abundant appetite ; but progressive loss of bodily weight usually occurs in spite of the enormous quantities of food apparently digested. The temperature is often somewhat higher than the norm, but rarely reaches 100° F.

In the mildest forms of acute mania, incoherence, irrationality, restlessness, hallucinations, and delusions, with marked insomnia and total loss of modesty, may be the only symptoms. *Hypomania* is a form of the disorder corresponding to the mildest cases of melancholia ; in it there are no hallucinations or delusions, but only a condition of emotional exaltation with a change of character, a peculiar egotistic hilarity, perpetual extravagances, restlessness, and increased sexual appetite with lessened control of the will-power, leading to great sexual excesses and a tendency to brutal violence.

PROGNOSIS.—Recovery may occur in a few days, but usually it is delayed from three to six months or even longer. Death takes place in about ten per cent. of the cases from exhaustion ; in about twenty per cent. the mental aberration passes into chronic mania.

Chronic Mania.—Chronic mania may develop from acute mania or come on gradually. It is characterized by incoherence of speech, lack of power of association of ideas, delusions, often increased activity of the perceptive faculties with hallucinations, and mental and physical excitement. Both in acute and in chronic mania the delusions are unsystematized. In most cases of chronic mania there are intermissions in which the symptoms are much less severe. Recovery rarely occurs ; usually, by a progressive failure of the intellectual power, the patient drifts into terminal dementia ; not rarely chronic mania changes into chronic melancholia.

TREATMENT.—The indications for treatment in acute mania are to support the patient and to quiet the nervous system : any antiphlogistic or reducing measures do great harm. Hyoscine hydrobromate is the most valuable of all the calmatives, but chloral, sulphonal, codeine, conium, and other narcotic drugs must be used to procure quiet and sleep. Counter-irritation, in the form of blisters to the scalp and the nape of the neck, may be used, but rarely accomplishes much good. Hot baths and packs are sometimes serviceable.

CONFUSIONAL INSANITY.

DEFINITION.—An acute functional insanity without distinct constant emotional depression or exaltation, with marked abatement of mental power, often, but not invariably, accompanied by hallucinations and great mental excitement.

SYNONYMES.—Primary curable dementia ; Stuporous insanity ; Delusional stupor ; Mania hallucinatoria ; Surgical insanity ; Puerperal mania ; Post-febrile insanity ; Mania following typhoid and other acute fevers.

ETIOLOGY.—Great emotional strain or shock, severe surgical operation, childbirth, various acute diseases (such as rheumatism, typhoid fever, diphtheria, epidemic influenza), famine, starvation (especially when accompanied by hardships), may any of them be the cause of confusional insanity.

PATHOLOGY.—The underlying condition of confusional insanity appears to be a peculiar exhaustion of the cerebral cortex. The most careful microscopic examinations by thoroughly capable observers have proved that there is no demonstrable congestion or inflammation of the brain cortex, and thus far no one has been able to detect any characteristic changes in the ganglionic cells of the brain.

SYMPTOMATOLOGY.—In extreme cases of confusional insanity* (*Primary Curable Dementia*) there is almost complete paralysis of all the functions of the brain cortex, so that the patient remains in a condition of more or less pronounced stupor or stupidity, with progressive loss of weight, great reduction of the muscular strength, a feeble, small, quick pulse, and various shifting kaleidoscopic anomalies of vaso-motor innervation, such as wandering œdematous swellings. All the reflexes and the sensibilities are diminished, the pupils react feebly and slowly, the bodily temperature is subnormal, the urine is scanty and loaded with phosphates.

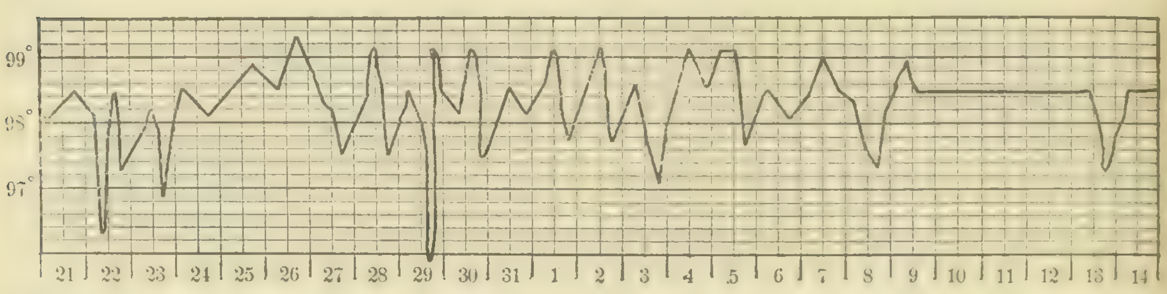
In milder cases of confusional insanity (*Hallucinatory Insanity*) there is depression of consciousness or of emotion shown in a peculiar quietude or apathy,—replaced by stupor in more severe cases. An almost characteristic symptom is the peculiar mental confusion, which may show itself in the inability of the patient to talk coherently and consistently or to follow out any train of thought, or may be so pronounced that the subject fails to recognize his friends or his surroundings. Along with these symptoms of mental failure there are frequently extraordinary and vivid hallucinations, so that the patient is unable to distinguish between subjective and objective sensations, realities and imaginations being intermingled in his consciousness into a hopeless chaos. The delirium of confusional insanity may counterfeit that of acute mania, the patient being perchance violently erotic or excessively aggressive; the aggressiveness, however, probably always rests upon the foundation of fear, the patient making the assault in self-defence,—that is, with the idea of protecting himself from an enemy.

---

* There is a rare form of confusional insanity, ending in death, simulating acute periencephalitis. See article *Acute Periencephalitis.*

The physical conditions of confusional insanity are always those of feebleness and exhaustion; the temperature disturbances in severe cases are pronounced, there is usually either an habitually low temperature or a marked tendency to paroxysms of subnormal temperature, with in many cases very pronounced febrile reactions; the swing of the temperature is remarkable for its irregularity and its activity.

FIG. 2.



Temperature curve of a case of confusional insanity. The figures at the bottom represent the days of the month, beginning with May 21.

DIAGNOSIS.—In most cases the true nature of confusional insanity is easily recognized. The history of the outbreak having been preceded by an exhausting disease, by famine, by traumatism, by emotional shock, the failure of bodily nutrition, the general depression of muscular and nerve force, the absence of dominating emotional excitement, the tendency to apathy or stupor, the peculiar mental confusion, the shifting character of the hallucinations and delusions,—all these form a picture which resembles that of no other disease.

PROGNOSIS.—The prognosis in confusional insanity, provided there be no pre-existing bodily lesion, such as unsound kidneys and degenerated arteries, is highly favorable. Krafft-Ebing gives the recovery at seventy per cent.; in our practice the proportion has been even larger. The recovery is usually complete, but the mental powers may be enfeebled; there is never a reasoning insanity nor a state resembling that of paranoia produced. When the attack has been caused by a very sudden and overwhelming emotion and the mental powers are completely lost, the outlook is very grave.

TREATMENT.—In confusional insanity the treatment should be carried on outside of an insane asylum, unless the pecuniary position of the patient makes it impossible to get a sufficient supply of trained nurses, etc. The great indication is for support; there is usually no appetite, but the powers of digestion are commonly much better than they seem, so that forced feeding is well borne. The most nutritious food, especially milk and raw eggs, should be given at intervals of two or three hours, in as large quantities as the stomach will tolerate. It is essential that the bodily warmth of the patient be maintained and sudden failures of temperature and collapse guarded against. External heat, by means of the hot-water bed, or bags of hot water, or the hot bath, should be used whenever the temperature falls below 97.5° F. In bad cases the rest must at first be absolute ; even massage increases the exhaustion. Later in the treatment, massage and electrical stimulation of the muscles may be of great service. During convalescence the best results are sometimes obtained by removing the patient to the sea-shore, mountains, or other places of resort ; but all over-exertion must be avoided. Drugs are used—first, to increase the nutrition, especially of the nerve-centres ; iron combined with bitter tonics in small doses, strychnine given in ascending doses to the limit of physical tolerance, and phosphorus continually exhibited in such small doses ($\frac{1}{120}$ to $\frac{1}{150}$ of a grain) as not to disturb the digestion, are the best remedies : second, to quiet delirious excitement and to obtain sleep ; the bromides are to be avoided, as they not only depress the functions but probably also the nutrition of the cortical cells of the brain ; hyoscine is of great value, cannabis indica may be essayed, and chloral, sulphonal, and opium may be useful as hypnotics. Sometimes an acute delirium yields to blistering of the scalp. The bowels should be kept free, but there should be no purgation.

TERMINAL DEMENTIA.

Almost any acute insanity may be followed by a chronic condition of mental weakness having no distinctive characteristic of the original insanity. The completeness of the mental ruin varies indefinitely. In some cases the individual is little more than a vegetating automaton ; in other instances the dement is restless, full of obtrusive or destructive activity, overflowing with animal spirits, possessed, it may be, by a peculiar egotism, but useless for any purpose requiring consecutive action. Again, in some cases there is a childish weakmindedness. No medical, hygienic, or moral treatment is of any avail.

NEUROPATHIC INSANITIES.

Insanity, nervous diseases, syphilis, poverty and the lack of the necessities of life, dissipation, excessive luxury,—these and other similar active causes in the parent lead to degradation in the offspring ; whilst the multitudinous ills which are possible to the young life, especially in the midst of extreme poverty, act upon the child itself, so that there is a peculiar degradation of mental development or failure of development of the nerve-centres which produces a peculiar constitution known as the neuropathic and furnishing root-stocks from which spring criminals, lunatics, and a multitude of beings of whom the world wonders whether they should be considered sane or insane. Space is wanting to speak further of this subject ; suffice it to say that these children of degradation may have great intellectual power, and that it is impossible in many instances to decide how far the individual should be considered legally responsible for his acts. In common with most alienists, we believe that there are neuropathic subjects who should be considered insane although they have no actual delusions, and that these cases may be divided into two groups: first, Reasoning Insanity; second, Moral Insanity.

In reasoning insanity there may be cases with emotional exaltation and emotional depression corresponding to mania and melancholia. In this class are included those persons who suffer from imperative conceptions or morbid impulses. (See page 385.) It must be remembered that the sequence of cases from eccentricity to reasoning insanity without delusions, and from reasoning insanity without delusions to paranoia, is unbroken.

Moral lunatics are those who are not only devoid of a conscience but are actually driven by their natures to what seem to others horrible crimes. Among the moral insanities must be classed the so-called sexual perversions, which may be due to long-contined vice, but which may be congenital.

PARANOIA.

DEFINITION.—Neuropathic insanity with more or less pronounced systematized delusions and without cyclical changes.

SYMPTOMATOLOGY.—Paranoia may come on abruptly, but it usually develops slowly out of a character which from the very beginning of life has shown evidences of neuropathy, especially in its morbid egotism. Its course is essentially chronic, but frequently there are intermissions which may last for months and which sometimes seem to be almost complete. Exacerbations are also liable to occur, taking the form of stuporous dementia, or of violent hallucinatory delirium, or of a fierce mania. Paranoia very rarely ends in complete dementia, but rather in a condition of psychical weakness and good-natured stupidity, through which may be preserved in part the artistic or technical abilities originally possessed by the subject.

Paranoia may be divided into two forms, according to the time of its development. When it appears about puberty it constitutes the *hebephrenia* of authors. This psychosis frequently begins with symptoms of mild melancholia, with apathy and an hysterical desire for sympathy; in a great majority of cases the subjects are excessive masturbators.

Late paranoia may not appear until past middle life. In its most common form it is attended with delusions of persecution: at first the subject feels that the world is becoming hostile, then suspicion attaches in his mind to certain individuals or groups of individuals, and increases until it becomes a fully formed delusion, the paranoiac knowing that he is the object of persecution, but the form of persecution varying in his mind according to his environment. Usually the delusions have a sexual tinge, one of the most common being the belief that the wife or the husband is unfaithful. About the time when the delusions become fixed, hallucinations appear ; those of hearing are most frequent, but any one of the special senses may be involved. The emotional state is one of depression, but this depression is essentially different from that of melancholia, for, whilst the victim of melancholia believes himself deserving of his sorrow, to himself exaggerates his guilt and his humiliation, the paranoiac, depressed though he may be by his persecutions, knows that these persecutions are unmerited and rebels against them. Not infrequently, either in self-defence or to revenge himself for wrong, or perchance simply goaded into fury by a sense of injustice, the paranoiac assaults his fancied persecutor. In very many cases he continually attempts to assert his rights through the law.

A second form of paranoia is that in which the delusions take a religious form ; this madness almost invariably has blossomed out from an early character of excessive piety, and often ends in a condition of religious ecstasy, sometimes alternating with states of depression. A third form of paranoia is that which has already been spoken of under the name of erotomania.

PERIODICAL INSANITY.

Definition.—A neuropathic insanity in which the attacks come on at regular or irregular intervals.

Among the periodical insanities may be mentioned the so-called *Menstrual insanity*, in which the attacks return at the menstrual period ; *Mania periodica*, in which there are periodical attacks of acute mania ; and *Melancholia periodica*, a very rare disease, in which during the paroxysms the symptoms are those of ordinary melancholia with usually extraordinarily active suicidal impulses.

Certain cases with imperative conceptions or morbid impulses also belong among the periodical insanities. Thus, there have been instances of periodical kleptomania, pyromania, sexual perversion, dipsomania, etc.

In *Circular Insanity*, or *Cyclothymia*, the symptoms follow a more or less regular cycle through life. A melancholia may be followed by a mania and then by a lucid interval, which ends in a recurring melancholia, and so on ; or mania may begin the cycle, or the lucid interval may occur between the melancholia and the mania. There are no symptoms which separate the stages of a cyclical insanity from a similar mental condition having other causes : so that the diagnosis must rest on a knowledge of the existence of the cycle.

Treatment.—Neuropathic insanities have no specific treatment ; being ingrained, they are usually incurable, and require moral management, maintenance of the general health, and often restraint. The greatest care is necessary in the use of narcotics lest the narcotic habit be formed.

NEURASTHENIA.

DEFINITION.—A condition of lack of power of the nerve-centres, not dependent upon the existence of organic disease in any portion of the body.

ETIOLOGY.—Primary neurasthenia very commonly has for its predisposing cause an original feebleness of constitution of the nervous system. It may be produced by overwork, especially when this overwork is combined with emotional strain. As the endurance of human individuals varies almost indefinitely, overwork, as just used, is a relative term, meaning that the daily expenditure of nerve-force is greater than the daily income.

MORBID ANATOMY.—There is no recognizable change in the nerve-centres in neurasthenia. In many cases, undoubtedly, there is a peculiar vaso-motor weakness in these centres, which makes them exceedingly liable to congestions, as well as an exhaustion of the nerve-cells themselves.

SYMPTOMATOLOGY.—Neurasthenia may be local or general. Very frequently a local neurasthenia precedes the development of a general neurasthenia. Thus, a cerebral asthenia the result of mental overwork, or a sexual spinal asthenia the result of sexual excesses, may exist by itself, but in most cases the local weakness is soon followed by a general neurasthenia. Usually neurasthenia develops slowly, but it may develop abruptly. The symptoms vary in accordance with the portion of the nervous system most affected. They may be generalized as a loss of power of performing functional acts, associated with great irritability. Thus, loss of power of fixing the attention, slight weakness of memory, disturbance of sleep, sense of weight and contractions in the head, tinnitus aurium, asthenopia, depression of spirits, great distress on mental effort, are the usual manifestations of a brain-exhaustion, whilst failure of muscular power, of endurance, of sexual power, of vaso-motor power, of control over circulation and calorification, result from weakness of the lower nerve-centres.

In neurasthenia the vaso-motor symptoms are often pronounced. Excessive blushing on the slightest provocation or on the use of alcohol, cool extremities, occasional pallors, excessive sweating, especially at night and during sleep or during emotion or excitement, are ordinary symptoms. The heart is often very irritable, palpitation, shortness of breath, and exaggerated increase of the pulse upon exertion being usually present. Irregularity and intermission of the pulse are especially frequent when the subject has the tobacco habit or there is gastric irritability. Apical systolic murmurs are often present when there is no decided anæmia or cardiac disease. In most cases there is, at times, subnormal temperature, and not very rarely the daily range is from one to two degrees of temperature below 98.5° F. A very large proportion of neurasthenics are lithæmic and suffer much from neuralgia. Atonic dyspepsia, disordered hepatic function, and constipation or diarrhœa are often the result of the improper innervation of the digestive organs.

DIAGNOSIS.—The only difficulty connected with the diagnosis of neurasthenia is the danger of mistaking a primary neurasthenia for one dependent upon the presence of stomachic, intestinal, renal, or other organic disease. It is essential in every case that the examination for local or constitutional disease be rigorously complete.

PROGNOSIS.—The prognosis in neurasthenia depends chiefly upon how far the condition is dependent upon original constitutional feebleness. The more severe the symptoms and the more slow their development, the greater the length of time required for their relief.

TREATMENT.—In nervous exhaustion recovery can be obtained only through rest and food, aided by the use of remedies for stimulating nutrition. Minor disagreeable symptoms may be met as they arise by drugs. Strychnine, arsenic, and phosphorus given for a length of time are often of service as alterative nutrients, but the chief reliance must be upon hygienic treatment.

Local neurasthenia, whether existing by itself or as the foundation of a general neurasthenia, requires rest of the organ primarily worn out. Thus, in sexual neurasthenia sexual abstinence is absolutely essential. In brain-tire it is the brain which must be rested. To rest an over-wearied, excited brain is often not an easy task. In attempting it the effort should be to obtain the following results: first, the removal of all cares, anxieties, and brain-work, especially brain-work of such character as has been connected with the break-down; second, the maintenance of the interest of the patient, so that the past shall for the time being be forgotten and the present not be overweighted with irksomeness; third, invigoration of the physical health of the whole body, and especially of the nervous system. In order to obtain the first of these measures of relief, isolation of some sort is essential; for the second, mental occupation is usually required; for the third, fresh air, exercise, or some substitute is to be superadded to abundant food and rest.

The proper method of meeting these indications varies greatly, not only with the varying physical conditions and idiosyncrasies of patients, but also with their diverse domestic and pecuniary relations. To give detailed directions for every case is impossible, and we shall therefore limit ourselves first to simple cases of brain-tire in which the muscular strength is preserved; second, to cases of profound general neurasthenia.

In brain-tire travel is usually recommended, and travel affords, when properly directed, separation from old cares and thoughts, a maintenance of interest by a succession of novel sights and experiences, and the physical stimulation of fresh air and exercise. In bad cases general travel is too stimulating. Ocean-voyaging gives complete isolation, fresh air, mental stagnation, and, if the patient be fond of the sea, complete enjoy-ment. Camping in the wilderness offers also all these advantages, and as a further good the possibility of obtaining exercise in exactly the amount desired. The subject may live in his tent and be nursed and fed by his guide, or may do the work of a day-laborer. Quiet travel in the mountainous districts of foreign countries is often very efficient, but sight-seeing, and even visits to cities, must be avoided. The quiet of Switzerland or of the Tyrol may bring restoration when the bustle of London and Paris might complete the ruin. In all cases strict attention must be paid to the individual tastes of the sufferer in deciding what measures should be carried out.

There are cases of neurasthenia in which the slightest exercise, or even the unconscious effort and excitement of seeing personal friends, will do harm. In these cases the so-called "rest-cure" often acts most beneficially. It rarely gives permanent relief, but often lays the foundation for an eventual restoration by means of out-door life and exercise taken after a certain amount of strength has been gained. A word of caution seems necessary against the routine employment of this rest-cure. It is simply the carrying out of a principle, and, although in the pages of a book like this it is necessary to give a fixed formula, success in practical life will depend upon the skill of the practitioner in modifying this and adapting formulæ to the needs of the individual case. The principles of the rest-cure are absolute rest, forced feeding, and passive exercise. The rest must be for the mind as well as for the body, so that in severe cases complete and absolute isolation must be insisted upon ; and especially when there is a decidedly hysterical element is it necessary to separate the patient entirely from her friends. Under these circumstances there must be a well-trained nurse who is personally agreeable to the patient. The confinement would be very irksome to any except the most exhausted patient were it not for the daily visits of those engaged in the treatment. Further to provide against ennui, the nurse should be a good reader, so that under the definite instructions of the physician she can occupy a certain portion of the time in reading to the patient. In the worst cases the patient should not feed himself or herself or perform any of the acts of the toilet. Directly after breakfast the sponge-bath should be given by the nurse, the patient being between blankets. Hot water should be used, or hot sea-brine, and after each part has been sponged over it should be momentarily rubbed with a piece of ice, followed by brisk friction with a Turkish towel. The greatest care should be given to the question of feeding. The end to be attained is to give as much food as can be digested, without deranging the digestion by overfeeding. It is usually better to give the food, which must be both light and nutritious, at short intervals. In most cases milk should be used very largely, sometimes exclusively. Often, especially when there is a tendency to obesity or when the digestive powers are feeble, the milk should be skimmed. Frequently koumiss, matzoon, or other fermented milks are agreeable to the palate and stomach. More rarely peptonized milk is serviceable. Beef and other concentrated meat-essences are valuable as stimulants, and may be used especially as the basis of soups. Various farinaceous articles of food may be added to them, or if an egg be broken into the concentrated bouillon or beef-essence just as it ceases boiling, a nutritious and, to many persons, palatable dish is obtained. When constipation exists, oatmeal porridge, Graham bread, and fresh or dried fruits may be allowed if readily digested by the patient. In order to give a general plan of the dietary the following schedule of the daily life is appended. Such a schedule should always be put into the hands of the nurse, who should be required to follow it strictly. It must be so altered from day to day as not to weary the patient with monotony. It is especially important to remember that the diet must be carefully studied for each patient and be adapted to the individual requirements of the case. Success will in a great measure depend upon the practical skill and tact of the physician in this adaptation :

8 A.M. Rolls or toast ; cocoa, weak coffee, or roasted wheat coffee ; beefsteak tenderloin or mutton chop.

9 A.M. Bathing.

11 A.M. Oatmeal porridge or wheatena with milk, or a pint of koumiss.

12 M. Massage.

2 P.M. Dinner : bouillon with or without egg ; beefsteak ; rice ; roast white potatoes ; dessert of bread-pudding, blanc-mange, or a similar farinaceous article of diet.

4 P.M. Electricity.

5 P.M. Milk toast.

9 P.M. Half-pint of skimmed milk or koumiss.

In many cases the patient at first can take very little food, and it is very frequently best to begin the treatment with an entirely liquid diet, giving milk every two hours or using Liebig's raw-meat soup, with milk or plain farinaceous food, and only after a time gradually accustoming the patient to solid food. Not rarely a prolonged milk-diet is of great service. The rest-cure is indeed largely based upon a careful regulation of the food ; but a full discussion of the various dietaries to be used would require a treatise upon dietetics.

Exercise is of value in health by its stimulating the general nutrition, aiding the flow of blood back to the heart, and increasing the excrementitious output from the emunctories. In the rest-cure these effects are obtained in a more or less imperfect manner without the expenditure of the patient's nerve-force by the use of electricity and massage. The electrical current not only produces muscular contractions, but probably affects the tone of the minute blood-vessels. Its action is so decisive that, as has been shown by Dr. S. Weir Mitchell, it will often temporarily elevate the temperature of the whole body. The faradic current alone is used. It is applied in two ways : first, to the individual muscles; second, to the whole body. The séances should be daily, the operator beginning at the hand or foot and systematically faradizing each muscle of the extremities and trunk. The slowly interrupted current is generally preferable, but advantage is sometimes gained by varying the rapidity of the interruptions. The rule is to select that current which produces most muscular contraction with the least pain. The poles should be applied successively to the motor points of the muscles, so that each muscle shall be made to contract firmly and thoroughly. This process should occupy from thirty to forty minutes. The electrodes are then to be replaced by large sponges well dampened with salt water: one of these should be placed at the nape of the neck and the other against the soles of the feet, and a rapidly interrupted current, as strong as the patient can bear, should be sent through the body for twenty minutes or half an hour. In some cases the electrical programme may be so varied as to get a local stimulant action from the general current: thus, when the digestion is enfeebled and the bowels are costive, for a portion of the time one of the sponges may be placed upon the epigastric region. In women, when there is great abdominal and pelvic relaxation, one pole may be placed high up in the vagina. We have seen moderate long-standing uterine prolapse cured in this way. Some electro-therapeutists hold that great advantage may be obtained by galvanizing the cervical sympathetic ganglia, but we do not believe that these ganglia can be reached by currents of therapeutic strength.

Massage, like electricity, affects greatly the peripheral circulation, empties the juice-channels, and gives tone to the muscular system. It must be clearly distinguished from rubbing of the skin. It consists in manipulations of the cellular tissue and of such of the muscles as are not too deep to be reached. In order to lessen as much as may be the skin-friction by these manipulations, it is often well to anoint the surface with the oil of cocoa-nut or other bland fat. In practising massage it is essential to remember that the natural course of the venous blood and the lymph is towards the centre of the body; therefore all general massage movements should be practised in this direction. The manipulations are percussion, rolling, kneading, and spiral. They consist of movements made with the pulpy ends of the fingers and thumbs, and spiral movements with the whole hand so folded as to adapt its palm to the limb. In percussion the strokes should be from the wrist, and should be quick and short. It seems hardly possible, even by long, strong strokes, to affect the deepest muscles. In the rolling manipulations the effort is to roll the individual muscles beneath the pulps of the fingers. This manipulation may be varied by pinching the muscles, not the skin, and kneading them. In each case the aim should be that of intermittent pressure upon the muscles. The circular movements are to be in opposite directions with both hands simultaneously, the limb being grasped by one hand a little above the other, and a spiral sweep made up the limb, the ball of the thumb and the palm of the hand resting upon the patient, and the pulpy parts of the thumb and the fingers grasping the limb. It is especially such motions as these that affect the circulation of the lymph.

The length of time for which a patient should be kept in bed varies from three to six weeks. The getting up should be gradual, the time of sitting up and the amount of exercise being carefully increased from day to day. The electrical treatment should be rapidly withdrawn, but massage may often be continued with advantage every other day for some time. So soon as may be, the patient should be sent out of the city, to consolidate by out-door life that which has been gained.

HYSTERIA.

DEFINITION.—A functional nervous disorder, characterized by depression of the will-power and of the inhibitory functions of the nervous system in general, and by increased emotional activity and sensibility, with an infinitude of shifting polymorphic nervous disturbances.

ETIOLOGY.—The hysterical temperament may be a congenital form of neuropathy, or it may be produced by luxury, license, over-indulgence, a lack of out-door life and active exercise, etc., during childhood or early life. It has no direct relation with the sexual organs, but is frequently the result of the exhaustion produced by sexual excesses, especially in the young. In boys it is often caused by masturbation. Climatic influences affect greatly the development of the hysterical temperament, which is much more abundant among the French and Italian than among the Anglo-Saxon and Teutonic races. Hysteria may be developed in persons who have no distinct hysterical temperament by overwork, depressing emotion, long-continued severe pain, or the exhausting depression of disease. It is psychically contagious, so that a single hysterical individual will affect a whole school or ward. During the Middle Ages, when by misery, poverty, and religious excitement the ground had been especially prepared, whole communities became involved in this way in epidemics of religious madness: hence the Flagellants, Children's Crusade, etc.

MORBID ANATOMY.—There is no anatomical peculiarity of the nervous system in the most profoundly hysterical person that can be recognized, and space cannot here be afforded to discuss the numerous theories of the disease which have been suggested.

SYMPTOMATOLOGY.—Hysteria exists in almost every possible degree, and the variations of symptoms are so infinite that the only method practicable in a brief space is not to attempt any description of the disease, but to discuss under separate headings the separate symptoms or classes of symptoms. In ingrained hysteria there are usually certain physical peculiarities which at least suggest to the observer the temperament, such as the large, full, liquid eye with mobile pupil, especially when associated with a brilliantly clear skin and with the ever-changing whims or the slow, languid movements of self-consciousness.

Hysteria is practically divided into the minor and the major disease, the latter comprising cases in which there are violent epileptic attacks with automatic manifestations. Major hysteria is rare among Anglo-Saxons in either continent; whilst in the extreme southern United States, peopled by the Latin race, it is said to occur as frequently as it does in France.

Mental Symptoms.—The basis of the hysterical character is selfishness, which shows itself rather in the overmastering desire to be the centre of sympathy and admiration than in the indulgence of grosser appetite. The hysterical woman is self-conscious and self-centred, always occupied with her own needs and wishes. The will is also weak, the emotional nature extremely sensitive, and the tendency to impulse pronounced, so that the individual is almost devoid of self-control. Emotional instability and lack of control over the emotional nature by the will are two of the most characteristic manifestations of the hysterical state. With or without reason the patient laughs and cries, and, perchance on the slightest provocation, passes into the most violent paroxysms of laughter or of weeping, whose nature is recognized by every one. The morbid desire for attention and sympathy, joined with the extraordinary importance to the subject of everything that pertains to his or her personality, leads always to great exaggeration of symptoms, and not rarely to intentional simulation of disease. Especially common is it for the simulated symptoms to take the shape that will bring great personal attention on the part of the young and inexperienced physician and mayhap minister to the morbid sexual desires of the patient. To swallow pins and needles and to thrust them into the tenderest parts of the body, to fill the uterine cavity overnight with the bones of small animals, to distend the rectum with foreign bodies or with starch-jellies, to retain the urine with absolute recklessness of the suffering involved,—all in order that the person may be talked over and worked with by the doctor,—are common arts among hysterics.

In hysteria not only purposed but also unconscious simulation of disease is very frequent, the unconscious simulation being largely, if not altogether, the effect of an idea formed in the mind. The dread of some disease, a suggestion from the doctor himself, the sight of a peculiar symptom in some one who is really ill, may be as powerful as is wilfulness in multiplying the symptoms of an hysteric.

The confirmed hysterical temperament belongs among the neuropathies, and the mental condition in its highest development resembles that of the paranoiac, and is almost as incurable. Moreover, like the paranoiac, the hysteric is liable to explosions which may take the form of a violent acute mania or of a deep melancholia; indeed, hysterical mental disturbances may simulate any form of insanity, so that the diagnosis of the true nature of such a case must rest upon the previous history of the patient.

Disturbances of Consciousness and Motion.—Spasms, tremors, clonic movements, and paralysis in all forms and degrees are very common in hysteria, but besides these motor disturbances there are convulsive paroxysms with or without characteristic phenomena. In major hysteria the convulsion is usually but not invariably preceded by some warning, and it may be by a distinct aura. The patient generally falls gently, not abruptly as in true epilepsy, often with an initial scream; the face is pallid. The spasm is at first tonic, with arrest of respiration, which may continue until the swollen, turgid face indicates imminent suffocation. In from two to three minutes a furious clonic convulsion occurs, with bloody foam about the mouth, the movements preserving, however, to some extent the appearance of wilfulness, and the head and arms being dashed with seeming purpose against surrounding objects. The clonic convulsion is in a very short time followed by the characteristic stage of opisthotonos, in which the body is bent so violently into the arc of a circle that it rests upon the head and feet, or in severe cases upon the toes and the face, which latter from the excessive retraction of the head looks towards the floor,—*i.e.*, behind the body. Opisthotonos is by and by replaced by violent purposive clonic spasms, the patient suddenly leaping from the bed or rising into a sitting position, and as quickly falling back again into opisthotonos. This to-and-fro movement may occur with extraordinary velocity. The opisthotonic stage finally subsides or is suddenly interrupted by the emotional stage, when the patient assumes an attitude of intense emotion, not rarely the so-called posture of the crucifix, with outstretched arms and legs and widely opened eyes, dilated pupils, and an expression of intense religious ecstasy. Usually emotion changes from time to time; religious joy gives way to an intense voluptuousness, or to an outburst of terror, or to a passion of penitence, and so, now singing, now weeping, now rejoicing, now reproaching, the subject passes into a slowly perfected consciousness. Very commonly during the paroxysms there are hallucinations whose character corresponds with the emotional state.

Any one of the stages of the major hysteria just spoken of may be omitted, or may constitute the whole of the paroxysm.

In the so-called minor hysteria there is usually no distinct aura, but very commonly in the beginning a *globus hystericus* (a sense of constriction or of the rising of a ball in the throat). The emotional disturbance is generally well developed, and is prone to express itself by uncontrollable laughter or equally uncontrollable sobbing or crying. Clonic convulsions may occur, but the contractions are usually tonic, and more or less pronounced; wide-spread muscular rigidity is a very frequent and very characteristic phenomenon. Under certain circumstances the hysterical paroxysms take on peculiar features. A form which we have seen especially among children is *beast mimicry*, in which the patient bites or snaps or snarls like a dog, or crows like a cock, or in some other way imitates the movements and sounds of the lower animals. In *spurious hydrophobia*, with symptoms simulating those of the true disease, the true character of the attack is always revealed by the snarling, the barking, and the attempts at biting, which do not occur in true hydrophobia.

Hysterical somnolence may take the form of a *narcolepsy* (the subject being continually drowsy, but passing only the nights in profound slumber) or of a lethargy or trance. *Hysterical trance* usually, but not always, commences with marked hysterical symptoms. In the early days of the attack the face may be red and hot, the pulse regular and slow, and the bodily temperature somewhat elevated. Later, there is pallor of the face, with rapid feeble pulse, the respiration is above the norm in number, or so nearly abolished that the movements of the thorax can scarcely be traced, the muscular system is thoroughly relaxed, the eyes are open or closed, and the pupils are dilated. There may also be complete anæsthesia,—so that neither bright lights, nor loud sounds, nor pinching, nor cold, nor heat, will elicit response,—scanty or almost suppressed urine, protracted constipation, and a subnormal bodily temperature. In the most extreme cases the appearance is that of a corpse; the pupils are immovable and the cornea filmy, and it may be that no motion can be detected in the heart or chest, so that in many instances death has been announced to have taken place. In some cases of hysterical trance there is great muscular rigidity, with set jaws, or periods of rigidity may alternate with periods of relaxation.

Catalepsy is a form of hysterical lethargy characterized by a peculiar condition of the nervo-muscular apparatus which causes the body or the limbs to remain almost indefinitely in any position in which they may be placed. There is no power of voluntary movement, but the limbs are not rigid, bending under slight force with the plasticity of wax. During the whole period there is complete anæsthesia.

Hysterical sleep may last for a few hours or may endure for months, or even for years, provided the patient be regularly fed with liquid food. In some cases the patient awakes at regular intervals to take food. Considering the small amount of food taken, the bodily nutrition is sometimes surprisingly maintained, but in prolonged cases there is great emaciation.

Hysterical paralysis may simulate any form of organic palsy: the face is not often affected, though hysterical facial paralysis and hysterical inequality of the pupil or strabismus do occur. In any form of hysterical paralysis the muscles may be relaxed or contracted, and the reflexes abolished or increased. Thus, in hysterical paraplegia there may be normal, abolished, or exaggerated knee-jerk, and there may be ankle-clonus. In hysterical hemiplegia the paralysis is rarely complete, rarely affects the face, and usually is distinctly more severe in one extremity. Hysterical monoplegias are common.

Hysterical paralysis is very frequently accompanied with disturbances of sensation, such as hyperæsthesia,—more commonly anæsthesia. Hysterical disturbances of sensation may exist without motor paralysis.

The hysterical anæsthesia may take the form of a hemianæsthesia, but, like hysterical hyperæsthesia, is frequently irregular in its distribution. Large local areas, especially affecting the ovarian region of hyperæsthesia or anæsthesia, are characteristic of hysteria. Hyperæsthesia of the genitals is very frequent in the female, in whom it is usually associated with vaginismus and loss of sexual desire. A very common form of hysterical hyperæsthesia, which occurs especially in neurasthenic women, is the peculiar superficial tenderness over the vertebral column which is frequently, without reason, considered as a distinct disease, under the name of *spinal irritation* or *spinal anæmia.* In many of these cases the general hysterical symptoms are not pronounced.

The disturbances of sensation often affect the different forms of general sensibility. Thermo-anæsthesia is common, whilst wide-spread analgesia existing by itself is almost always of hysterical origin. This is also true of loss of electro-sensibility. The disturbances of sensation may be limited to a single organ, as the cornea of the eye, and may involve mucous membranes as well as skin and deeper tissues. Hysterical anæsthesia is usually accompanied by the so-called ischæmia. In this condition the surface is pale and the needle-prick or even superficial incised wounds do not bleed. In anæsthetic ischæmia is found the explanation of the alleged miracle that in the Convulsionnaires of the Middle Ages superficial wounds were not followed by loss of blood.

The special senses are frequently affected in hysteria. Sometimes there is a mere hyperæsthesia, so that the normal function is performed with pain. Thus, photophobia is both frequent and severe. A true functional exaltation, especially of hearing, does, however, occur. In hysterical hemianæsthesia the special senses are usually involved. Hysterical amblyopia is frequently accompanied by a concentric narrowing of the field of vision, and sometimes by a loss of color-sense, *achromatopsia.* There may be simply contraction of the color-field, or contraction of both form- and color-field, or partial or complete reversal of the normal color-sequence, and the contraction of the field for form or for color may be greater in one eye than in the other. According to the researches of S. Weir Mitchell and de Schweinitz, in America achromatopsia is rare even when there is distinct contraction of the visual field, and, when it does occur, usually takes the form of reversal of the normal sequence of the colors, so that red is the largest field.

A peculiar psychic involvement of the special senses is sometimes present. The thing put before the hysteric is seen or felt, but suggests no corresponding psychical idea, and hence is not recognized. This condition occurs along with hysterical sensory and even ataxic aphasia.

Hysteria frequently invades those functions of the body which are not directly connected with voluntary life. Cardiac irritability is very frequent and often very annoying; in some cases a violent pain in the cardiac region, shooting down the arm, accompanied by excessive rapidity and smallness of the pulse, closely simulates angina pectoris. Disturbances of the vaso-motor system are very common: of this nature must be considered the sudden "flushings," which may be wide-spread, unilateral, or local; also the peculiar local œdematous swellings which are often accompanied with so much cyanosis that there are great blueness and coldness of the surface (*blue œdema*), as well as the swellings and enlargements about joints which may closely mimic organic disease.

Possibly as the result of vaso-motor relaxation, hemorrhages occur from the nose or the stomach, and are especially prone to be severe when there is suppression of the menstruation. Care is sometimes necessary to avoid mistaking for a true hæmoptysis the bleeding produced by sucking or otherwise irritating the gums. At times even the trophic nerves appear to be affected: at least, cases of hysterical skin-irritation and hysterical gangrene and hysterical erythromelalgia have been reported.

The temperature rarely departs from the norm in hysteria, but according to the French writers there are three types of hysterical fever: in the first form the paroxysms are irregular, of long duration, and accompanied by various nervous disturbances; in the second variety the fever continues from one to four weeks, and is accompanied by disturbance of the nutrition, in some cases the whole course of the affection closely mimicking that of a typhoid fever; in the third form the paroxysms of fever recur with regularity, so as to give the appearance of a true intermittent fever. Rummo, according to Gilles de la Tourette, has found a decrease of urea-production in hysterical fever. Exaggerated temperatures, 120° or 130° F., have been recorded from time to time as occurring in hysterical patients. Most, if not all, of these high records have been due to skilful manipulation of the thermometer by a designing patient, but there is reason for suspecting that extraordinary local elevations of temperature happen in hysteria.

Respiratory disturbances are common; violent paroxysms of acute dyspnœa from hysterical laryngeal spasm may simulate true laryngismus stridulus. Hysterical aphonia from laryngeal palsy, and a hoarse, croaking, laryngeal cough, are very common. Intensely rapid breathing, from fifty to one hundred and fifty per minute, without alteration of the pulse-rate, with or without dyspnœa, occasionally occurs.

Secretion is often affected; profuse sweating, general or local, may occur, and may be attended with sufficient hemorrhage to color the perspiration deep rose-red (*hæmatidrosis*, *bloody sweat*). One of the most characteristic symptoms of the hysterical paroxysm is its passing off with an enormous discharge of limpid, almost colorless, urine of low specific gravity. More serious is the partial or even complete suppression of urine (*anuria*), which may for a long time so prevent the secretion of urea that the sweat and other secretions become loaded with it.

Disturbances of digestion are almost universal; excessive flatulence and constipation, enormous tympanitic distention of the bowels, œsophageal spasm interfering with swallowing, vomiting, which may be excessive and continue for days and weeks, are ordinary phenomena. In the severest cases of vomiting, fæcal matter, or even rectal injections, may be discharged from the mouth. The "fasting girls" of popular literature are hysterics, who are able to live upon the smallest quantity of food and shrewd enough to deceive watchers.

DIAGNOSIS.—The recognition of the pronounced hysterical paroxysms is so easy as to require no discussion, but in the more larvated forms of hysteria the diagnosis may be made with great difficulty. In all cases of doubt great stress is to be laid upon the presence of an hysterical history or of present symptoms of hysteria. Nevertheless, hysteria may coexist with organic nervous disease, as with organic disease of any character,—a combination which sometimes leads to the gravest of errors. We have seen the diagnosis of nervous hysteria persisted in by good medical practitioners to within a few hours of the death of the patient, when an examination of the urine would have demonstrated the uræmic nature of the attack. In all serious cases it should be an invariable rule to examine in the most thorough manner for the existence of an organic disease, and not to settle upon the conclusion that the symptoms are all hysterical because the patient has hysteria. On the other hand, hysterical palsies of the most severe type may exist without other symptoms of hysteria and without an hysterical history that can be made out. In recognizing the nature of such a palsy attention must be especially paid to the following points: the hysterical paralysis is apt to be transient and shifting, to go and come suddenly, and not to conform in its minor phenomena with the sequences and coincidences of organic palsy; also to be accompanied by symptoms which do not occur in the organic paralysis which is simulated. Thus, an hysterical hemiplegia may be attended with paralysis of the bladder, or an hysterical hemianæsthesia accompanying the hemiplegia is not properly situated in its anatomical relations with the coexisting motor palsy, or electro-sensibility is lost when general sensibility is preserved, etc. An atypical paralysis in women is in the majority of cases hysterical; in men it is, perhaps, somewhat more frequently syphilitic.

An hysterical paralysis may immediately follow a slight injury. If contractures develop at once, or if after a time, in spite of complete paralysis and relaxation, there is no wasting of the muscles, or if there is any irregularity of position between the disturbances of sensibility and the alterations of mobility, or if there is even a very pronounced local anæsthesia, the paralysis is probably hysterical. In coexisting organic palsy of sensation or motion, sensation almost always improves first; in mimicking hysterical states, motion usually improves before sensation.

In hysteria consciousness may be completely lost, but partial loss is more common and characteristic. In some cases the patient seems to be conscious during the attack, and afterwards has no remembrance of what has occurred; in other cases the patient appears to be unconscious during the attack, but has a complete after-memory of what has happened. Both these conditions of subconsciousness are almost diagnostic of hysteria.

In an hysterical affection of the joint simulating inflammation the true nature of the attack can usually be recognized by attention to the following considerations: first, the muscular rigidity can be overcome by mildly persistent efforts while the patient's mind is diverted, and yields readily during sleep and disappears during anæsthesia or even under a full dose of chloral or opium; second, there is no rise of temperature in the joint, although the parts look red and inflamed; third, in muscles which have apparently undergone atrophy the electrical reaction remains normal; fourth, the loss of function of the part varies greatly from time to time as the patient's attention is diverted from it, and is also exaggerated by fatigue and nervous exhaustion. In the onset of an organic disease of the joint interference with function is prone to precede the development of pain; in the hysterical disorder pain generally appears first.

Hysterical lateral spinal curvature occurs in neurasthenic women, in whom a true lateral curvature is very frequent; unlike true lateral curvature, it is usually an outcome of spasm and disappears during sleep or anæsthesia.

A localized swelling in the abdomen of hysterical women sometimes gives upon palpation the feeling of a hard tumor (*phantom tumor*); ordinarily the presence of percussion clearness renders the diagnosis easy, but even in moderately obese women this sign sometimes fails. During anæsthesia the phantom tumor, being the outcome of muscular spasm, disappears.

The nature of the *hysterical breast* is to be recognized by the excessive superficial tenderness, so that slight irritation produces as much distress as hard pressure, by the diffuseness of the swelling and the constant variation in the size and hardness of the breast, and by the increase of symptoms at the menstrual period, at the approach of stormy weather, or after general fatigue. In many instances the coming and going of the symptoms make the nature of the case unmistakable. In girls, and not rarely in neurotic boys, at the period of sexual unfolding, one breast will suddenly become hot and exceedingly painful and tender, and perhaps there will be escape of a few drops of sero-lacteal fluid, the whole being a neurotic phenomenon and subsiding without injury.

PROGNOSIS.—In our opinion, it is doubtful whether death ever occurs from hysteria: the reported cases have probably been instances of confusional insanity, acute mania, or other disease. The prospect of cure in a case of hysteria is in inverse proportion to the age at which the hysterical temperament has appeared. An acquired hysteria, due to some removable or temporary depressing cause, may rapidly yield to treatment, but a congenital hysterical temperament can only be abated.

TREATMENT.—By proper education from early childhood it is possible largely to modify the nervous temperament and often to prevent the development of hysteria. The indications are—first, to increase the robustness of the whole person, and especially of the nervous system; second, to reduce excessive sensitiveness by accustoming the nervous system to moderate exposure and hardships; third, to develop in the child the habit of obedience (first to those who are above it, and afterwards to its own personality, led by a sense of right and wrong; in other words, to teach the habit of subjection to control from without, in order that the power of self-control from within may later be developed); fourth, to bring about as much of intellectual development as shall give to the child abundance of interest outside of itself and its immediate surroundings and shall form a basis for character; fifth, to inculcate unselfishness and to develop other traits of character such as are recognized as worthy of imitation throughout the world. When, where, and how these things shall be done must depend upon the circumstances of the individual child. Country life is usually preferable to city life; a moderate living, to a home of luxury; home training, to training in boarding-schools or other institutions; plain food, to high living. In all cases it is essential that those who have charge of the child be themselves not nervous or hysterical.

In developed hysteria the basal treatment must ordinarily be that of neurasthenia. The rigidity with which this treatment must be enforced depends upon the needs of the individual case. Living in the open air, with plain and simple but nutritious food, graded exercise, and freedom from care, social dissipation, or other excitements, in some cases will suffice; but in severe cases the removal from home, the putting to bed, the isolation of the so-called "rest-cure," are not only of the greatest physical benefit, but afford opportunity for that domination and moral training by the physician and the nurse which are so essential to the welfare of the hysteric. Under these circumstances the selection of the nurse is a matter of the greatest importance. She must have the tact to control the patient without causing unnecessary irritation, and must in her person be, as far as may be, agreeable to the patient. The intent of the moral management of the hysteric is to develop, first, a willingness to be unselfish, and, secondly, the habit of self-control. In exceptional cases a careful, skilful unrolling of her own character, of its difficulties, its dangers, and its possibilities, has a most happy effect upon the intelligent victim of hysteria. Usually, however, it is necessary first to teach the habit of obedience or submission to control from without, which habit, when acquired, becomes the basis of advance of character to self-control.

The patient must first be made to perceive that complaints do not bring sympathy, but cause disgust and disregard. The hysterical attack must be made as disagreeable as possible; this not simply to prevent the wilful bringing on of the attacks, but to afford a motive which shall aid the will of the patient in preventing an attack. The paroxysm may usually be cut short by the hypodermic injection of apomorphine, by the cold bath, and by other procedures. In hospital practice the severest paroxysms are sometimes set aside by threatening to cut the hair, by opening soda-water siphons in the face, and by other rough measures, which, although they may be justifiable against professional hysterics who are using the paroxysms for definite purposes, in the continuous treatment of the disorder do harm rather than good. Sometimes, however, measures of such character are of the greatest service if used with due caution and judgment. Some time since, a furious epidemic of hysteria occurred in a Philadelphia charitable institution for the care of children, and required the temporary scattering of the children among various hospitals. In two of the cases no treatment interrupted the return of the paroxysms until the children were kept without food for three-quarters of a day and then heavily fed, after which to one of them in the presence of the other was given ether as slowly and as disagreeably as possible, so as to provoke screaming, fighting, and excessive vomiting, the result being the immediate cure of both cases.

It is often of the greatest importance to afford a strong motive which shall reinforce the weak will of the hysterical person. As is well known, emotional excitement will temporarily, or perhaps permanently, put an end to long-continued hysterical palsies; but the creation of an emotional excitement is entirely different from the installation of a powerful permanent motive.

Even of more influence than a strong motive are faith on the part of the patient and powerful dominant ideas. It is probable that in this way act hypnotism, mock surgical operations, bread-pills given with absolute carefulness as to detail of administration, faith-cures, pilgrimages, metallic tractors, magnetism, and the numerous procedures which, having little or no direct remedial powers in themselves, have yet cured numerous cases of hysteria. Hypnotism is sometimes of the greatest value, but it is necessary, in employing it, to exercise great care, lest the attention which it brings to the patient, and even the state itself, may do injury. In accidental or acquired hysteria hypnotism may be used more freely than in the original neuropathic disorder. It is not, according to our belief and experience, any suggestions made in the hypnotic state that do good; we have cured hysterical contractures, tremors, paralysis, etc., by hypnotism without any suggestions, the result having been, no doubt, due to a mental impression.

In bad cases of hysteria it is very important that the physician do not intensify the symptoms by too much attention. The hysterical woman craves sympathy inordinately, and occasionally has towards the doctor distinctly sexual feelings. In hysterical retention of urine the nurse, not the doctor, should draw off the urine. In hysterical vomiting no attention should be paid to the symptom unless it becomes so severe as to threaten the general nutrition, when the various anti-emetics, especially cocaine, and blisters upon the epigastrium, may be tried. Artificial feeding should soon be resorted to; semi-liquid, half-digested food should be given by means of the nasal œsophageal tube, and, if it be not retained, by the rectum.

In hysterical special-sense hyperæsthesia it is essential that the patient should not be shut up in a dark room or an extremely quiet apartment, but that she should be forced to endure at least a moderate portion of the normal stimulus which is affirmed to cause pain. Blisters behind the eyes, closure of one or both eyes with sticking-plaster, etc., may sometimes be resorted to with good effect, but are liable if used injudiciously to do harm by riveting the attention of the patient upon the symptom.

Tonics and antispasmodics are sometimes useful in the treatment of hysteria. Asafetida, valerian, musk, and even camphor, may be employed freely, as there is scarcely any danger of doing injury with them. The bromides have more power than any of these remedies, but are distinctly more capable of harm ; whilst chloral, morphine, and other narcotics, though sometimes it may be necessary to use them, are dangerous. There is always overhanging the use of these narcotics, and especially the use of alcohol, the gravest danger of the narcotic habit. It must not be forgotten that the pains of the hysteric which are complained of as unutterable agony often disappear in a moment under pleasurable excitement. Cannabis indica is especially useful when the headache takes on the character of migraine.

In hysterical pseudo-angina it is probable that the pain is due to some disorder of the cardiac apparatus, and is not itself, therefore, directly hysterical. Certain it is that the pain may often be relieved by nitroglycerin or amyl nitrite, and the paroxysm set aside by the persistent use of cardiac tonics, such as digitalis and caffeine.

In spinal irritation and in hyperæsthetic ovaries, mild counter-irritation is sometimes of service, though a better result is often obtained by the application of a belladonna plaster. Hypodermic injections of water are frequently most efficacious in relieving severe pains or overcoming insomnia, provided the hysteric believes that the water is a strong solution of morphine.

ASTASIA ABASIA.

DEFINITION.—A condition in which the patient when lying down can move both legs and arms without disturbance of power of coördination, but cannot stand up or walk.

SYMPTOMATOLOGY.—This condition, which appears to have first been described by Blocq in 1888, may develop suddenly or may come on gradually. It may exist in its complete form or in any degree of partialness. Relapses are said to be common after coitus. Three forms of it can be made out: the distinctly hysterical (*hysterical ataxia*, Weir Mitchell), in which at first the patient is often unconscious of the existence of the condition ; the hypochondriacal ; and the psychical, in which it is dependent upon a dominant idea. In the hypochondriacal form the attempts to walk or stand are usually associated with great anxiety and mental distress, violent palpitation, giddiness, various paræsthesiæ, and disturbances of vision. The most plausible explanation of astasia abasia is that it is the result of loss of power of the cortical centres which coördinate the actions of standing or walking, although the existence of such centres has never been demonstrated.

TREATMENT.—The underlying neurasthenic or hysterical condition must be rectified. Friedländer advises also careful systematic gymnastic training: first, passive movements lying down; second, resistant movements lying down; third, resistant movements in sitting posture; fourth, resistant movements in standing posture; fifth, standing without support; sixth, practice in starting; seventh, practice in walking.

SINGULTUS.

The peculiar convulsive motion of the diaphragm and neighboring parts, known as *Hiccough* or *Hiccup*, may be of reflex origin, due to disease of the lungs, peritoneum, stomach, liver and gall-bladder, intestines, uterus, or prostate, or to mediastinal tumors. It also may be the outcome of disease at the base of the brain, or may be purely functional. In alcoholism, rachitis, or typhoid and other low fevers, etc., it may be the cause of death.

TREATMENT.—Very cold or very hot or irritant drinks sometimes are of service. Cocaine, musk, the bromides, camphor, chloroform, given by the mouth in full dose, are often efficient. Chloral, amyl nitrite, hypodermic injections of morphine with atropine, and even inhalations of ether, may be used on occasion. In very persistent cases threatening life, the stomach may, if circumstances favor, be washed out and then allowed to rest absolutely from food, drink, or medicine for one, two, or even three days. The patient during this time should receive digested nutritive enemas and an enema of feebly alkalized water (sodium carbonate), as large as can be retained, alternately every four hours, sufficient chloral and opium being put in these enemata to maintain a semi-narcotism, or at least to control the hiccough. Stimulants may, if required, also be given by the rectum.

VERTIGO.

DEFINITION.—A sensation of motion or swimming in the head, or an appearance of motion in surrounding objects which really are at rest.*

As vertigo is really a symptom, it may arise from very many causes. The peculiarities of a vertigo have no known relations with its causes. For the purposes of diagnosis and subsequent treatment it is essential to recognize the cause of the symptoms. The most important etiological varieties of vertigo are enumerated as follows:

---

* Strictly speaking, vertigo is the whirling of objects around the person; giddiness, the sensation of swimming in the head or motion in the head. But, as the two conditions vary indefinitely in their manifestations and grade in every possible way into one another, the term vertigo has come to be employed as including all disorders of cerebral sensation affecting coördination.

Organic vertigo may be produced by any form of brain-disease, as well as by locomotor ataxia, probably from involvement of the portion of the base of the brain.

Epileptic vertigo. See EPILEPSY.

Cardiac vertigo is usually the result of cardiac fatty degeneration or other form of failing heart; it is really syncopal. *Mal de montagne* (the headache, vertigo, dyspnœa, nausea, and vomiting produced in some persons by high mountain ascent), and the swimming of the head seen in individuals with arterio-sclerosis, are probably also due to lack of blood-supply to the brain.

Hysterical vertigo usually, but not always, takes some bizarre form.

Peripheral vertigoes are produced by irritation in parts of the body distant from the brain. The most important of them is the so-called *gastric vertigo*, which in its acute form is often associated with intense headache and partial blindness or double vision, and is usually relieved by vomiting. It is often directly traceable to the use of strawberries, lobsters, shell-fish, or other articles of diet out of harmony with the digestion of the individual. Chronic gastric vertigo, due to persistent dyspepsia, in which the vertigo recurs frequently from two to four hours before eating, is a much rarer affection even in dyspeptics than is the gastric vertigo directly due to irritation of the stomach. How far chronic dyspeptic vertigo is the outcome of gastric irritation, and how far it is due to the presence in the blood of poisons from imperfect digestion, is uncertain. A peculiar giddiness, with a sense of weight or intense pain in the eyes, may be the only manifest symptom of tape-worm. Polypi and other gross laryngeal lesions have in some cases produced distinct vertigo. Irritation of the nasal mucous membrane, and of the gums by retained teeth, may also be enumerated among the causes of peripheral vertigo.

Special-sense vertigo is in one of its forms the condition which is produced by whirling movements, by swimming, by the rocking motion of the ocean, and is probably due to disturbance of the circulation, especially in those portions of the special-sense apparatus which are connected with equilibration or are in some way irregular. Distorted sense perceptions have, however, in themselves the power of producing giddiness. Thus, nystagmus, paralysis of the external rectus, and more rarely of other eye-muscles, may produce a vertigo which is probably the result of the confusion caused in the nerve-centres by the non-agreement of the perceptive organs in their registration of objects. Usually, closure of one eye puts an end to ocular vertigo. In some cases, however, it fails to do so, probably because there is still a confusion between the reports to the brain of vision and of the senses of touch and hearing. An acute paralytic squint is almost always accompanied by double vision and giddiness. The concomitant squint—*i.e.*, the squint due to diseases of the eye itself—is rarely accompanied by either of these symptoms, probably because it is usually very slowly developed and the brain-centres form the habit of disregarding the visual images in one of the two eyes. Disturbances even of the external ear or of the Eustachian tube may produce vertigo which is probably reflex. Diseases of the aural labyrinth are very prone to cause pronounced vertigo, and sudden congestion or apoplexy of the semicircular canals may give rise to a sudden, violent vertigo, accompanied by excessive pallor, sweating, and symptoms of imminent syncope, or even death. Such cases were first described by Ménière in 1861, and constitute *Ménière's disease,* which must be distinguished from *Voltolini's disease,* which is an acute purulent labyrinthic otitis, accompanied by violent pain, and followed by complete unconsciousness, high fever, and delirium. Chronic labyrinthine disease may cause a persistent aural vertigo, which, however, should not be called Ménière's disease.

Toxæmic vertigo may be produced by alcohol or various poisons or by uræmia, or may be the result of lithæmia; indeed, a *lithæmic vertigo* is the most common form of vertigo, and often causes the greatest alarm. Its true nature is to be recognized by detecting the lithæmia.

Essential vertigo represents a class of infrequent cases in which the vertigo may be very severe and yet no cause be discoverable. It is probable that in these cases there are lesions of still unrecognized brain-centres of equilibration.

TREATMENT.—There is no known specific treatment of vertigo. Relief is to be obtained by learning the cause of the vertigo. When this cannot be reached nothing can be accomplished.

EPILEPSY. IDIOPATHIC EPILEPSY.

DEFINITION.—A disease of unknown pathology, in which at irregular intervals and without obvious existing causes nerve disturbances occur, in most cases accompanied by loss of consciousness and very frequently by convulsive movements.

ETIOLOGY.—Epilepsy is probably in about twenty-five per cent. of the cases due to direct inheritance. In a large number of cases it is the exaggeration of the neuropathic root-stock. Alcoholism, consanguineous marriages, scrofula, rachitis, extreme poverty or dissipation, anything which exhausts the vitality of the parent and tends to the production of nerve-degeneration in the child, has a great influence in the production of the congenital epileptic diathesis. This diathesis is probably also the outcome of transitory unnoted conditions in either parent at the time of conception, or in the mother during pregnancy. Epileptiform attacks produced by chronic poisonings (especially chronic alcoholism), by peripheral irritation, by violent emotional disturbances, if repeated, may cause a continuing epilepsy by forming in the nervous system the habit of discharging paroxysmally nerve-force at irregular intervals. Epilepsy is somewhat more frequent in the male than in the female, and may develop in very early infancy. In a large proportion of cases it appears about the time of puberty. Probably about one-fourth of the cases have the first convulsion under thirteen years of age, one-half under nineteen years, and the remaining one-fourth under thirty years. The number of cases occurring beyond the age of thirty years is so small as not to affect statistics.

MORBID ANATOMY.—Although after death the brain of the epileptic is often found diseased, and although various assertions have been made as to the nature of the lesion of epilepsy, no clear light has been thrown upon the subject, unless it be by the researches of Chaslin, who asserts that the basal lesion of epilepsy is a non-inflammatory degeneration, in which the neuroglia of the brain is transformed into an abnormal tissue composed of bundles of fibrillæ much longer and much more distinct than those in normal brains. The nerve-cells are reduced in size and number, and their processes shrunken or altogether removed; the capillaries are for the most part completely intact, without that cellular infiltration of their walls, and especially of their sheaths, which is so pronounced in inflammatory sclerosis. Some of the capillaries, however, are dilated, and occasionally there is one with its walls thickened. There is no reason for believing that there is any special connection between the disorder and disease of the cornu Ammonis, although Sommer, Hoffmann, and Fischer have reported cases in which there was demonstrable disease of that convolution. It is much more probable that the lesion, whatever it may be, especially affects the psycho-motor zone.

Of the numerous theories of the disease which have been brought forward, only two are worthy of notice here. These are—first, the vaso-motor theory, that the convulsion is due to the sudden overaction of the vaso-motor centre in the medulla, contracting the brain-vessels and causing a convulsion by anæmia; second, the discharge theory, in accordance with which the cortical cells become at irregular intervals so surcharged with nerve-force that an overflow occurs and produces a general disturbance. Of these theories the latter is the more plausible. It should be noted, however, that it does not elucidate the ultimate nature of epilepsy, but simply explains the mechanism of the paroxysm.

SYMPTOMATOLOGY.—The typical epileptiform convulsion begins with a peculiar sensation, which is known as the aura, and is succeeded at once by a wild, harsh scream, the epileptic cry, and also by complete unconsciousness, accompanied by a general tonic spasm. This period of tonic spasm is accompanied by pronounced pallor of the face, and lasts from a few seconds to two minutes. In it the head and eyes are usually turned violently to one side, and the facial muscles contracted,—in most cases most markedly on the side towards which the head is turned. The jaws are fixed, the arms flexed at the elbows and still more strongly at the wrists, whilst the fingers and thumbs are bent into the position assumed in grasping a pen, on account of the conjoint spasm of the interosseous and flexor muscles. The position of the extremities is that of universal tonic spasm, the parts being drawn in the direction of the muscles of superior power, so that there is violent extension of the limbs, with inversion and extension of the feet, clenching of the hands, and some opisthotonos. After a time vibratory tremors commence, and gradually grow more and more severe, until they are lost in furious clonic spasm in which the limbs are alternately relaxed and jerked in movements as wild and bizarre as they are uncontrollable. Purposive movements never occur. During the period of clonic spasm the face becomes red, congested, bloated, livid, with a continual change of expression as the spasm involves now this and now that muscle of the face. Owing to the violent working of the muscles of mastication, the saliva is forced from the mouth in the form of bloody froth. Even in the tonic spasm the tongue may be bitten, but during the clonic spasm it is continually thrust in and out of the mouth and is almost sure to be badly cut. The pupils at the beginning of the fit may be contracted, but absolutely immovable dilatation occurs very early and is the characteristic condition, interrupted sometimes by extraordinary oscillations. The return of the pupils to the normal state may be one of the earliest evidences that the paroxysm has passed. During the height of the attack all reflexes are abolished. The sphincters may be relaxed.

According to Magnan, in the early tonic stage of an epileptic attack the pulse may be very slow, but during the height of a paroxysm it certainly is increased in frequency and in force. The respirations are stertorous, slow, and irregular. In severe fits the pauses between them are sometimes so long that the patient appears to have stopped breathing. If death occurs in the fit, it is from such arrest of respiration. The bodily temperature usually rises,—rarely, however, above 102° F.

The stage of clonic convulsion seldom lasts over three minutes, and is followed by coma, and then by deep sleep, which may continue three or four hours. The patient, after awakening, suffers from headache, malaise, and general muscular soreness. Directly after the paroxysm the knee-jerk is very often wanting, but the reflex activity is regained in from one to thirty minutes, and may become excessive.

The description just given is that of a typical epileptic major convulsion; but any of the phenomena may be wanting. Before discussing further these anomalous epilepsies, it seems advisable to say a few words in regard to some of the individual symptoms.

The epileptic cry is frequently absent, and if it occur more than once, or more than twice at the most, the suspicion of hysteria should be aroused. The aura, which is often absent, takes a great number of forms. Probably the most common is the gastric aura, a sense of distress, or weakness, or trembling, or of some paræsthesia, starting in the stomach and ascending. The initial point of the aura may be a finger, the hand, the foot, the face, the tongue, the larynx, the pharynx, or any other part of the body. In either case, when the ascending wave reaches the neck, unconsciousness usually develops at once. The aura may originate in the brain or in one of the special senses. The psychical aura may consist of an emotion or of an idea. Usually the emotion is one of terror, and the idea something disagreeable.

Of the special sense auras the ocular is the most frequent, the gustatory the rarest. The ocular aura may consist of colors, of megalopsia, of micropsia, of double vision, of amblyopia deepening into complete blindness. In the auditory aura various sounds and even words may be heard. Any of these auras may be accompanied by a distinct hallucination, which usually takes the same form in successive fits. The aura commonly is instantaneous, but in rare cases it travels so slowly that one or even more minutes are required for the development of the fit.

Anomalous Epilepsies.—The most important anomalous epilepsy is the so-called petit mal, which in its ordinary form consists of a momentary loss of consciousness, accompanied (not always) by excessive pallor of the face, usually followed by immediate resumption of intellectual action, sometimes by consciousness of thought. Between this petit mal and the gros mal, or major attack, every grade of paroxysm occurs. The petit mal may be accompanied by an aura, by the epileptic cry, by general quiet muscular relaxation, and varies in its length. The most characteristic and essential feature of the epileptic paroxysm is the loss of consciousness, and yet without doubt during an epileptic attack consciousness may be preserved. A slow aura may sometimes be arrested and the paroxysms be aborted by a tight compression of the limb above the initial point ; and we have seen cases in which the aura habitually suffered spontaneous arrest at a certain point with abortion of the other symptoms.

In the early development of an epilepsy in a child the whole paroxysm may consist of a sudden painful sensation in the stomach, with pallor, but without loss of consciousness, these paroxysms continuing in spite of treatment, and slowly changing, after years it may be, into a fully-formed epilepsy.

Hughlings Jackson has reported a case in which the attacks of petit mal consisted of a brief mental confusion with aphasia ; C. H. Vere one in which the chief phenomenon was a furious salivation.

Nocturnal epilepsy is that form in which the attack occurs at night, either with or without the patient being awakened.

Epilepsia procursiva, or *procursive epilepsy*, is that form in which, with or without a primary epileptic cry, the patient starts to run forward or in a circle and after a time wakes up or falls in a convulsion or coma. This procursive epilepsy is rarely preceded by an aura : it is sometimes due to organic diseases of the brain, but may represent idiopathic epilepsy, and may develop into the ordinary form, or continue for years unchanged. It is said to be closely associated with moral degradation.

Epileptic automatism is that condition in which simple or complicated acts apparently involving consciousness are performed by the subject, who has at such time no proper control of himself or knowledge of his surroundings, and also has no after-memory of the occurrence. Epileptic automatism may represent the whole paroxysm, or may precede a convulsion, but in the majority of cases it follows a convulsion, which under such circumstances is rarely severe. In its mildest form the automatism consists in doing something out of the common, such as removing the clothing, secreting small objects, etc. In many cases the series of acts are so complicated and apparently rational that it is almost impossible to persuade by-standers that the subject is not conscious. Usually there is no emotional excitement in epileptic automatism ; sometimes, however, the contrary is the case, when epileptic automatism passes into *epileptic mania.* This mania may take the form of acute mania or of an agitated melancholia. In either case the incoherence is usually less than in the corresponding non-epileptic affection. The attack is in most cases sudden, and often has for its first phenomenon a period of violent disconnected speech, which is followed by the mania, or by an ambitious or mystic or erotic delirium, in which sentence after sentence flows forth with extraordinary volubility. Sometimes there is a delirium of persecution. Hallucinations are almost universal, and affect all the senses ; they and the delusions to which they give rise conform to the type of the emotional disturbance. The delirium may last from a few minutes to several days, and is characterized by a tendency to acts of extreme violence, destructive, suicidal, homicidal. Even when the mania is apparently of mild type there may be a sudden outbreak of the greatest fury, in which the patient runs amuck at all objects within reach, and, if possible, commits homicide.

The course of epilepsy is essentially chronic, with in most cases a final disturbance of intellection which frequently ends in great mental degradation and even complete dementia. Sometimes a permanent insanity develops in the epileptic, usually in the form of a melancholic paranoia, with delusions of persecution and suicidal impulses. In these cases it is probable that both the epilepsy and the insanity are the offspring of an original neuropathic vice of constitution. The characteristic mental state of chronic epilepsy is a progressively lowered mental power, with a peculiar irritability and brutal selfishness, accompanied by outbreaks of furious anger on the slightest provocation. Even while the mental powers are still active, epileptics are frequently peculiarly irritable and revengeful ; after a paroxysm these tendencies are increased. The *epileptic status* is a condition in which the patient remains for many hours unconscious, with constantly recurring paroxysms. The likeness of such an attack to apoplexy is increased by the rise of temperature, which may reach 110° F.

The epilepsy which has just been described is the ordinary disorder occurring without apparent cause ; but epileptic attacks may be produced by various causes and simulate the so-called idiopathic or true epilepsy. *Reflex epilepsies* are those in which the convulsions are due to peripheral irritation. *Toxæmic epilepsies* are those in which the convulsions are due to poisons, notably to alcohol, to lead, to veratrine, etc.

In *cardiac epilepsy* it is doubtful whether the nervous attacks should be considered primary or secondary. Two varieties of the affection exist. In one form the pulse becomes very slow, falling, perchance, to ten or even less per minute, with generally subnormal temperature, a face at first pale and then livid, and, it may be, stertorous breathing. The paroxysm may be ushered in by a distinct aura-like sensation; the convulsive movements are usually not severe. In the second form of cardiac epilepsy the heart's action is tremendously excited and powerful, the face deep purple, spotted or blotched with ecchymotic exudations, the conjunctiva greatly swollen, deep red in color, often ecchymosed or even freely bleeding. Epistaxis is apt to be severe, and may seemingly bring relief. The convulsion is usually violent. There is strong probability that both forms of cardiac epilepsy start from the heart or its innervation.

DIAGNOSIS.—The recognition of an epileptiform convulsion is so easy as to require no further discussion. The decision that the convulsion is a manifestation of idiopathic epilepsy requires that it should be shown that it is not due to organic epilepsy, or to reflex epilepsy, or to toxæmic epilepsy. The final decision that the case is not one of these disorders must in the end rest chiefly upon the failure to find disease of the nervous system, a peripheral irritation, or a possible toxic cause.

If the convulsion begin habitually in one limb, one side of the face, or other limited muscular territory, and especially if it be confined to such part, the gravest suspicion should be aroused that the case is one of Jacksonian epilepsy, due to organic focal brain-disease. An epilepsy in which no change can be demonstrated in the nerve-centres may, however, take on the Jacksonian type, so that it is usually wise to wait for distinct symptoms of organic brain-disease which shall be permanent in character (a temporary partial aphasia or monoplegia may follow a paroxysm of idiopathic epilepsy) before reaching a positive conclusion.

Reflex epilepsy usually conforms in its type to the idiopathic disease; its nature is to be made out only by discovering and noticing the effect of the removal of the point of irritation. The practitioner should therefore thoroughly examine every epileptic for points of irritation. Wounds in the head and other portions of the body, astigmatism and other imperfections of the eyes, diseases or malformations of the nasal cavity, carious teeth, and especially retained milk teeth, aural diseases, adherent prepuce, intestinal worms, have in numerous cases provoked reflex epileptiform convulsions.

Of the toxæmic epilepsies the alcoholic may simulate not only the grand mal but also the petit mal. In our experience reflex epileptic convulsions rarely, if ever, occur in groups, but the alcoholic epilepsy simulates the idiopathic disease in the tendency to the occurrence of groups of paroxysms and of an epileptic status. Further, in some cases the attack of alcoholic epilepsy is followed by a mental derangement simulating epileptic automatism, but in most cases differing from it in that the alcoholic subject during the condition obeys immediately and mechanically all influences coming from without. The convulsions of uræmia and of saturnine encephalopathy may very closely resemble those of idiopathic epilepsy.

The greatest aid in the diagnosis of a true epilepsy may be obtained from the consideration of the age at which it appears. If the first convulsion occur after the age of thirty years has been reached, the diagnosis of idiopathic epilepsy must be made with great reluctance, and it may be considered as a fixed rule with the rarest of exceptions that an epilepsy (not hysterical) which develops after the thirty-fifth year of age is not idiopathic, but is due to organic brain-disease, to alcoholic or other toxæmia, or to reflex irritation.*

The greatest difficulty is sometimes experienced in distinguishing between hysteria and idiopathic epilepsy. It should be remembered that hysterical phenomena frequently follow a purely epileptic convulsion, and we have seen long series of hysterical convulsions which have resembled idiopathic epilepsy closely enough to warrant the diagnosis of the idiopathic disorder. Usually the nature of the case can be made out by noticing the following points. In hysteria the paroxysm is attended with great emotional disorder, purposive movements, and especially tetanic rigidity; the muscular contractions also are irregular and produce lasting bizarre movements. The epileptic paroxysm has no emotional disturbance until after the fit; the muscular contractions, except in the Jacksonian type, involve the whole body, and there is no apparent purpose in them.

Aid to diagnosis can often be obtained by a study of the temperature. In uræmic convulsions the temperature is often subnormal; it may, however, rise. In the single isolated epileptic attack the temperature usually rises distinctly, and in the epileptic status is often very high. The single hystero-epileptic attack is accompanied by only a slight rise of temperature, and when there is a series of convulsions the temperature falls immediately after each convulsion, and does not after successive attacks reach distinctly higher than with the first convulsion.

PROGNOSIS.—In epilepsy the prognosis so far as the individual fit is concerned is highly favorable, death occurring in the fit with extreme rarity. So far as the disease itself is concerned, the prognosis is highly unfavorable, it being very doubtful whether complete recovery ever occurs from true idiopathic epilepsy. We have seen the fits recur, under continuous treatment, after having been absent for six years. The disease does not necessarily or even usually greatly shorten life, the epileptic very often living to advanced middle life or even to old age. The question as to the intellectual future of an epileptic is always of most serious import. The resistance of the brains of different individuals to retrograde epileptic changes varies very greatly, but three rules may assist the practitioner in his prognosis: first, the earlier the age at which the epilepsy commences, the greater the probabilities of serious mental deterioration; second, very rarely, if ever, is there any distinct recovery of power, so that symptoms once established are usually permanent; third, the more frequent and severe the fits, the greater the chances of intellectual ruin.

---

* In an experience including over a thousand cases of epilepsy I have not met with more than one or two, and those doubtful, exceptions to the rule. (H. C. W.)

Epilepsy does not, however, necessarily end in mental degradation, even when it has come on early; when it has existed some years without producing any serious intellectual results the chances are always in favor of the escape of the patient from deterioration. Many epileptic persons pursue with activity and success a business or even a professional life. In estimating the prospects of a case it is essential not to mistake emotional excitement, pseudo-convulsions, or even pseudo-mania, for symptoms really of epileptic origin and evidences of permanent intellectual change. Such symptoms may be hysterical, and under treatment may disappear; whereas the true epileptic mental failure is without doubt the result of change in the brain-structure, and is hopeless of relief.

In estimating in any individual case the chances of amelioration, the question whether the patient has or has not been skilfully treated enters very largely into the judgment. Again, a petit mal usually yields less to remedies than does a severe epilepsy. The more frequent and severe the attacks are, provided the patient has not been already under proper treatment, the more brilliant are the results to be hoped for.

TREATMENT.—The treatment of idiopathic epilepsy naturally divides itself into that of the individual paroxysm and that of the series. As the epileptic fit very rarely does immediate harm, all that is usually necessary is to place the patient so that he cannot injure himself; to loosen at once all tight bands, especially about the neck, and, in order to prevent cutting of the tongue, to push between the teeth a piece of flat cork or rubber with a string tied to it, so that if by any means it should get into the throat it may readily be withdrawn. The inhalation of ether will usually put an end to the convulsion, and in our observation has never done harm. While, therefore, it is not necessary, it may be useful to quiet the alarm and satisfy the craving for action of nervous mothers and other care-takers. Resistance aggravates the convulsive movements, and should not be employed unless the patient be in position of peril. After the attack the subject should be allowed to sleep quietly. In those cases in which there is sufficient time for action between the beginning of the aura and the unconsciousness, the paroxysms can very commonly be arrested either by mechanical or by medicinal means. If the aura be properly situated, the patient should be taught to grasp the limb firmly with the hand, or to encircle it with a tight band; usually the aura will be unable to pass the constriction. When the attack commences as a local spasm, forcible breaking of this spasm by stretching the part occasionally suffices. Amyl nitrite will in most if not in all cases, if promptly inhaled, arrest the fit; ten minims of it in pearls, or in a little vial, should be carried in the pocket of the patient, and crushed or emptied upon the handkerchief and deeply inhaled at the first inception of the aura. This mode of treatment seems to be entirely free from danger.

The general management of the epileptic should be both hygienic and medicinal. Moderate exercise, intellectual and physical, with abundant sleep, should be strictly enjoined. In the young it is extremely important that the education go on, and that obedience, discipline, and self-control be taught. Over-study, of course, will do harm, but moderate study may be of service. So far as possible, the epileptic should conform in his daily life to the habits and customs of his class, and every effort should be made to prevent that withdrawal of the patient from society and business which the fear of publicity or of physical injury during the fit, and a false sense of shame, tend to produce. It is much better to take the risk of the convulsion occurring in an inconvenient position than unnecessarily to seclude the patient.

The diet of the epileptic should be chiefly, but not altogether, vegetable. Abstinence from meat, which has been advocated by some authorities, is certainly of no value, and flesh may be taken twice a day in moderate quantities without any evil results. Tobacco, tea, and coffee are forbidden by authorities, but when used in moderate quantities probably do no harm to the adult.

The number of the older anti-epileptic remedies is in exact proportion to their worthlessness; valerian, artemisia, belladonna, zinc oxide, copper sulphate, silver nitrate, and even borax,—which has been recommended comparatively recently,—are of no value.*

The only remedies which we have seen do positive good in epilepsy are antipyrin, antifebrin, sulphonal, and the bromides. The value of sulphonal is very slight; it should be used only as a temporary expedient when for any reason it is necessary for a time to withdraw the more potent remedies. Antifebrin has distinct power in some cases; its action seems to be closely allied to that of antipyrin, but in our experience it has been less efficient. In rare cases antipyrin, given by itself in doses of from ten to fifteen grains a day, acts most happily, and there are a few individuals in whom it is preferable to the bromides. There seems to be no way of clinically determining in any individual case except by trial whether antipyrin given alone will suit or not. The chief value of the drug is in its being a coadjutor of the bromides.

---

* The changes in the recurrence of the paroxysm and in the paroxysm itself in an epilepsy are so irregular and so apparently causeless, and the effects of mental impression so powerful in temporarily affecting the return of the fits, that the greatest care is necessary in deciding as to the value of individual remedies. Having a large ward of about fifty epileptics under my care, I kept all of them for a length of time without specific remedies, and then administered the bromides and afterwards borax. Under the bromides the weekly aggregate number of fits was reduced to about one-third of what it had been. The borax was given in as large dose as could be borne without gastro-intestinal disturbance, but caused no perceptible decrease in the number of fits per week. (H. C. W.)

Of all the remedies against epilepsy the bromides are the most serviceable. Although Albertoni has shown by direct experiment that they diminish decidedly the irritability of the cerebral cortex in the motor zone, they are palliative rather than curative, and act only while present in the cortex. They do not remove the tendency to epileptic attacks, but antagonize the action of such tendency, and must therefore be in most cases administered continuously for many years after the occurrence of the last fit. The potassium, sodium, lithium, and ammonium bromides have been chiefly used. Of these potassium bromide has been most employed, but there is no reason for believing that it is superior to sodium bromide. Lithium bromide has yielded in our hands results not distinguishable from those produced by potassium bromide: having no advantage over the latter salt, and being more expensive, it is less available. On the other hand, strontium and ammonium bromides have very distinct superiority over the older bromides in being less apt to produce either physical or nutritive depression. It is possible to produce with either of them bromism, but the acne rash, the fetid breath, and the general nerve-depression are certainly less in proportion to the anti-epileptic effect than with potassium bromide or even a mixture of the older bromides. Both ammonium and strontium are stimulants to the circulation, whilst potassium is a powerful depressant. The strontium salt differs from the ammonium salt in being directly less active as a bromide, but in having a most happy effect upon the alimentary canal. It may be that it is an intestinal antiseptic, but certainly in some way it improves digestion instead of harming it. The best results have in our experience been obtained from a mixture of the two salts in the proportion of two of the ammonium and one of the strontium.

It is a matter of great importance to obtain, if possible, some combination which will enable us to reduce the amount of the bromides necessary to control the epileptic fits. The addition of one one-hundredth of a grain of atropine per day to the bromides sometimes is of service; more rarely the inconvenience of dry mouth, etc., which it produces exceeds any advantage derived. On the other hand, antipyrin is an exceedingly valuable addition to the bromides. Again, arsenic certainly has some power in lessening the severity of the skin eruption produced by full doses of the bromides. The combination of the mixture of ammonium bromide, strontium bromide, antipyrin, and Fowler's solution is invaluable in epilepsy. Within the last two years we have in a number of cases added to this combination the fluid extract of *Solanum carolinense.* This drug given by itself has with us failed entirely in epilepsy, but in doses of a teaspoonful it has in a number of cases enabled the patient to get along with less of the bromides. Antipyrin should never be given in sufficient doses to produce cyanosis ; ten grains per day can usually be administered without the induction of any apparent effect for many months, but in from one to two years there is often to be noticed a disorder of the thermogenetic functions of the body, so that the patient is continually cold and has a tendency to extreme coldness of the extremities, with colliquative sweating, requiring the withdrawal of the antipyrin. Sulphonal in a measure will replace the antipyrin, in daily doses of from ten to fifteen grains, but care is necessary to avoid its too protracted administration, for fear of chronic poisoning. As the bromides act by accumulation in the system, it is not necessary to give them more than twice in the twenty-four hours,—a great boon, since the frequent taking of medicine is extremely irksome. In the beginning of the course of treatment ascending doses should be used until the paroxysms are controlled or until the presence of acne, somnolence, or excessive weakness shows that bromism has been induced ; later in the case the effort should be to keep the patient just within the limits of bromic saturation,—*i.e.*, of a distinct physiological manifestation of the drug.

In 1894, Flechsig claimed extraordinary results in epilepsy from the use of opium and the bromides in the following manner. The opium is at first taken in the dose of three-quarters of a grain of the extract three times a day, and regularly increased until fifteen grains a day are ingested. After this has been kept up for six weeks the opium is suddenly withdrawn and the bromide given in very large doses (two drachms) for two months, when it is gradually withdrawn. Usually the cessation of the attacks begins when the bromide is given, the opium treatment having no immediate effect.

In the rare instances of Jacksonian epilepsy occurring without demonstrable organic brain-disease, the question as to the propriety of the removal of the cortical centres which appear to be implicated in such a case cannot as yet be positively answered. *A priori* it is to be expected that removal would be followed by a sclerosis, and that any benefit achieved would be temporary. There is, further, the serious inconvenience of paralysis which may be permanent. The clinical results so far obtained are, on the whole, not encouraging. Nevertheless, in clear cases surgical interference may be tolerated, provided the whole situation has been carefully explained to the person or persons most interested.

PERIODIC PARALYSIS.

Definition.—A paralysis without obvious cause, apparently not of hysterical origin, coming on in repeated attacks of brief duration.

Symptomatology.—A periodic palsy is occasionally produced by malarial poisoning, a paralytic attack representing an ordinary malarial paroxysm. A number of cases have been reported in which, without loss of consciousness or sensation, but with loss of the reflexes and of the electrical reaction of the affected muscles, paraplegia or paralysis of both arms and legs has come on in paroxysms, lasting a few hours, the patient being between the paroxysms apparently normal. In several instances more than one member of the family suffered ; and Shakovitch reports a case in which the father of the affected patient was said to have died from an increase in the frequency of attacks of the same disorder. Of the nature of these cases we have no knowledge. In malarial paralysis quinine should be very freely given (forty to fifty grains in the interval).

LARYNGISMUS STRIDULUS.

Definition.—A violent spasm of the larynx, attended with dyspnœa and a peculiar crowing sound, not dependent upon an infectious or a local inflammation, and recurring at irregular intervals.

Etiology and Pathology.—The most common cause of this disorder is rickets (see Tetany, p. 441) ; but the attacks do occur in adults, in some cases replacing paroxysms of migraine, in others apparently representing epilepsy, in others hysteria.

Symptomatology.—The phenomena of a paroxysm of laryngismus stridulus are a sudden pallor, a violent laryngeal dyspnœa, greatly altered or suppressed voice, loud stridor, and, as the spasm relaxes, a peculiar crowing noise. Severe attacks, especially as seen in adults, may last some minutes, and be attended with great distress and excitement, the subject, with extended arms, anxious, cyanosed face, bent-forward body, wide-opened mouth, and straining muscles, laboring for breath, now clutching at the throat, now tearing open the clothes in an agony. Death may seem imminent, but it is doubtful whether it ever occurs during a paroxysm.

Diagnosis.—The laryngeal crises of locomotor ataxia closely simulate laryngismus stridulus, if indeed they should not be considered a form of it. Their nature is to be recognized by the recognition of the ataxia. Violent acute suffocation may be produced by hysterical (or other) *paralyses of the abductors of the larynx*, but in this affection the voice remains almost unchanged, and, although the inspiration is highly stridulous, the expiration is nearly noiseless.

Prognosis.—The prognosis depends upon the cause of the disorder ; in epileptoid cases the paroxysms may continue for years.

Treatment.—Amyl nitrite, ether, or chloroform may be inhaled during the paroxysm. The treatment between the paroxysms is that which is appropriate for the condition underlying the attacks: phosphorus in the rachitic cases; bromides in the epileptoid cases; etc.

CONVULSIONS.

Convulsions are symptomatic conditions which have been discussed in various portions of this volume, but their practical importance seems to warrant a brief return to the subject.

In any case of convulsions to which the practitioner is called it is necessary to decide, first, whether the convulsion is hysterical, tetanic, or epileptiform. For the discussion of the diagnostic points in hysterical and tetanic convulsions, see pages 407, 424, and 199.

In the epileptiform convulsion it must first be decided whether the convulsion is isolated or one of a series. If it is one of a series, the case is some form of epilepsy. (See page 423.) The isolated convulsion may be the first one of the series, but is in most cases the outcome of animal, vegetable, or mineral poisoning, or of peripheral irritation. In the adult it is most commonly due to a toxæmia, which in the majority of cases is uræmic or alcoholic, but which may represent some other poisoning. Peripheral irritation, especially gastric irritation, does in rare cases cause convulsions in adults.

In young children the convulsion very often marks the commencement of some exanthematous disease or of a severe pneumonia; but perhaps more frequently it is a reflex convulsion, due to an irritation caused by teething, or by indigestible substances in the gastro-intestinal tract. The nature of a convulsion ushering in scarlet or other malignant fever can usually be recognized by the peculiar expression of illness and the general vital depression which attend it, and by the disturbances of the temperature, aided in some cases by knowledge of exposure to contagion. As, however, the diagnosis is frequently impossible in all cases of acute convulsions, the gums of the young child should be carefully examined, and if they be found swollen and inflamed should be lanced. Further, unless the case be a clear one, it should be a uniform practice to administer at once an emetic by the stomach, or to give a hypodermic injection of apomorphine. Especially is this routine proper because gastro-intestinal irritation in a susceptible child may cause so much vital depression and fall of temperature as to simulate the oncoming of a systemic fever. A dose of castor oil should be administered after the action of the emetic. The convulsion itself may be met by the free administration of the bromides, the extremely careful use of chloral, or, in severe cases, the inhalation of ether. The hot bath is useful in all forms of convulsions of children unless there be high bodily temperature. If there be high temperature it may be replaced by the tepid or the cold bath. We have seen children who had ceased to breathe and were apparently dead, as the result of violent gastro-irritation, recover by the use of artificial respiration carried out in the hot bath, the emetics which had failed to act producing free vomiting as soon as the bodily temperature was raised and the accumulated carbonic acid pumped out of the lungs.

LOCAL SPASMS.

Local convulsions, clonic or tonic, may occur within any nerve territory,* but, unless distinctly due to cortical brain lesion or to hysteria, are very rare except in the face, the neck, and the trapezius muscle.

Facial Spasm, Tic, Convulsive Tic, Painless Tic.—Clonic contractions of the facial muscles very often accompany trigeminal neuralgias or neuritis, constituting *Tic douloureux*, or painful tic ; but facial spasm may come on without obvious cause, or may be produced by distal irritations, especially of the female genitals, and may follow a violent fright or other emotional nerve-storm, or even, it is said, traumatisms of the nerve or of the cerebrum. No lesion can usually be discovered after death ; rarely can any change be detected in the cortex.

Facial cramp is usually unilateral, and may be confined to any muscle or affect the whole group supplied by the facial nerve. It usually occurs in paroxysms of tonic contraction, in which the eye is tightly closed, the forehead deeply wrinkled, and the nose and mouth drawn strongly to the side. With these tonic contractions, or replacing them altogether, are clonic movements. The attacks, which are less severe during absolute quiet, are greatly intensified by excitement, by efforts at chewing or speaking, and often by a blast of cool air. *Blepharospasm* is a tonic contraction limited to the muscles that close the eye ; *blepharoclonus* is a similar clonic contraction. In these, and occasionally in other forms of facial spasm, strong pressure upon the place at which some branch, especially the supraorbital, of the trigeminal escapes from the skull, will often suddenly end the attack. When the muscles of the inner ear are affected, tinnitus aurium may be produced.

Torticollis, contraction of the sterno-cleido-mastoid muscle, is in its most ordinary form rheumatic ; the nature of such a case is to be recognized by the violent pain produced by passive efforts at motion of the head or by pressure on the affected muscles. Congenital torticollis is no doubt often due to injuries during birth, but its frequent association with irregular facial development indicates that at times it has a deeper origin, the nature of which is obscure. It may not be noticed for a long time after birth, when, on examination, the sterno-mastoid on the affected side will be found hard, shortened, and distinctly atrophied. In some cases the trapezius muscle is also involved.

Spasmodic torticollis occurs both as tonic and as clonic spasms. When the contraction is tonic the occiput is drawn towards the shoulder of the affected side, the face is rotated towards the opposite shoulder, and the chin is raised ; an involvement of the trapezius increases the drawing of the head towards the affected side and raises the shoulder. In the clonic variety of the disease the contractions recur at very short intervals, and usually involve the trapezius muscle. Not rarely the splenius and even the muscles of the back of the neck are involved. *Rotary spasm* of the head (obliquus capitis muscle), and *nodding spasms* (the deep recti capitis muscle), sometimes complicate or replace the torticollis.

---

* For study of localization in any case, see the discussion of paralysis of peripheral nerves in Chapter VI., Section II.

Unless some point of irritation can be found, or the case is syphilitic or hysterical, the prognosis in these various spasms should be very guarded. Blepharospasm and clonus usually depend upon disease of the conjunctiva or of the eyelids, and disappear when this is cured. In the great majority of cases treatment is of no avail; bromides, conium, aconite, hyoscine, and all the spinal sedatives may be tried seriatim, almost invariably with equally little result. The local applications of electricity and massage rarely do other good than to keep up the morale of the patient. The treatment should be, first, the up-building of the patient's health by the rest-cure or other measures adapted to the individual case; second, the continuous, merciless use of the actual cautery. We have seen permanent cure obtained by such use in cases certainly not hysterical nor rheumatic. The burning should be repeated as often as the effects disappear. The very cautious use of tartar emetic ointment will sometimes serve advantageously in maintaining the effects of the burn. It should always be remembered that torticollis or facial spasm may be the only apparent outcome of a cerebral syphilis, under which circumstances the effect of antispecific medication is immediate. Various mechanical devices have been proposed for the relief of the patient; all of them are of doubtful utility. The spinal accessory and other nerves have been frequently cut, with the effect of substituting paralysis for spasm. Often the spasm has returned, evidently after the reuniting of the nerve; and still more frequently have the contractions, not long after the operation, appeared in the opposite side.

ST. VITUS'S DANCE.

DEFINITION.—A non-febrile disease, not necessarily dependent upon demonstrable organic affection of the nervous system; usually occurring in childhood; characterized by generalized choreic movements and loss of nerve-power.

SYNONYMES.—Chorea; chorea minor; chorea of childhood.

ETIOLOGY.—Neuropathic heredity, luxury, poverty, whatever lessens the robustness of the nervous system of the child, predisposes to chorea. The disease is much rarer among negroes than among whites; is more frequent in girls than in boys; about four-fifths of the cases occur between the fifth and the fifteenth year.

Chorea, like other diseases connected with nervous exhaustion, is in the northern United States much more frequent in the spring, probably on account of the lowered nerve-tone produced by the long winter. So large a proportion of the sufferers from chorea are of the rheumatic diathesis, and so frequently does chorea develop from or into rheumatism or alternate with that disorder, that there must be some relationship between the two affections. What this relationship may be is at present unknown. Various authorities have recently maintained that the chorea of childhood depends upon the presence of a poison in the blood; and the relation of chorea to rheumatism corroborates this view. Pianesi asserts that the disease is of bacillary origin, and that he isolated a bacillus which inoculated into the dog produced chorea. On the other hand, the sudden development of the disease by a powerful emotion does not seem compatible with the idea that it is of necessity due to a toxæmia.

Nevertheless, the view of Thiboulet, somewhat broadened, is very plausible, and in general accordance with the natural history of the disorder. This is that a chorea may be due to various poisons acting upon a nervous system which is predisposed to the disease. Chorea might thus be defined as a peculiar condition of the whole nerve-tract, capable of being produced by various poisons, and also by other disturbing agencies, such as violent emotion or anatomical alterations; the latter perhaps due to wide-spread, minute thromboses. The action of these causes is favored by the existence of a peculiar predisposition of the nervous system to become choreic under their influence.

Chorea is prone to recur,—not because one attack predisposes to another, but because a pre-existing foundation weakness renders the nervous system liable to be easily thrown off its balance time and again.

Morbid Anatomy.—Various lesions have been found in the brain and in the spinal cord after death from St. Vitus's dance, such as minute cerebral embolism, softening, interstitial proliferation, and hyperplasia of the neuroglia. F. C. Turner found in five cases of chorea that the nerve-cells in the Rolandic region were swollen and opaque. The ganglionic cells of the spinal cord have been found shrivelled, with an abnormally granular protoplasm and an obscuration of their nuclei. H. C. Wood has found similar lesions in the brain and cord of choreic animals, but only in the later stages of the chorea. Examination of animals killed at various stages of the process shows that in the beginning no change can be detected, but a little later the cells are incapable of being stained and the nuclei are absent. The processes then become detached, and finally the cells are reduced to irregular, globose, crumpled masses.

It must be remembered, however, that chorea may be developed in a few minutes from fright, and is usually recovered from in a few weeks; hence it is absurd to suppose that it is necessarily based upon serious organic change of the nerve-centres. Moreover, even in cases of fatal chorea, competent observers have failed to find alterations in nerve-centres.

Since choreic movements may originate in either the brain or the spinal cord, and the condition of the knee-jerks in the choreic child (see below) demonstrates that the ganglionic cells of the cord are in an abnormal state, it seems clear to us that the basal lesion of St. Vitus's dance is a change in the nutrition of the ganglionic structures of the whole cerebro-spinal axis. Structural changes sufficient to be detected by the microscope may not result, or pronounced alterations may follow.

As has been pointed out by H. C. Wood, depression of the inhibitory spinal function is an important feature of chorea, the choreic exaggeration of voluntary movements occurring because of the failure of Setschenow's inhibitory nerve-centres to arrest motor discharge from the spinal cells at the instant when it must cease in order that the motion be exactly as desired. It was found in Dr. Wood's experiments upon dogs that drugs which, like belladonna, depress spinal inhibition greatly exaggerate choreic movements, whilst drugs which, like quinine, stimulate spinal inhibition greatly lessen these movements.

It must not be taken for granted that the pathology of true St. Vitus's dance, or chorea of childhood, is that of all forms of chorea. The choreic movements may occur from diseases of any portion of the motor tract, and there can be no doubt that multiple emboli of the cerebral cortex and other gross lesions of the brain may produce a chorea.

SYMPTOMATOLOGY.—The invasion of this disease may be sudden or gradual. The attack may come on in the midst of apparent health, but ordinarily it is preceded by languor, irregular action of the gastro-intestinal tract, and a pronounced nervous irritability. The motor disturbance may be first indicated by a peculiar restlessness of the child, who is not rarely punished for fidgeting. The true choreic movements usually appear first in the fingers, and shortly afterwards in the face, and spread until they involve the whole body. In severe attacks the arms are in almost constant movement, the fingers opening and closing, the wrists flexing and extending, and the elbow-joints in almost incessant activity, so that every imaginable position of the hand and arm is rapidly taken and lost. During the violence of the disease it is impossible for the child to control the movements of the arm sufficiently to dress or feed himself, or to perform any act requiring precision of motion. At this time the legs are similarly affected, so that walking is gradually interfered with, or may be rendered impossible. The steps are irregular, jerking, often with lateral movements, now rapid, now slow, and if progression occur at all it is zigzag and uncertain. The face and head are no less affected : there is a constant, ever-changing distortion of the countenance, giving rise to fleeting expressions of sadness, terror, grief, rage, etc., and to grimaces innumerable. The mouth is opened and shut, the corners jerking up and down : the tongue is protruded, or sometimes moved rapidly in the mouth so as to produce a peculiar clacking sound. Articulation grows indistinct, the child speaks irregularly and badly, perhaps only in monosyllables, and finally the voice may be converted into a succession of irregular, unintelligible sounds. In very bad cases mastication becomes almost impossible, and even the muscles of deglutition are involved, so that the child is unable to swallow at the proper moment, and the food is spluttered and spilled about. The head itself is moved rapidly to and fro, backward and forward, sometimes laterally, sometimes in perpetual rotation. In the most violent cases all the muscles of the body are in a condition of furious action. The rolling, twisting movement of the trunk, and the perpetual beatings and thrashings of the extremities, render it almost impossible for the patient to lie in bed unless fastened down, and the utmost care is necessary to prevent severe bruises and excoriations of the skin. The knee-jerks in chorea are usually diminished or altogether absent, but on reinforcement are apt to be above the norm,—an indication that the usual spinal overflow from the brain-impulses for voluntary movements does not meet with the normal resistance, or, in other words, that there is weakness of the spinal inhibitory function.

The respiratory muscles are the last to be affected, but cases have been reported in which hiccough, crowing inspiration, irregular respiratory rhythm, and other evidences of choreic action of the respiratory muscles were abundantly present. The choreic movements cease at night, or at least during sleep, but in the most severe cases by keeping the patient awake they produce an insomnia which constitutes an additional factor in the rapid wearing out of the strength and the bringing about of a fatal result.

In chorea there is a peculiar nervous irritability often associated with apathy, which is almost characteristic, and which in bad cases is accompanied by a loss of power of fixing the attention upon any one subject for a length of time, as well as by a weakness of memory. Hallucinations are very rare, and usually indicate that a chorea is hysterical. They may, however, occur in typical St. Vitus's dance. In fatal cases the mental disturbances are very pronounced; there may be even an acute dementia; sometimes the patient is seized with maniacal delirium, which is always of exceedingly serious import.

The heart-muscle may participate in the choreic disturbance. Chronic valvular lesions or acute endocarditis may give rise in chorea to mitral murmurs, but such murmurs are frequently heard in cases in which there is no distinct anæmia, no cardiac disease, and no permanency of the murmur after recovery from the chorea. Moreover, these murmurs vary from hour to hour, at times entirely disappearing; and fatal cases have been reported in which no valvular lesion was found at the autopsy, although cardiac murmurs had existed during life. These murmurs must be due to irregular choreic contractions of the chordæ tendineæ preventing the proper closure of the heart-valves.

The course of chorea is always slow. Rarely is recovery complete under five weeks; it may be delayed for months. In some cases the condition is permanent. Usually, however, the child finally gets entirely well. Death is exceedingly rare in the uncomplicated St. Vitus's dance of childhood.

Diagnosis.—The recognition of the existence of chorea is so easy as to need no further discussion. Care is sometimes necessary to avoid mistaking an hysteria for an acute chorea. The most important distinctions are the rigidity and the tendency to rhythmical movements in the hysterical cases. The choreic neurosis is, however, so closely allied to the hysterical neurosis, and in some cases the symptoms of the two disorders are so intermingled, that it may be equally correct to speak of a case as one of choreic hysteria or one of hysterical chorea.

Prognosis.—The obstinacy of a case of chorea is usually in direct proportion to the severity of the symptoms; but, as the mildest cases sometimes prove extremely obstinate, the prognosis should always be guarded as to time, although a final complete recovery is to be expected. According to Guillemand, two and a half per cent. of all the cases prove fatal; death, however, is extremely rare in children.

Treatment.—The hygienic treatment of St. Vitus's dance is the use of rest, fresh air, exercise, careful feeding, and tonics to restore the lowered nerve-tone. Great care must often be taken that the feeble child does not overexercise; indolence rather than activity must usually at first be urged upon the choreic. Many hours a day ought ordinarily to be spent on the bed, while in severe cases a rest-cure treatment may be necessary. The child should be kept in the open air as much as possible, in a hammock, reclining chair, carriage, etc., according to the circumstances of the case. The food should be nutritious, but not stimulating, thoroughly digestible, and given in as large quantities as the alimentary canal will assimilate,—milk and farinaceous articles, with a restricted use of meat and of sugar. Bitter tonics and alcohol in small quantities may be administered to increase the activity of the digestive organs, whilst cod-liver oil and iron, if well borne, may be employed as nutrients. The bitter vegetable purgatives are valuable if given only in such doses as will keep the digestive tract thoroughly cleaned out and stimulated.

Drugs which depress motor activity will check choreic movements, but are only palliative. They accomplish no permanent good except by procuring rest and sleep. The bromides are not very effective, are distinctly depressing to the nutrition of the nervous system, and are to be used only under peculiar circumstances. Chloral will, for the time being, quiet almost any choreic movements; especially is it active when combined with morphine; and in all cases of chorea threatening life a combination of these drugs, in the proportion of ten grains of chloral to one-eighth of a grain of morphine, should be given at night in such amounts as may be necessary to procure quiet sleep. Trional may at times be substituted for the chloral.

There are three drugs which have a specific permanent curative effect. Arsenic is extraordinarily effective: it should always be given by itself, so that its dose can be altered independently of that of other remedies, and must be administered in ascending doses up to the limit of toxic action. To a child five years old may be given three drops of Fowler's solution in milk after meals, the dose being increased every third day one drop until there is distinct puffiness of the face or gastro-intestinal disturbance, when the medicine may be temporarily withdrawn. Cimicifuga sometimes succeeds after the failure of arsenic. A freshly prepared fluid extract, having a strong odor of the drug, should be given in increasing doses until it causes headache or vertigo. Thirty minims may be the commencing dose for a child nine years old.

Led to experiment by his belief that quinine is a stimulant to the inhibitory function of the spinal cord, and that failure of spinal inhibitory power is a large element in the development of the choreic movement, H. C. Wood found that in dogs choreic movements are immediately arrested by moderate doses of quinine, and subsequently he experimented with the drug upon choreic children. The alkaloid is certainly of great value in many cases, but must be given in very large doses. In those cases in which quinine does good there is an extraordinary tolerance of it, so that it is almost impossible to produce cinchonism. Thus, Wood gave as much as one thousand grains in a month to a child of twelve years without cinchonism, but with the cure of a chorea of two years' standing.

REFLEX CHOREA. CHOREA OF PREGNANCY.

Definition.—Local or general chorea due to some peripheral irritation.

Any form of chorea, from the most purely localized to that which closely simulates a St. Vitus's dance, may be produced, in a person having a predisposition, by a peripheral irritation. Among these irritations may be mentioned intestinal parasites, diseased teeth, neuromatous tumors, nasal deformities or diseases, and irritation about the genitalia. In any case, therefore, of persistent choreic movements, it is essential for the practitioner to examine thoroughly for some point of irritation, and, if such be found, to remove it as soon as possible. The very serious chorea which occurs during pregnancy may be looked upon as a form of reflex chorea, due to the irritation of the fœtus acting upon a nervous system predisposed to chorea perhaps by inheritance, perhaps by the nervous exhaustion produced by hydræmia and other disturbances of pregnancy.

As death is not infrequent in the chorea of pregnancy, due to the extremely violent and incessant movements depriving the sufferer of sleep and causing a rapidly progressive exhaustion, no time should be lost in bringing the patient under the influence of chloral and opium, aided by antipyrin and the bromides. It is essential also to maintain the bodily forces by moderate stimulation and high feeding. If the symptoms continue, the general consensus of obstetrical opinion is in favor of the production of abortion before the patient's strength is too much exhausted.

CONVULSIVE CHOREAS.

DEFINITION.—Affections with violent choreic movements which do not simulate complicated purposive acts and are not attended with loss of consciousness.

For the convenience of the student, it is purposed here to discuss certain groups of cases in which the symptoms, although more or less similar, are of diverse origin.

Hysterical Chorea.—Any form of movement may occur in hysteria, but the choreic movements of hysteria are usually rapid and more or less rhythmical, and are frequently seen in limbs already distorted by hysterical contractures. The rhythmic spasm of hysteria may affect any portion of the body, giving rise in the face to all forms of grimaces, with or without the simultaneous thrusting out of the tongue, or, when attacking the muscles of the larynx and of respiration, causing various strange sounds. The so-called *electric chorea*, in which the whole body or a portion of it is the seat of a more or less rapidly repeated clonic peculiarly brusque spasm, resembling that produced by an electric shock, is probably always hysterical.

Choreic Tic.—In this group may be included various cases of local chorea, including the so-called *habit choreas*, in which it is believed that the origin of the chorea has been in a habit which was at first controllable by the patient but afterwards became a fixed nervous manifestation. Some of these local choreas are certainly from the onset uncontrollable. The tic or spasm may involve a single nerve-distribution, or a wide-spread area; may be irregular, having no apparent relation with life; or may continue the form of a purposive act in which, perchance, it had its start. A brow may be lifted at intervals, an eye winked, a jaw dragged forward, a shoulder shrugged, a trick of gesture incessantly repeated, even a cough or a snuffle perpetually indulged in. When the paroxysm is wide-spread and accompanied by a diaphragmatic contraction, which by forcibly expelling the breath produces some bizarre sound, the case may assume the appearance of an automatic chorea, but is in fact essentially different from those cases in which the movements are directed towards an end and are caused by a dominating impulse.

The treatment of cases of choreic tic usually ends in failure, and must be chiefly hygienic. Antispasmodic remedies are of no value.

Organic Choreas.—Cases in which the choreic movements are due to various organic diseases of the nerve-centres.

In this group belong the pre-hemiplegic and post-hemiplegic choreas, also athetosis, and allied affections elsewhere considered. It seems necessary only to state further that there is a form of chorea occurring in very old people (*senile chorea*) in which the symptoms resemble those of the St. Vitus's dance of childhood, and are probably due to obstructive interference by diseased blood-vessels with the blood-supply of the ganglionic cells of the pyramidal tract. The prognosis is always very grave; but under the free use of tonics, high feeding, and alcohol we have seen the choreic movements disappear.

AUTOMATIC CHOREA.

Definition.—An affection in which paroxysms of apparently purposive actions occur independently of the will of the subject, as the result of an impulse which arises spontaneously in the individual, or which occurs in response to some impulse received from without the individual.

The definition just given covers two classes of cases: the *chorea major* or *chorea Germanorum* of some authors, including the salaam convulsions (*tic salaam*), and the extraordinary affection described in America as the *jumpers*, in Southern Asia under the Malay name of *latah*, in Eastern Siberia as *miryachit*, and in France as *tic convulsif* (in part) and as *Gilles de la Tourette's disease.*

Etiology.—The only cause that can be assigned for automatic chorea is an hereditary neuropathy.

Symptomatology.—Automatic chorea may occur in acute violent outbreaks or may be a chronic condition. In the former case there are usually prodromes, such as melancholia, apathy, malaise, cardiac palpitations, cramps, etc. A paroxysm usually begins with a general excitement which affects all the nerve-functions. Thus, songs are sung, foreign tongues spoken, events described, poetical quotations given in eloquent or usually more or less incoherent ravings, which often seem entirely beyond the normal intellectual power of the individual. In the height of the paroxysm the affected person dances, sings, springs from the ground, rolls himself from side to side, hammers violently with the hands, stamps with the feet, or in a fury of motor excitement whirls with mad rapidity until, completely exhausted, he falls to the ground in a condition of unconsciousness.

Chorea major may exist in a sporadic form or may be epidemic, as in the hysterical religious epidemics of the Middle Ages and in the performances which occasionally occur during revivals in camp-meetings in the United States and among the howling dervishes of Mohammedan countries. The epidemic disease must be looked upon as hysterical. Some of the sporadic cases are probably instances of epileptic automatism. It may well be that a morbid impulse (see p. 385) underlies many cases, such as those in which the attack consists in the person bowing repeatedly on entering a room or approaching an individual (*tic salaam*), or as that of a Philadelphia patient who would suddenly leap from his chair, seize his hat, jam it down on his head two or three times, and then throw it on the ground violently before going on with his business.

The essential feature of *latah* is an extreme excitability of the patient, so that an abrupt excitation by a sudden salutation, by a blow given, etc., causes violent disorderly actions conjoined with a condition of the cerebro-nervous system which necessitates the repetition of voices or sounds (*echolalia*) or the ejaculation of some word, usually obscene (*coprolalia*). In some cases the impulse of imitation is so great as to force the victim to repeat not only the spoken word, but also any act done by a by-stander. Very frequently the sudden nervous excitement is accompanied by an excessive emotion, especially of fear, although such emotion may be entirely foreign to the ordinary nature of the individual. The disease appears to be hereditary. In the Jumping Frenchmen of Maine, described by Dr. George M. Beard, these phenomena were very pronounced, and were associated with a mental condition which required the jumper to obey a loud command, the act of obedience being accompanied with an inarticulate cry of alarm. Dr. Beard tested the echo-speaking or repetition by reading portions of Latin and Greek, when the untutored jumper repeated the sounds of the words as they came to him in a quick, sharp voice, at the same time jumping or making some bizarre motion.

There is no reason for believing that specific medicinal treatment is of avail in any automatic chorea. The general health should be built up, and an effort made to form the habit of self-control.

HEREDITARY CHOREA. HUNTINGDON'S CHOREA.

DEFINITION.—An hereditary affection characterized by general choreic movements, and usually associated with other evidences of disturbed innervation.

ETIOLOGY.—The only known cause is direct heredity. The assertion that if the disease fail to appear in one generation the remaining generations will remain free expresses a general but not invariable rule.

MORBID ANATOMY.—We have no determinate knowledge of the basal lesion of hereditary chorea, although it probably is some developmental departure from the norm in the nervous system. The lesions which have been found at autopsies have been so various that none of them can be considered essential. It is true that Greppin was led to believe, as the result of his own studies, that the histological basis of the disease consists in numerous focal groups, chiefly in the white substance beneath the cerebral cortex, formed of cellules having a nucleus with a highly developed nucleolus, and that Klebs found in one case somewhat similar foci in the same position. On the other hand, Cirincione and Mirto, and also Wharton Sinkler, failed to detect any such bodies in cases carefully studied by them.

SYMPTOMATOLOGY.—Hereditary chorea usually develops in middle life, although it has appeared at or even before puberty. The choreic movements resemble those of St. Vitus's dance, but are more constant, more rhythmical, and less under the control of the will. While standing or sitting the patient is continually repeating the same irregular jerking movements. The walk is especially peculiar: for the first few steps it may be nearly normal, when suddenly it is interfered with by one leg being thrust violently forward and the other one being jerked up to it, so that the subject seems to go with a quick, short hop, almost like a dancing step. The course of the disease is exceedingly slow, and in some cases many years are required before the subject becomes unfit for physical labor. The mental condition is usually but not always abnormal. Excessive irritability, moroseness, melancholia, chronic mania, and dementia have all been noted. The reflexes are often exaggerated, but may be sluggish. The sensations are normal. In some instances there is a peculiar muscular stiffness.

Hereditary chorea never gets well, and is in no way amenable to any known treatment.

TETANY.

DEFINITION.—A disease consisting of tonic spasms, either continuous or paroxysmal ; usually symmetrical ; affecting especially the extremities, but often wide-spread or sometimes confined to one limb ; accompanied by disturbances of sensation, which may be severe, but never by unconsciousness, and not depending upon any known lesion of the brain, cord, or nerves, or upon hysteria.

ETIOLOGY.—In the great majority of cases tetany is due to rickets.* Both in human beings and in dogs and cats it has often followed extirpation of the thyroid. It is said to be directly produced by excessive lactation, by the puerperal state, by exposure to cold, by prolonged fatigue, by diarrhœa, by dilatation of the stomach, by the irritation of intestinal worms, by exposure, and even by the rheumatic diathesis or the infectious fevers, and by ergot, stale fish, and other poisons. Further, it is affirmed that it may result from excessive emotion and spread from patient to patient as an epidemic. Such epidemics have, however, probably been hysterical.

MORBID ANATOMY.—There is no demonstrable lesion in tetany. Some authorities assert that it may be due to the presence of a poison produced in the alimentary canal. Bouveret and Devic have isolated from the contents of dilated stomachs (of persons who had not had tetany) a convulsant which they believed to be identical with Brieger's peptotoxine; whilst I. Jacobson and C. A. Ewald obtained from the urine of a typical case of tetany a ptomaine-like body which disappeared when the patient was restored to health and which was believed to have been formed by bacteria. It may be that various poisons and diathetic conditions have the power of causing a similar condition of the nerve-centres.

---

* The nervous disturbances of rachitic children are :

1. Insomnia. Sweating of the head. Great nervous timidity, so that they are easily affrighted.
2. Face phenomena.
3. Expiratory apnœa and laryngismus.
4. General convulsions.
5. Universal hyperidrosis.
6. Trousseau's phenomena.
7. Spontaneous tetany.
8. Nystagmus and spasmus nutans.

These nerve phenomena may occur in any combination or singly, and are all remarkably improved by the use of phosphorus, which acts by curing the rickets.

SYMPTOMATOLOGY.—Typical tetany consists essentially of successive tetanic convulsive attacks separated by intervals of quiet and repose. The paroxysms may continue for minutes or hours, may cease gradually or abruptly, and may recur at intervals of hours, days, or weeks. Arthralgic pains, formication, or numbness in the hands, radiating pains in the fingers, temporary partial blindness, headache, sense of fatigue, etc., are assigned as occasional prodromes. Usually the spasms are most marked in the upper extremities, and sometimes are confined to them; the fingers are often so drawn together as to form a cone. Rarely there is a more accentuated flexion of the fingers, and still more infrequently the hand and the fingers are stiffly extended. The feet may be attacked; sometimes cramps of the calf occur without distortion, but in other cases the feet are violently extended, with the toes pointing downward; more rarely the feet are flexed. The thigh usually escapes, but spasm of the abductors and crossing of the feet have been noticed. Only in the severest cases are the trunk-muscles affected, but opisthotonos and menacing dyspnœa do occur. Even more exceptional than these are spasmodic closures of the jaw and distortions of the face. During the attack the pulse is usually accelerated; the temperature may be subnormal, normal, or slightly elevated. The course of the disease may be painless. Sometimes, however, neuralgic pains run along the nerves, and usually cramp-pains are present in the affected muscle. Anæsthesia and analgesia are ordinary phenomena.

In some cases which must be considered at present as representing tetany the spasms are continuous, lasting without interruption for many weeks. Such cases appear chiefly among adults, and in our experience have been accompanied by anæsthesia and numbness, but not by actual pain.

The knee-jerks in tetany may be increased or weakened. Anomalies of secretion, such as hyperidrosis, polyuria, albuminuria, and glycosuria, have been noted in some cases; whilst redness and œdematous swelling of the skin, urticaria, zoster, pigmentation of the skin, and a remarkable falling out of the hair and nails have been recorded. There are certain important and peculiar symptoms more or less characteristic of tetany, any, all, or none of which may be present. These are the *Trousseau*, the *Chvostek*, the *Erb*, and the *Hoffmann symptom*, so named from their respective discoverers.

Trousseau discovered that if during the period of quiet the main nerve or artery of the limb were pressed upon firmly from two to three minutes tetanoid contractions would occur in the limb. Chvostek's symptom is due to an exaltation of the mechanical excitability of the motor nerves, so that if a nerve be struck with the fingers or with a percussion-hammer pronounced contractions will be produced in the tributary muscles. As this symptom is most frequent and most marked in the face, it is often spoken of as the *face phenomenon.* Erb's symptom is an increase of the electrical excitability of the motor nerves, which is shown to some extent with the faradic but is more marked with the galvanic current. Hoffmann's symptom is the outcome of an increase of the mechanical and electrical sensibility of sensitive nerves, so that slight pressure or slight faradic or galvanic irritation will produce severe paræsthesia throughout the whole distribution of a nerve. A similar hyperexcitability has been noted in the nerve of hearing. Of these symptoms, according to Krafft-Ebing, Erb's is the most frequent; Trousseau's is present in not more than sixty per cent. of the cases; whilst Chvostek's is comparatively rare.

Latent Tetany.—Sometimes in rickety children the face phenomenon may exist without other symptoms, and sometimes tetanoid attacks may be produced by Trousseau's method when they do not occur spontaneously.

DIAGNOSIS.—The spasms of tetany are so characteristic that usually there can be no difficulty in making the diagnosis. The disease is at once separated from tetanus by the absence or late and feeble development of trismus, by the peculiar positions taken by the hands, and by the lack of intenseness of the symptoms. Trousseau's phenomenon is characteristic, and occurs in no other nervous disease; but the peculiar nerve excitability may exist in tuberculosis and other conditions, although it is very rarely so pronounced as it often is in tetany.

PROGNOSIS.—Tetany almost always, unless dependent upon an irremovable cause, such as extirpation of the thyroid, is recovered from. When it occurs during pregnancy it may continue until the birth of the child, and then disappear. The length of the attack depends upon the character of the cause. It is, however, essentially an enduring affection, usually continuing for some weeks or even months.

TREATMENT.—In the treatment of tetany it is necessary to remove, if possible, the cause. Thus, in rachitic cases phosphorus should be administered. In gastro-intestinal cases the stomach and intestines should be thoroughly emptied, and be kept as nearly normal as possible. Cases (rheumatic?) have also been reported in which the whole attack was put an end to by a full dose of pilocarpine.

In very bad cases it may be necessary to control the attacks by morphine and chloral. Hyoscine has recently been very strongly recommended, and would seem to be indicated by its physiological action. We have had no opportunity of testing it. The bromides, especially ammonium and strontium bromides, are often of great service. Warm baths and hot packs, either general or to the affected limbs, are sometimes very useful. Little is to be hoped for from electricity: the faradic current will probably do more harm than good; a very weak galvanic current passed from the centres upward rarely does good.

Hygienic treatment looking towards the strengthening of the system should always be assiduously practised.

PARAMYOCLONUS MULTIPLEX.

Definition.—An affection characterized by paroxysms of clonic contractions chiefly of the muscles of the extremities.

Etiology.—Most of the cases have originated in fright, and have occurred in male adults. Weiss asserts that he has seen several cases in one family.

Morbid Anatomy.—Tambroni and Pieracini have recorded cases dependent upon organic lesions of nerve-centres. Other cases have been clearly instances of hysteria. Weiss maintains that out of fifty-one recorded cases only thirteen were really instances of the disease; whilst Möbius believes that Weiss's own cases were, in fact, cases of hereditary chorea.

Symptomatology.—The clonic spasms usually begin in the muscles of the legs. They may at first be controlled to some extent by the will and be not severe enough to prevent work. They are, as a rule, bilateral, and vary from fifty to one hundred and fifty in a minute. In the intervals between the attacks there may be tremors in the muscles. Sometimes the contractions are definitely rhythmical. They may involve the muscles of the back and of the abdomen, and become so severe as to make it difficult to keep the patient in bed.

Prognosis and Treatment.—The cases usually recover, but fresh attacks at shorter or longer intervals are common. The treatment is that for hysteria and neurasthenia.

PARALYSIS AGITANS.

Definition.—A disease of advanced life, characterized by tremors which continue during the waking hours and are associated with muscular weakness and rigidity.

Etiology.—Paralysis agitans rarely occurs in persons under forty years of age, is most frequent between fifty and sixty, and is more common in men than in women. It is not distinctly hereditary, and can rarely be traced to an exciting cause, although it has been produced by violent fright, prolonged anxiety, exposure, and even physical injury.

Morbid Anatomy.—None of the pathological theories which have been brought forward in regard to paralysis agitans have sufficient plausibility to require discussion here.

Symptomatology.—The onset of paralysis agitans is usually insidious, but in occasional cases may be abrupt. A transitory tremor appears in the hand or foot, or even in one finger or toe. At first it can be controlled by an effort of the will, and is suspended by voluntary motion.

Little by little, however, it becomes more fixed and more wide-spread, until at last it continues throughout the waking hours, during repose as well as during action, and is uncontrollable. From the limb first involved it usually passes to the limb of the same side, constituting the hemiplegic form, or, especially when it begins in the leg, it may cross to the opposite limb. The face is rarely attacked by the tremors, although in the later stage it is affected by the rigidity and takes on a peculiar fixed immovable, usually melancholy, expression. According to Charcot, the head is never directly affected, any apparent trembling of it being due to transmission of motion from the trunk ; but this is certainly too absolute an assertion. In the advanced stages of the disease the peculiar prone or forward position of the head and the weakness of the lips often produce a dribbling of the saliva. The speech at the same time becomes a little slow and labored, but is never profoundly affected. Neither eating nor swallowing is interfered with. The tremors are short, rapid, sometimes rhythmical, and in the fingers occasionally assume an appearance of purposive action, as though the patient were rolling something between the digits. Regular rhythmical sounds will often alter the rate of the tremor without the patient's being conscious of it. Violent muscular contractures never occur, but a peculiar rigidity develops, giving rise to the characteristic fixation of the affected parts, and especially to the statue-like rigidity of posture.

In standing the trunk is inclined forward, with the face looking obliquely downward ; the forearms are usually flexed somewhat upon the arms, the hands a little bent upon the forearms, and the fingers partially closed, so that the hands assume a position similar to that in which the pen is held; hence the term "writing hand" as given by Charcot. The same tendency to flexion of the legs exists, so that in standing the knees are bent. Occasionally, peculiar distortions of the hands or other portions of the body may be met with. On attempting to restore the normal position of the parts the muscles usually offer but little resistance until the restoration is nearly perfected.

Complete paralysis never occurs in paralysis agitans, but very early in the disease loss of endurance manifests itself, and it commonly increases until only brief muscular efforts are possible.

An almost characteristic symptom is festination,—that is, a progressive increase in the rapidity of gait : apparently owing to the position of the body, the subject in walking thrusts one leg forward more and more quickly in order to prevent toppling over, so that the walk becomes more and more rapid, and in a little while in extreme cases may break into a run, which grows faster and faster, until the patient either falls or arrests his course by seizing hold of some stationary object. That the festination is not dependent simply upon the position of the body is shown by the facts that a normal gait sometimes exists in subjects who are strongly bent forward, and that in very rare cases there is a tendency in the patient to run backward instead of forward. Sensibility is not usually markedly affected, nor is there suffering, except in the advanced stages from the perpetual sense of fatigue in the affected muscles, which may amount to a severe aching. There is often a feeling of excessive heat, accompanied by continual sweating ; in such cases the central bodily temperature is normal, but according to Grasset and Apollinario there is an elevation of the surface temperature. Thus, these observers found that in the normal individual the surface temperature was 33.6° C., whilst in a case of paralysis agitans placed under exactly the same circumstances it was 36.8° C. According to Regnard, the elimination of urea is normal, that of the sulphates less than the norm. Chéron affirms that there is a constant increase in the excretion of the phosphates, which is characteristic and may precede the development of the tremors.

Paralysis agitans requires many years for its full development, but, unless the patient dies of some intercurrent disorder, hypochondriasis, great depression of spirits, loss of intellectual power, general failure of nutrition, marked emaciation, marasmus, and finally death from exhaustion occur.

DIAGNOSIS.—Senile tremors may simulate paralysis agitans. In them, however, the head is especially affected, and there are usually tremblings of the tongue and lower jaw. It is stated that the loss of power, the rigidity and fixation of the limbs, and the peculiar gait and later evidences of general nerve degeneration of paralysis agitans may develop without the tremors. The diagnosis of such a case could be arrived at only after long watching. Blocq and Marinesco have reported a case with hemiplegic tremors, which was diagnosed as paralysis agitans, in which the lesion was a tumor in a cerebral peduncle.*

PROGNOSIS.—Recovery never takes place in paralysis agitans.

TREATMENT.—There is no specific treatment. The patient should lead a quiet regular life, with absolute avoidance of physical or mental labor. Electricity, which has been much used, has no real value. The hot bath is often of service as a palliative, especially in the advanced stages. Conium, hyoscyamine, cannabis indica, morphine, chloral, alone or in various combinations, may be useful for the purpose of procuring sleep, if necessary, or for giving relief. The formation of the narcotic habit, however, is always imminent. In our hands the best results have been obtained from hyoscine hydrobromate, given at night, or during the day when the tremors are excessively painful.

TRAUMATIC NEUROSIS.

DEFINITION.—A condition of neurasthenia, usually with hysterical symptoms and local lesions, produced by severe injuries.

Traumatic neurasthenia is so invariably associated with symptoms which are of local origin, the direct result of the injury of the part affected, that it is necessary to speak of these complicating symptoms before discussing the main subject. A blow upon a muscle suspends its function without lacerating its structure, probably by a concussion of the nerve-endings. This condition is rare, except in the deltoid muscle, in which it is frequently produced by a fall. The treatment consists of acute antiphlogistic measures, followed when all inflammation has subsided by the injection of strychnine into the muscle, of the use of massage for the purpose of freeing the muscles and muscular fibre bundles from exudation, and of stimulating nutrition, with the application of that electrical current which will produce the greatest contraction of the affected muscles with the least pain to the patient. In cases of traumatic neurasthenia the so-called *traumatic back* is very common. The condition may be produced by a direct blow, but is more commonly the outcome of a sudden jerk or wrench which produces a sprain of the fibrous tissues of the back and probably gives rise to a deep-seated inflammation, which, although primarily situated in the fibrous structure, may implicate periosteum and nerve-roots and produce very serious results. Directly after an accident the amount of injury to the back may not be apparent. The symptoms are tenderness more marked upon deep firm pressure than upon slight pressure, and restriction of movement by pain and by spasm of the erector spinæ muscles. Reflex spasms may also be producible in the back muscles by jarring, or by pressing upon the head or the vertebral column. As in other forms of sprain, rest is the basis of treatment of the traumatic back, and for the purpose of obtaining it and of relieving the effects of pressure the plaster jacket, or some other form of the Sayre jacket, is often very useful. To its employment in the ordinary manner may be added that originally suggested by H. C. Wood, which we have found very useful, especially when the traumatism is in the lumbar region of the back. It consists in swinging the patient many hours a day more or less completely from the upper of the two cones forming the human trunk. In carrying it out the patient should be suspended in the ordinary way for putting on the plaster jacket, and, when the first layer of the plaster jacket has been put in place, two broad, strong linen bandages, well wetted, are to be so arranged, one over each shoulder, that they shall form above a loop, whilst the ends hang down front and back below the plaster bandage. With new turns of the plaster bandage these vertical linen bandages are to be fastened in position, and afterwards the loose ends of the linen bandage are to be drawn up and firmly secured by further turns of the plaster bandage. Often by fastening such a jacket to the head-board the bed may be kept strongly inclined without inconvenience to the patient, but with a constant drag from the lower extremities, separating the sore vertebræ. Severe counter-irritation is sometimes useful in traumatic back, and careful massage should always be tried.

---

* Compt.-Rend. Soc. Biolog., v., 1893.

Etiology.—Railroad injuries, falls from hatchways, press of steam from exploding boilers, any violence acting upon the trunk through crushing local force or so as greatly to shake and shock the whole system, may produce traumatic neurasthenia, as may also sudden twists and wrenches of the back in railroad and other accidents.

SYMPTOMATOLOGY.—The symptoms of traumatic neurasthenia may appear at once after the injury or may come on insidiously. The subjective symptoms are malaise, loss of ambition, marked nervous irritability, failure of the power of mental and physical labor, depression of spirits, occasional headache, pronounced tinnitus aurium, broken sleep, diminished sexual power, and general failure of health. Almost invariably to these symptoms are added various hysterical manifestations. Probably among these must be classed the extraordinary cerebral attacks which come and go often without obvious cause or explanation. Sometimes these attacks resemble petit mal, in that they consist of short moments of unconsciousness; sometimes the paroxysm is prolonged and consists of an active delirium, which may amount to a furious and aggressive mania. Often the patient has no remembrance of these attacks. Distinctly hysterical paroxysms are not rare. Neurasthenic vaso-motor weakness is common, so that sudden flushings of the face and abrupt outbreaks of sweating are frequent. The muscular irritability is often greatly augmented; the knee-jerks may be exaggerated, but vary from day to day, and become rapidly exhausted by re-excitation, or even by general bodily fatigue. Ankle-clonus is rare. Paradoxical contractions may often be produced in the anterior leg muscles by flexure of the foot, and the slightest irritation may in some cases cause a general reflex contraction of the erector pilæ muscle, with a consequent "goose-flesh." The sexual power is commonly not altogether lost, but sexual irritability and weakness are usually shown in men by premature emissions. True diabetes, with its secondary results, may be present.

PROGNOSIS.—The course of this disorder is essentially slow, requiring years for recovery, which is apt to be imperfect. Death is very rare, unless the violence has been sufficient distinctly to compromise the nerve-centres.

DIAGNOSIS.—Owing to medico-legal complications, the first question to be asked in a case of traumatic neurasthenia is whether the symptoms are real or feigned, and, if real, how far they are exaggerated. It is next essential to determine, for the purposes of prognosis, the proportion of local diseases, of neurasthenia, and of hysteria. Purely traumatic hysteria, the outcome largely of fright, yields usually to skilful treatment without very great delay; whereas both the local disorder and the traumatic neurasthenia, if well developed, are always very serious, requiring very long continued careful treatment, and in many cases remaining permanent to a greater or less degree. It is essential, therefore, upon the witness-stand clearly to distinguish between a simple hysteria produced by an injury and the more serious condition. It must also be remembered that these neurasthenics are from the very nature of their cases more or less hysterical.

It is further necessary in cases as they present themselves to decide as to the involvement of the spinal cord; absolute loss of sexual power, paresis of the bladder, trophic lesions, and persistent uniform rigidity or a complete paralysis of any part are very strong evidences in favor of the spinal lesion. The same is true of an anæsthesia which is pronounced and not hysterical.

TREATMENT.—Absolute rest in bed, with a very careful use of massage and electricity, and the administration of narcotics for the purpose of relieving pain, are the chief measures to be early practised in traumatic neurasthenia. Tonics are of very little value. With narcotics there is always the very grave danger of the formation of the narcotic habit. In the most successful case we have ever seen, a physician treated himself chiefly by drinking from three to four pints of strong ale daily, through extraordinary strength of character finally escaping the narcotic habit. The faradic current ordinarily irritates and does harm; the continued galvanic current, passed from the centres to the periphery of the nerves, often is soothing, and perhaps permanently advantageous. The moral support of the patient by care in the arrangement of the details of life, and by the semblance of medication, is often of the greatest practical importance. Later the subject should be induced to settle down, preferably in a rural district, to quiet life, free from excitement and serious labor. Any fatigue usually aggravates the condition.

CAISSON DISEASE.

DEFINITION.—A disease of uncertain pathology, occurring in those who work in compressed air.

ETIOLOGY.—All reported cases have occurred in those who work in caissons or other chambers in which the air is highly compressed. As the abrupt passage from the caisson to the outer air is exceedingly dangerous, arrangements are usually made to have the pressure gradually reduced to the norm; but no precautions have hitherto been completely effective.

MORBID ANATOMY.—No immediate autopsies are on record. Two months after exposure, Dr. Caspar W. Sharpless found foci of softening apparently due to minute hemorrhages and secondary inflammation. In other cases similar disseminated focal myelitis has been found. The theory that the symptoms are due to sudden evolution of compressed gas from the blood into the nerve-centres is plausible, but has not been proved.

SYMPTOMATOLOGY.—The symptoms of caisson disease usually develop in from half an hour to two hours after the return of the subject to the outer air. Violent pains occur in the limbs and in the hands, followed in a few minutes by progressive loss of motor and sensory power in the legs. Notwithstanding the anæsthesia may become complete, the pains continue, whilst headache, dizziness, double vision, incoherence of speech, mental aberration, and sometimes unconsciousness, rapidly develop. The patient may convalesce in a few days, or death may take place quickly with apoplectic symptoms, or may follow from paralytic bedsores and cystitis after some months. Usually, however, recovery occurs after a prolonged period of atrocious suffering and motor disablement. The treatment is expectant and palliative.

HEAT EXHAUSTION.

Definition.—A condition of profound exhaustion, with lowered bodily temperature, excessive sweating, and disturbed innervation, due to the combined action of heat and exertion.

Symptomatology.—The symptoms of heat exhaustion vary from those of intense tire to those of collapse. In severe cases the attack may develop rapidly, even with the absolute abruptness of a syncope, the symptoms being unconsciousness or semi-unconsciousness, muttering delirium, great restlessness, facial expression of collapse, rapid, feeble, scarcely perceptible pulse, and a temperature which may be as low as 95° F.

Diagnosis.—With a knowledge of the history it is hardly possible to mistake heat collapse for the collapse due to organic disease. The important points in the diagnosis are the temperature and the absence of evidences of internal hemorrhage and of heart or other chronic diseases. From true thermic fever the case is at once set aside by the temperature.

Treatment.—The free immediate use of external heat,—if possible, the hot-water bath,—the hypodermic injection of atropine, strychnine, cocaine, and digitalis to dry the skin and to stimulate the heart and vasomotor system, and a very moderate internal use of hot alcoholic drinks and of ammonia, will invariably lead to cure, unless some chronic disease underlies the condition.

THERMIC FEVER. SUNSTROKE.

Definition.—Acute fever produced by exposure to heat.

Etiology.—Thermic fever is always dependent upon exposure to heat, natural or artificial. Owing to interference with evaporation, a hot moist atmosphere is much more dangerous than is dry heat. Hence sunstroke is rare in dry climates and frequent in tropical lowlands, as well as in sugar-refineries, laundries, and similar places. It may occur in the night as well as in the day. Very powerful as predisposing causes are lack of acclimatization, excessive bodily fatigue, and intemperance. Males are more frequently affected than females, because of their more frequent exposure.

Morbid Anatomy.—Owing to the intense heat of the body, post-mortem changes begin at once after death, and of such nature were most of the lesions described as occurring in sunstroke by early writers: moreover, the post-mortem findings are greatly modified by the treatment and the time of death. If the patient have died during an acute sunstroke, with high temperature, and the post-mortem be made at once, the left heart will be found contracted, the right heart usually engorged, the semi-fluid blood collected in the venous trunks, and the arterial coats, or it may be the whole body, marked with petechiæ or stained with decomposing blood. In some cases the blood has an acid reaction. (Wood.) Many years ago H. C. Wood proved that the cause of the symptoms and the structural lesions in thermic fever is simply excessive heat. The history of the development of an attack is probably at first a slow rise of the bodily temperature, produced by the inability of the system to get rid of the heat which is formed in it; after a time the inhibitory heat-centres at the base of the brain, which control the formation of bodily heat, become exhausted by effort or by the fever itself; and as a consequence of the removal of inhibition there is a sudden increase of the formation of heat, with a corresponding up-bound of the bodily temperature and consequent unconsciousness from the paralyzing influence of the heat upon the cerebral cortex. All the higher tissues of the body are affected directly by the excessive temperature, and death from a pure heat paralysis of the respiratory centres may quickly occur.

Myosin (the substance whose coagulation produces post-mortem rigidity) coagulates at about the maximum temperature of sunstroke. After severe exertion the muscles, including the heart, contain an excess of a myosin which is more prone to undergo coagulation than is normal myosin. In this fact is found the explanation of the extraordinary positions of the corpses of those who have been killed in battle: instantaneous death has been followed by an equally instantaneous coagulation of the myosin of the general muscles, so that the body has been frozen in the attitude at which life was stopped. The heart is in the centre of the bodily heat: not rarely in tropical battles, especially when troops have been charging uphill, the overstrained heart has been suddenly arrested by the coagulation of its myosin, and the man has fallen on his face in instantaneous syncopal death.

SYMPTOMATOLOGY.—The mildest form of sunstroke is the subacute variety, which was described by the physicians of India many years ago under the name of *ardent continued fever*, and which was especially studied in America by Professor John Guitéras, who showed that the so-called typhoid fever of Key West is in most part this affection. The symptoms are a continued fever without local disease, with a tendency to weakness and the typhoid state, and various disturbances of function. In India, it is stated, the cases are apt to end in the sudden development of the severest type of thermic fever and death; in America they usually recover under treatment.

Sunstroke commonly begins with abrupt complete unconsciousness, although prodromes, such as general distress, a great burning heat, and chromatopsia, or colored vision, do occur. With the unconsciousness there are usually muttering delirium, great muscular restlessness, partial convulsions, or violent epileptiform attacks; sometimes there is quiet coma with relaxation. The surface of the body is always hot; at first dry, it may later be bathed in a profuse perspiration; the face is flushed and the eyes are suffused; the pulse may be bounding and full, but is almost invariably compressible, and if not originally rapid and feeble becomes so as the case progresses. Vomiting and purging are very common. The whole body is apt to exude a peculiar odor, which is especially strong in the fæcal discharges. The characteristic symptom is a temperature which is rarely below 108° and may reach 113° F. The urine is scanty, sometimes albuminous, not rarely suppressed. The breathing is more or less labored and irregular. The pupils are dilated. In most cases some response can be obtained by shaking the patient, except very late in the disorder. Death may occur in about half an hour, but usually is postponed for a longer period; it is ordinarily the result of a slow simultaneous failure of respiration and of heart action, but may be due to asphyxia, or in very acute cases to cardiac arrest.

A condition similar to sunstroke may develop in so-called cerebral rheumatism and other affections with very high temperature. As was first pointed out by Dr. Comegys, many of the cases of so-called cholera infantum occurring in young children in the large cities of America during the summer months are really forms of thermic fever. The symptoms in such cases are high fever, intense thirst, rapid pulse and respiration, vomiting, purging, and more or less pronounced evidences of cerebral disturbance, such as insomnia, headache, contracted pupils, delirium, and finally coma ending in death.

DIAGNOSIS.—In a certain sense the diagnosis of thermic fever is made at the moment the temperature is found to be 108° F. or upward, because such temperature produces a thermic fever whether it (the temperature) is due to external heat or not. The diagnosis of thermic fever in the narrow sense of the term requires, however, a knowledge of the exposure to heat.

PROGNOSIS.—The prognosis in thermic fever depends chiefly upon the time at which treatment commences. If the high temperature has lasted a sufficient length of time to produce alteration in the nerve-centres and in the blood, the symptoms may not be interrupted by the reduction of the temperature. Almost invariably, if the temperature be reduced immediately after the subject falls, consciousness will return and the case go on favorably, if relapses be guarded against. The persistence of nervous symptoms after a reduction of the temperature is of the most serious import.

TREATMENT.—When exposure to high temperature cannot be helped, it is essential that the bodily health be maintained and that all excesses in labor or in pleasure be avoided. The diet should be largely farinaceous, and the emunctories should be kept active by the eating of fruit, and by the free use of cold water and of lemonade, and of mild salines if necessary. Ice-water, if taken in large quantities, may do harm by sudden chilling of the stomach, and drunk at meals may interfere with digestion; but ingested in moderate quantities at short intervals between meals it does great good by reducing the general temperature and aiding free perspiration. All alcoholic drinks are to be avoided, except that a little claret or red wine may often with advantage be added to the water taken, to make it more acceptable to the gastro-intestinal tract and more active in promoting perspiration. Cold baths should be used frequently, especially if at any time the bodily temperature be found to be rising.

The use of cold is the basis of all treatment of sunstroke. According to Guitéras, in subacute or continued thermic fever the best plan is to wrap the patient in a dry sheet, lift him into a tub of water at a temperature of 80° F., and then rapidly cool this water by means of ice, the immersion continuing from forty-five to fifty-five minutes, according to the effect upon the mouth temperature. The patient is then to be placed upon a blanket, the skin partially dried, and the body covered. Guitéras states that it is very important to avoid currents of air blowing upon the patient, and to have the bath given in a small warm room; also that in most cases great advantage is to be obtained by giving moderate doses of whiskey, with from twenty to thirty minims of tincture of digitalis, about twenty minutes after the bath. Guitéras never found it necessary to give more than two baths in the twenty-four hours, but in some cases the baths had to be used for many days.

In acute thermic fever the bodily temperature is to be reduced at once by the means most convenient: there should be no waiting for the summoning of a physician. The patient should be carried into the shade and have cold affusions over the face and body, or should be put at once under a pump and be pumped on, or should be plunged into a bath of ice-water. City ambulances, when it is practicable, should be furnished with ice and antipyrin, so that when the sunstroke patient is reached treatment may be commenced at once. In giving the cold bath a non-registering thermometer should be in the mouth or the rectum of the patient, the axillary temperature not being a true indication of the temperature of the body. The patient should be removed from the bath when the bodily temperature reaches 101° F., since it is not rare for the fall of temperature to continue after removal from the bath. Alcohol, and strychnine and digitalis hypodermically, may be useful even while the patient is in the bath, when the symptoms seem to point urgently to them; antipyrin is a valuable remedy for the purpose of preventing rise of temperature after the patient has been taken out of the bath.

When there is in sunstroke a hard pulse, and the symptoms are essentially those of a congestive apoplexy, free venesection is sometimes useful, especially as it is a powerful reducer of bodily temperature. When the convulsive tendency is very acute, morphine may be given hypodermically.

As soon as may be after sunstroke the patient should be removed to a cool atmosphere, and should be kept upon a light farinaceous diet, and generally treated as though in danger of an acute meningo-encephalitis. Especially if there be any tendency to headache, or cerebral flushings, local blood-letting followed by blisters and other forms of counter-irritation should be used. If the headache when the patient first comes to himself be intense, general venesection may afford a means of relief.

Sequelæ.—Cerebral distress or pain, with failure of general vigor, dyspeptic symptoms, and various indications of disturbed innervation frequently occur after thermic fever. In pronounced cases the pain in the head is more or less constant, but subject to exacerbations, and is sometimes associated with pain and stiffness in the back of the neck. With it there may be vertigo, decided failure of memory and of the power of fixing the attention, insomnia, and excessive nervous irritability. When this is the case there is usually a marked lowering of the general health, with loss of strength, and the peculiar invalid look which characterizes severe chronic disease. Epileptic convulsions, with very pronounced evidences of severe cerebral inflammation, occur in rare cases. The one symptom which is always present, and which is diagnostic in these cases, is the inability to withstand heat: not only are the symptoms greatly exaggerated, it may be to the point of severe illness, during the summer months, but in most cases headache and great distress are produced by going into a hot room even in winter. The lesion underlying the condition just spoken of is a chronic irritation of the brain membrane and cortex, passing, if the case be severe enough, into pronounced chronic meningitis with greater or less involvement of the cortex.

The treatment is, first, absolute avoidance of any exposure to even moderate heat, combined with intellectual and physical rest; second, the treatment of non-specific chronic meningitis,—*i.e.*, local bleedings and persistent, merciless counter-irritation, especially by means of the actual cautery, combined with the internal administration of mercurials and potassium iodide in very small continuous doses (one-fiftieth grain of corrosive sublimate, one to two grains of iodide, three times a day); third, restriction to a largely farinaceous, non-irritating diet, and careful attention to all minor symptoms as they arise.

OCCUPATION NEUROSES.

DEFINITION.—Localized disturbances of motion produced by the excessive use of groups of muscles in the daily occupation of life.

ETIOLOGY.—Occupation neuroses are almost indefinite in number. Piano-forte-player's cramp, violinist's cramp, telegrapher's cramp (chiefly among those who use the Morse machine), dancer's palsy (especially affecting the calf-muscles), hammer palsy (occurring chiefly among goldbeaters), chisel cramp (among mechanics), etc., are ordinary forms: but seamstresses, tailors, money-counters, watchmakers, engravers, knitters, etc., are occasionally disabled by peculiar occupation neuroses. The occupation neurosis is not caused by severe muscular efforts, but by the excessive repetition of movements which require fine coördination.

MORBID ANATOMY.—The pathological condition in the occupation neuroses is probably one of local neurasthenia, with habitual congestion and irritability of the affected centres.

SYMPTOMATOLOGY.—The characteristic symptom of the occupation neuroses is the coexistence of the disablement for the habitual fine action with preservation of muscular power. Thus, a man who cannot grasp the pen may wield with ease a fifty-pound dumb-bell. In 1868, Moritz Benedict distinguished three forms of occupation neuroses,—the paralytic, the spasmodic, and the tremulous. Whilst typical cases of these varieties are occasionally seen, usually the symptoms are mixed. According to our observations, paralysis is in most cases the dominant symptom, tremor the least developed.

As writer's cramp is the most common of the occupation neuroses, we shall consider it as the type. In it there is usually some painful fatigue in the arm, which may be associated with slight formication and numbness, and rarely with tenderness over the nerve-trunks. Any attempt at writing produces intolerable pain in the part, often accompanied by a sense of stiffness, or even by a distinct muscular resistance when the effort is made to grasp the pen. In severe cases the sense of fatigue continues even after long resting of the arm, and there may be with it a distinct pain between the shoulders. The stiffness and occasional cramp of the fingers around the pen reveal the nervo-muscular irritability, in the paralytic form of writer's cramp; but in the spasmodic form irregular muscular contractions are the most prominent manifestations. At first slight spasmodic movements of the thumb and first finger produce an occasional irregular stroke in the writing, but as the disease progresses, occasional sudden extension of the finger causes the pen to be dropped, or by a spasmodic action of the opponens pollicis, with abduction and coincident flexion of the index finger, the pen is rapidly moved from the paper, or occasionally a violent spasmodic flexion of all the concerned fingers holds the pen as in a vice.

In the tremulous form of writer's cramp the tremors may become so excessive that the pen follows them rather than the directions of the will, and no trace is left upon the paper but irregular, undulating, and angular strokes. According to our observations, tremors are especially common in telegrapher's cramp.

PROGNOSIS.—Occupation neurosis almost invariably gets well, provided that absolute rest from the occupation can be secured for a great length of time. Recovery is, however, always slow, and the disablement has a pronounced tendency to return upon repetition of the habitual acts.

TREATMENT.—In writer's cramp, as in other occupation neuroses, it is essential for the subject to cease doing the act which has produced the peculiar exhaustion. The direct treatment of the part is very unsatisfactory. No internal medication is of any use, except as it may benefit the general health of the patient and overcome any neurasthenic tendency. Massage seems to be of distinct value. Electricity has been very largely employed, and is by some authorities strongly commended, by others spoken of with despair. It seems, in fact, to do good in some cases, but more often its influence is not perceptible. Faradization may do harm, as the muscles are commonly irritable; it rarely, if ever, does good. The best application is the long-continued use of a mild current of galvanic electricity passed down the nerve of the affected member, of just such strength as to be distinctly but not painfully perceived. A small positive pole should be placed over the nerve-trunks in the groove of the inside upper arm, whilst the hand rests upon a large well-wetted sponge connected with the negative pole.

Clerks and others who are required to use the arm excessively in writing should employ a light pen-holder, half an inch in diameter (cork preferable), with a blunt-pointed steel pen or a quill pen, and should adopt the free-writing method, in which the movement is chiefly from the shoulder and the words are formed without lifting the hand from the paper. Various writing-machines have been invented, but are unsatisfactory. In many cases the solution of the practical difficulties is to be found in the use of a type-writing machine. When it is necessary that the writing shall be continued at all hazards, the left hand may be employed. In writing with it, it will be found easier to reverse the lines,—that is, to write with the slope from left to right. Usually the left hand soon develops the disorder, even though the greatest care be taken not to overwork it.

HEADACHES.

Headaches not due to diseases of the brain or its membranes can be for practical purposes classed as toxæmic; sympathetic, due to some peripheral lesion; and nervous, including various headaches not in the two other groups. It is not possible by a study of the headache itself to decide its nature, so that the examination of a case of persistent headache should be thorough and absolute.

Toxæmic Headaches.—Of the toxæmic headaches the most important are secondary to diseases of some organ, such as the kidney, the heart, or the lungs, or are malarial, rheumatic, lithæmic, alcoholic, caffeinic, or gastric. Any disease which interferes with the aeration of the blood, or with its purification by the emunctories, is liable to produce headache, whose cause is to be made out only by finding the disease.

Brow ague is an intense pain occurring in more or less regular paroxysms, usually in the immediate neighborhood of one supraorbital foramen, often associated with fever, sweat, or other malarial indications. Its nature is to be recognized by its periodicity and by its yielding to very large doses of quinine (forty grains).

Rheumatic headache is usually a heavy ache, but may be associated with sharp neuritic pains. It is to be recognized by the existence of a rheumatic diathesis, by the effect of weather, and by the marked soreness of the scalp which usually accompanies the pain. In doubtful cases the therapeutic test with salicylates should be made.

Gouty or lithæmic headache is often dull, heavy, and worse in the morning; but it may occur in very acute paroxysms at irregular intervals, and be associated with vertigo, staggering, or even epileptoid spells, so as to lead to the mistaken diagnosis of organic brain lesion. It is probable that in some of the cases there is really a gouty meningitis, with or without deposit of urates. The diagnosis is to be reached by exclusion and the recognition of the existing diathesis. The treatment is that of irregular gout.

The caffeinic headache is especially common in women of neurotic temperament, but occurs also in men. For some unknown reasons there are numerous individuals who can drink without suffering tea, but cannot take coffee; others, fewer in number, who can use coffee, but not tea. The worst cases of caffeinic headache occur in sewing-women and factory-girls who are underfed and stimulate themselves for work by tea. The caffeinic headache ceases when total abstinence from caffeinic drinks has been enforced for a month.

Gastric headache, when due to acidity, may occur in violent paroxysms and dizziness, and yields rapidly to ammonium and sodium bicarbonate. The headache of indigestion with hepatic torpor (biliousness) is usually frontal, may be occipital, and is often associated with defective vision, giddiness, and great depression of spirits.

Sympathetic Headache.—Headache may result from almost any peripheral irritation, but is extremely common from eye-strain, and occasionally occurs from disease of the nose. The headache of eye-strain is often frontal, perhaps more frequently occipital, but varies greatly in character, and may even be indistinguishable from a migraine. It is usually aggravated by the use of the eyes, is apt to be severe in the morning after an evening spent in a place of amusement, but is to be diagnosed with certainty only by finding and correcting the optic defect, when the pain disappears. Usually it is relieved almost at once by paralyzing vision with atropine and employing dark glasses. The character of a nasal headache may often be detected by the tenderness of the inner wall of the orbit when pressed upon by the finger, or by the pain caused by touching the middle turbinate bone with a probe. In many cases, however, the nasal significance of headache can be determined only by removing the nasal disease.

Nervous Headaches.—In this group may be put the headache of anæmia and exhaustion, the congestive headache, the hysterical headache, and certain rare headaches whose cause cannot be elucidated, and which may therefore be spoken of as essential. The headache of anæmia and exhaustion is very often rather a sense of weight and distress than a true pain. It may, however, be very severe, and be accompanied by so much flushing of the face and conjunctiva as to mislead the practitioner into supposing that there is a true brain congestion. It is commonly relieved by stimulating foods, such as strong beef-essence, or by milk-punch or other nutritive alcoholic drink. The headache of acute cerebral hyperæmia is usually due to some demonstrable cause, such as traumatism, exposure to the sun, malarial fever, etc. Its nature is to be recognized by the general excitement of the circulation, the strong pulsations in the carotid, and the tendency to coma and delirium. Headache in hysteria may take on any form, but may be considered as characteristic when it is a pain situated in the middle of the top of the head, in a point so small as almost to be covered with the tip of the finger (*clavus*).

MIGRAINE.

DEFINITION.—An hereditary paroxysmal headache, without obvious cause, usually appearing at puberty and gradually disappearing after the age of fifty.

ETIOLOGY.—The only known cause for this disorder is heredity.

MORBID ANATOMY.—Some authorities are inclined to believe that migraine is a vaso-motor neurosis, and Eulenberg has described two varieties, one with vaso-motor spasm, as shown by pallor of the face, dilated pupil, and hard temporal artery; the other with evidences of vaso-motor relaxation, such as redness and heat of the face, injection of the conjunctiva, lachrymation, and inflammation of the affected ear, with free lateral sweating; but as the disease occurs in America these two varieties cannot be made out.

Of the basal nature of migraine we have no knowledge. The paroxysms are evidently of the nature of nerve-storms, which suggested many years ago to Trousseau that migraine has a relation to epilepsy. There are undoubtedly cases in which migraine and epilepsy coexist; others in which the two forms of paroxysms seem to replace each other. Nevertheless it is in the highest degree improbable that there is any relation between ordinary migraine and the more serious disorder. Cases indicating relationship are probably not more than one in five hundred, and no interchangeability in heredity can be traced, epilepsy not being demonstrably more frequent in families having a strong migraine heredity than in the general community. The best explanation of the rare cases is, therefore, the coexistence of two neuroses. The relations, on the other hand, between migraine and gout seem very close, almost all cases seen in Philadelphia, at least, having a distinct gouty family history.

SYMPTOMATOLOGY.—Migraine occurs in paroxysms, which may be separated by a few hours or many months. The attack is usually preceded by malaise, chilliness, and a sense of languor, or more rarely by a condition of exhilaration. In most cases the pain commences in the forehead near the supraorbital foramen, and gradually increases in intensity until it becomes unbearable. It is variously described by sufferers as boring, throbbing, or shooting, and is sometimes situated in the occipital region. After a time repeated vomiting occurs, with relief which may be immediate or gradual. The whole paroxysm lasts from five hours to two or even three days, and is often accompanied with intense intolerance to light and sound and distinct hysterical manifestation. In some cases there is aphasia during the height of the paroxysm; vomiting may be absent.

An attack of migraine may be ushered in by an aura, which suggests that of epilepsy, although in most cases it takes the form of a disturbance of special sense. Rarely a peculiar bitter or very disagreeable taste, or, it may be, a peculiar odor, like that of osmic acid, marks the coming on of a paroxysm. The auditory prodrome, which is extremely rare, has been variously described as like the sound emitted from a marine shell applied to the ear, or as a gurgling similar to that which is heard when water enters the ear during bathing.

Of the special-sense auras the most important is the peculiar visual disturbance which has given rise to the special name of *ophthalmic migraine* (*hemiopia periodica*). The most frequent disorder of sight is an amblyopia, accompanied by vivid scintillations of light passing zigzag over the field of vision. Hemiopia, either monocular or binocular, sometimes lateral, sometimes vertical, may replace the amblyopia; or a central scotoma may be the chief phenomenon. Rarely during the attack these alterations of vision change one into the other, and still more rarely are they accompanied or replaced by distinct hallucinations. We have noted megalopsia.

An attack of migraine is usually attended with emotional depression, which may amount to a brief melancholy. O. Berger affirms that there is with the attack hyperæsthesia of the skin of the face, at least so far as the sense of locality and the electric senses are concerned, but there is commonly no hyperæsthesia to touch, and no nerve-tenderness even at the point of nerve-emergence. Firm pressure often gives relief.

DIAGNOSIS.—The diagnosis of migraine, usually easy from the history of the case, is to be confirmed by the exclusion of other causes of the attack and by a study of the family history.

PROGNOSIS.—Migraine is practically incurable, but abates after middle age, and is often much ameliorated by treatment.

TREATMENT.—The treatment of migraine consists primarily and chiefly in building up the general health of the patient. The higher the health the fewer the attacks. The most scrupulous care must be employed to search out all peripheral irritations, especially eye-strain, and to correct gout or other diathetic tendencies. The continuous administration of cannabis indica is often of great service in lessening the number and severity of the fits. A known extract should be given in ascending doses until it produces mild symptoms of intoxication, and then a dose just within the limit of the full physiological dose should be administered three times a day for months. Caffeine, antipyrin, and antifebrin are often useful in alleviating the pain in a migraine attack, and will in some persons even abort a paroxysm. Guarana, which has been much employed, depends for its activity upon caffeine. Of all palliatives the most certain is the combination of deodorized tincture of opium with potassium bromide (twenty minims to sixty grains). Unlike opium given by itself, this mixture rarely causes after-nausea and depression. The danger of forming the narcotic habit is never to be lost sight of in a disease so chronic as migraine.

SLEEP, ITS DISORDERS AND ACCIDENTS.

Sleep, stupor, and coma are not, as has been held by some, essentially diverse conditions, but are the outcomes of different degrees of completeness in the suspension of the function of the cerebral cortex, and are not to be separated by any fixed lines, every gradation being found in the sick-room between the lightest slumber and the deepest coma. Yet for discussion we may arbitrarily divide suspensions of consciousness into *sleep*, that condition of unconsciousness in which the subject is readily aroused, and when aroused is easily kept awake by ordinary external stimulations or by his will-power; *stupor*, that condition in which the subject is aroused with great difficulty and when left to himself relapses into unconsciousness; and *coma*, that state in which it is impossible by external irritation to restore consciousness.

For the purposes of treatment it seems essential to have a proper physiological understanding of the causes and nature of these grades of unconsciousness. Certain neurologists hold that the lapses from consciousness are due to changes in the circulation; but no proof of this has ever been given. It is true that during sleep there is more or less pronounced cerebral anæmia, which on awaking is replaced by turgescence of the cerebral vessels. It is a universal law that cessation of functional activity is immediately followed by lessening in the amount of blood in the part. We conceive, therefore, that the sleep or cessation of functional activity is the cause of the bloodlessness, and not the bloodlessness the cause of the sleep. Insomnia may be connected either with excessive anæmia or with excessive congestion of the cerebral cortex. One theory of the mechanism of sleep is that the latter is due to the formation by the processes of life during the waking moments of some poison which acts upon the cerebral cortex. This theory, again, is a purely speculative one. At present it is best simply to consider that it is a function of the cortical brain-cells when exhausted to pass into a condition of inactivity, during which their power of further effort is recuperated.

The disorders of sleep may be divided for our present purposes into —first, abnormal wakefulness; second, abnormal somnolence or morbid sleep; third, accidents or groups of symptoms which occur during sleep and which are not elsewhere spoken of in this book.

Abnormal Wakefulness.—In some cases of insomnia the subject is simply unable when bedtime comes to go to sleep; in other cases he goes to sleep readily, but wakes in two or three hours and is unable to slumber again. The latter form of insomnia is rarely the precursor of severe mental affections, but is often very obstinate.

As insomnia may exist as a prodrome, or as a symptom, of a general or local organic brain-disease, and as it also may be produced by diseases of various organs other than the cerebrum, and as, further, even in its most aggravated form, it may be the chief symptom of a cerebral disturbance and have no connection with any organic disease, it is essential in every case of insomnia that a very careful study be made of the whole individual, in order that the cause of the sleeplessness may be discovered, or else that by exclusion the case may be diagnosed as one of simple functional disorder. Insomnia not only produces great suffering, but in itself may lead to severe mental disease, so that its treatment is a matter of great importance.

In the treatment of insomnia it is essential to remove the cause: if any organic disease be found in the brain or elsewhere, its careful treatment is imperative. A frequent cause of simple insomnia is lithæmia; where this is the case relief may often be obtained by the use of the salicylates.

Insomnia produced by an active determination of blood to the head, that is, by active cerebral hyperæmia, is certainly rare except as a precursor of acute periencephalitis or other severe organic brain-disease. It may, however, develop as the result of excessive mental labor. Its nature is to be recognized by the fact that it accompanies an increased power of work, and often an exhilaration of spirits; the subject being able to go on night after night and produce work in quantity and quality above his normal powers. Such insomnia is always a very dangerous condition, and should be immediately treated by the cessation of work, by local blood-letting, by counter-irritation, and by full doses of sedative narcotics. The bromides are useful; hyoscine hydrobromate is a specific. Unless with the local hyperæmia there is a condition of general loss of power, the combination of aconite with the other remedies may act most happily. Sometimes in the insomnia of acute congestion sleep may be procured by giving the patient at bedtime a mustard foot-bath; a much more efficient procedure is to direct the patient to sit in a bath of very hot water and have a cold douche on the head from three to five minutes.

When insomnia is connected with local or general neurasthenia, rest-treatment modified to the individual case may be of great service. It would appear that in the majority of cases of ordinary simple insomnia there is a basal exhaustion; at least, it is not infrequent that food taken at bedtime, or when the patient wakes sleepless in the middle of the night, has a very beneficial effect. Bouillon, thickened with sago or lentil flour, oyster-soup, hot milk-punch, Mellin's Food, any easily digested hot food, may be used. Brandy or whiskey in a little hot water, strong ales, and other alcoholic drinks in some cases are very efficient. A half-ounce of whiskey or a single glass of very hot water, taken on awaking in the middle of the night, will often cause the wakeful subject to go to sleep within five minutes.

In the regulation of the habits of a person suffering from insomnia, careful study must be made not only of the general nature of the case, but also of the idiosyncrasies of the patient. Thus, in some persons massage before the time of sleep has a distinctly quieting influence, whilst upon others it acts as an excitant. Tire very frequently causes wakeful nights, but there are some cases in which exercise brings sleep. In all cases of insomnia it is essential that intellectual activity and emotional excitement during the latter third of the day be avoided; that the supper taken be light; that the patient sleep by himself or herself in a well-ventilated apartment, and that no caffeinic drinks be used after the morning meal.

In the treatment of insomnia narcotic drugs are to be avoided as far as possible, except in acute cases, when, if the symptoms have developed as the result of a sudden emotion or other strain, it is sometimes possible by exerting a strong influence for a few weeks to break up the habit of insomnia and then gradually to withdraw the remedy. If the insomnia be so severe that hypnotic drugs become necessary, two rules must be observed in their administration: first, always to use the least powerful narcotic which will achieve the desired object; second, not to use a single narcotic (unless it be a bromide) continuously for more than from one to two weeks, so as to prevent as far as may be the system from becoming accustomed to it, and also to avoid the danger of chronic poisoning by it. Opium is the worst of the whole class. The bromides are the least injurious, but rarely suffice, although they are very useful in combination, and often lessen the amount required of more powerful remedies. Hyoscine never produces chronic poisoning, nor, so far as we know, the hyoscine habit, and when it suits the individual case is, therefore, valuable. It should be given at bedtime. Sulphonal is one of the least harmful of all the narcotics, provided that its administration be not kept up too long. When there is any brain weakness, as in very old or paretic patients, it may produce mental confusion or continuing drowsiness the day after its administration. A number of deaths have been produced by a sudden outburst of poisoning when sulphonal has been administered for several months. It is so insoluble that the compressed pill readily passes through the intestines without change, and is therefore best administered in the form of a powder, given from one to three hours before the allotted time of sleep, in hot milk, or, if this be not obtainable, in hot water. Chloral still remains the most efficient of the hypnotics. It must be given in solution, well diluted, and, as it acts immediately, not until the subject is in bed. Its use may be continued for a length of time. Poisoning by it differs from that by sulphonal in that the symptoms come on gradually and abate upon the withdrawal of the drug. Chloralamide acts very much as does chloral, but is more uncertain. Trional is a useful hypnotic, fairly active and certain in its influences, and in many cases efficient without producing unpleasant symptoms. When continuously pushed it sometimes seems to cause general weakness. The relative doses of these narcotics may be considered to be: hyoscine, one one-hundred-and-twentieth of a grain; sulphonal, fifteen grains; chloral, ten grains; chloralamide, fifteen grains; trional, twelve grains. In obstinate cases the best results are obtained by a combination of chloral with hyoscine or morphine: in obedience to the law of crossed action of drugs, by using such a combination the practitioner obtains a double effect where the two lines of drug-influence come together, which in the cases just mentioned is the cerebral cortex. Urethan is very uncertain in its action. Paraldehyde is so disagreeable and irritating to most stomachs that it usually cannot be borne, but in rare cases it acts kindly.

Morbid Sleep.—Before discussing the more ordinary causes of morbid sleep it is necessary to speak of *nelavan*, African hypnosis or African sleeping disease, which is probably an acute fever, and is certainly often fatal. It is endemic on the west coast of Africa, but has occurred epidemically in the West India Islands. It attacks the negroes especially, but not solely. In most cases it comes on gradually, but it may begin brusquely. There is at first a slight frontal headache, with a sense of constriction in the forehead, attended by a mild fever. The vision may at this period be disordered. The gait becomes irregular, and not very infrequently there is a distinct ataxia. Even during the first hours of the headache an intense desire for sleep is manifested. The strength fails, the spirits are depressed, and there is some fever, but usually neither diarrhœa nor constipation develops, and the forces of the circulation are well maintained. Sleepiness in a short time deepens into a somnolence which becomes more and more intense and ends in a profound coma, which may pass quietly into death. Violent convulsions and sloughing bed-sores are liable to develop. There is no pathognomonic post-mortem lesion, unless it be swelling of the glands, and there is no known specific treatment.

Omitting toxæmic somnolence, most of the cases of morbid sleep seem to be referable to one of five groups:

Group 1. Sleep due to reflex irritations.
Group 2. Narcolepsy, or idiopathic sleep of unknown cause.
Group 3. Hysterical and epileptic sleep.
Group 4. Sleep of insanity.
Group 5. Somnolence connected with organic brain-disease.

Of the third, fourth, and fifth of these groups sufficient has already been said. Reflex sleep is very rare, but Katerbau has recorded a case in which a seventeen-year-old Jewess, who had slept four days and nights, immediately awoke after the passage from the rectum of a knot containing twenty-four round worms, whilst Mayer has related a similar case of a boy nine years old.

Under *narcolepsy* are grouped cases of morbid sleep, probably representing different affections, whose causation and nature are so obscure that at present no explanation of them can be offered. The best that can be done is to separate the cases into three subgroups, which are, however, connected by intermediate cases.

In the first of these groups belong instances of perpetual drowsiness, in which the subject habitually falls asleep at the slightest provocation, and whenever awake is sleepy, or in which there come daily paroxysms of overpowering drowsiness relieved by a long nap. It is, perhaps, justifiable to consider these cases as instances of excessive development of the normal sleep function of the brain.

In the second class of cases the normal relations between sleep and wakefulness are so altered that the two conditions, instead of alternating every twenty-four hours, alternate at long intervals. Thus, in the recorded case of a Jewess, the average length of the sleeping and waking periods was five and a half days, the maximum was seven days, and the intervals of wakefulness were broken only by short restless bits of slumber.

The third class of cases is that in which the sleep comes on without apparent cause and becomes more and more profound until the patient dies. Such a paroxysm suggests brain-congestion, and some cases have yielded to a very free venesection. That, however, fatal sleep without determinate cause may occur is shown by the case reported by Dr. S. Weir Mitchell, in which, after a prolonged seemingly causeless sleep ending in death, a most careful post-mortem examination failed to detect any lesion.

Accidents of Sleep.—*Sense-shock* is an aura-like sensation, rising from the feet or hands at the moment of waking, and passing upward to the head, where it disappears in the sense of a blow or shock, or of a bursting, with in some cases a subjective special-sense disturbance, such as a loud noise, a strong odor, a vivid flash of light. Such paroxysms may also occur during the daytime; they have no serious significance, and require no further treatment than the upbuilding of the strength of the patient.

Night palsy is a sense of numbness in one or more extremities of the body, usually felt on waking, but occasionally occurring in the daytime. It may be monoplegic, hemiplegic, or general over the whole body. S. Weir Mitchell speaks of it as preceding locomotor ataxia, but it is very common in hysterical women about the climacteric, and also occurs in gouty subjects. It has no special significance, and is certainly not indicative of failure of circulation or of organic nervous disease.

Somnambulism is a condition in which a dream so takes possession of the sleeper that he rises, walks, and acts. Movements and muttered words are very common evidences of dreaming in a sleeper, and every grade between the slightest dream-movements and the most active sleep-walking exists. If the somnambulist be approached, his eyes will be found to be closed, or, if open, they, with the rest of the face, are impassive and without expression, paying no attention to the brightest lights, and appearing to have no power of sight in them ; yet obstacles are avoided, narrow places passed through, feats of balancing performed, and numerous complicated movements made so perfectly that the by-stander can hardly persuade himself that the sleeper is not awake. When seized, the somnambulist usually resists with vigor. Left to himself, after wandering for a greater or less length of time he returns to his bed, covers himself up, and sinks into the quiet forgetfulness of normal sleep.

As it is often possible to direct a dream by answering the questions of the dreamer, so in somnambulism the thoughts of the sleeper can often be turned, and in obedience to a firm command he will return to bed without waking ; often, however, he is uncontrollable except by physical force. Acts the most difficult and complicated, crimes of various character, and murder even, have been performed by the somnambulist in response to his dream impulses.

The so-called *night-terrors* of childhood are a form of somnambulism, or are, in rare cases, epileptoid seizures. Nothing is more common than for a young child to go in the night to its parent's bed, trembling with terror or weeping bitterly, with the statement that it has had a bad dream. Such a dream may be so vivid as completely to enchain the attention, and if at the same time there be outward manifestations of the overpowering emotions from which the child is suffering, a paroxysm of night-terror results. Very frequently during the paroxysm the child shows terror of some one object,—a cat, a dog, an elephant, a monster of some kind, as is indicated by its semi-coherent cries. In a large majority of cases night-terrors are of no more serious import than an attack of somnambulism. They often depend upon gastric irritation or too much emotional excitement during the day. In a few recorded cases the cause of the attacks has been intestinal worms. Those rare night-terrors which are due to serious disease can be distinguished only by their tendency to recur continually and by their concomitant symptoms. We have seen one case in which the night-terror in an adult, repeated at intervals during every night, was not affected by innumerable treatments instituted by various physicians of the highest class, and finally absolutely destroyed the usefulness of the subject's life. Again, we have seen the re-enaction of an escape from drowning recur night after night until it produced a most serious condition. In the treatment of night-terrors of children, especial care must be taken to remove intestinal worms, glandular swellings, or any other possible source of local irritation.

Especially must the digestive system be put in complete order, and the use of stimulating foods and of caffeinic drinks positively forbidden. Any further general treatment of the night-terrors is that of neurasthenia and hysteria; in the very acute sudden cases it may be necessary not only to put the patient fully under the influence of the bromides, but also to use narcotics at bedtime, so as at once to break up the habit, which if left to itself may become fixed and irremediable.

CORRELATED DISORDERS OF MEMORY AND UNCONSCIOUSNESS.

Every functional act in a nerve-cell is accompanied by nutritive change, and this nutritive change, although recovered from, leaves behind it a residue of effect. Hence all nerve-tissue has memory,—that is, the faculty of being permanently impressed by temporarily acting stimuli; the thing remembered being, in fact, the functional excitement. In this is found the explanation of the results of training; and in it, also, is found the explanation of the tendency of various functional nerve-diseases or conditions to perpetuate themselves after the removal of their original cause. It is in this way that an accidental becomes a permanent epilepsy, that a physical habit becomes a habit-chorea. It is in this manner that the hysterical woman who gives way to an hysterical nervous impulse strengthens the hold of such impulse upon her nervous system until it may become irresistible.

Moral habits are formed in obedience to the same law. Self-control, enforced at first by discipline, may become at last in the child an integral function of the nervous centre by a method parallel to that by which an accidental epilepsy is converted into a permanent disease. In the prognosis and treatment of disease, as well as in the training of the young, the full recognition of the power of habit—*i.e.*, of unconscious memory—is a matter of vital importance.

What is true of the lower nerve-centres and fibres is true of the upper ones. Intellectual acts or thoughts and perceptions tend to stamp themselves upon the centres connected with them, and when the function of the nerve-cell is connected with consciousness the changes which occur in the nutrition give origin to conscious memory,—*i.e.*, to memory in the usual sense of the term.

These things being so, it must be that intellectual memory shall exist in as many varieties as there are intellectual actions. Disease sometimes dissects out, as it were, these different forms of memory. Thus, we have loss of word-memory, aphasia. In a case of dementia under our care, whilst memory for ordinary events was almost entirely lost, a joke or a ludicrous story would be remembered in all its details without effort. Again, the power of receiving new impressions is essentially different from that of preserving impressions which have been received; and so in senile or other brain-degeneration it is very common for the power of remembering recent occurrences to be lost, although the recollection of events which happened in childhood days is more vivid than the normal condition of the individual would warrant. In examining, therefore, for the detection of failure of memory the physician should question the patient as to the simple happenings of the past twenty-four hours, and not be misled by the vividness of the recollections of the long past. The power of receiving impulses is usually lost before the power of recalling impulses.

Exaltation of memory—that is, exaggeration of the power of receiving new impulses or acquiring new thoughts—is very rare, but does occur in certain conditions of exalted function in the cerebral cortex, as in the insomnia of acute hyperæmia or in the beginning of an acute periencephalitis.

Memory and consciousness are so united that they are often confounded as one thing; nevertheless they are distinct, and the link which binds them may be broken by disease. In this way arise certain cases of automatism, as that of the French soldier who, as the result of a wound in the head, was subject to attacks, lasting many hours, in which he gave no response to ordinary external stimuli and had no appearance of consciousness, but, if put in the position of marching or writing or smoking or what not, would go through the whole completed series of movements necessary for the performance of these acts, changing abruptly from one performance to another if taken hold of and put in a new position.

The sense of *personal identity* is dependent upon the existence of memory and consciousness. The unbroken chain of events recorded from an indefinite past correlated with the consciousness of the present gives the realization of the unity of the present with the past. This sense of personal identity is destroyed by a complete loss of memory, which loss may be abrupt and be unaccompanied by impairment of consciousness or of rationality.

Double personality, the condition in which the subject feels as if he were two distinct personalities, the one alternating continually with the other, or more commonly the two coexisting, has no connection with loss of personal identity nor yet with double consciousness. Its explanation is very difficult: it is occasionally seen as the result of hasheesh or other poisoning, and also in insanity, in which affection it may become the basis of a delusion, as in the case of a patient of my own who was overwhelmed by the constant doubt whether he was himself or his own double.

Double consciousness, so called,—*periodical failure of memory*, or *periodic amnesia*,—is a disorder of memory which also involves all the intellectual functions and the character of the individual. In a typical case there is, first, an abrupt loss of memory at the beginning of each paroxysm for everything that has happened during paroxysms not of the same series; second, a change in the personal character of the individual, the disposition, the habits of thought, and even the intellectual powers, being altered.

After a time the subject goes abruptly back to the first condition, and so leads a double life of alternating states.

Double consciousness is allied to epileptic automatism, which indeed may be considered a form of double consciousness, and also to certain conditions of insanity, in which the attacks come and go without apparent reason.

NEURALGIA.

Definition.—A violent pain following the course of a nerve-trunk and connected with no known disease, and for which we can give no adequate explanation.

Etiology.—Owing to the curious feeling of "knowing all about it" which the average human individual derives from a name, in practical life it is often necessary to have a name which shall answer as a cloak for professional ignorance. The word "neuralgia" means a nerve-pain, which is in fact no meaning, as all pains are nerve-pains; but almost from time immemorial the term has been used to signify a class of cases in which violent pains occur in nerve-distribution from causes which cannot be made out. As knowledge has increased there has been a great reduction in the number of cases of neuralgia; because most of the cases which were formerly said to be instances of neuralgia now are known to be examples of neuritis, organic disease of the nerve-centres, etc.

A form of pain which is still often spoken of as neuralgia is the reflex pain, in which the sensation is due to some distant irritation; as examples may be cited the violent pain in the angle of the jaw occasionally produced by eating very cold ice-cream or by other gastric irritation. Anstie has described various cases of what he termed neuralgia in the urethra and testicles, due to the irritation of self-abuse. A trigeminal pain may be due to a neuritis propagated from a diseased tooth-pulp, or it may be reflex, as is proved by its occurring in the side of the head opposite the affected tooth. Impacted fæces, and fissure of the anus, frequently produce so-called intestinal neuralgia and cystic neuralgia. We have seen as the chief symptom produced by a tape-worm a mastoid pain so violent as to lead to the diagnosis of disease of the bone and to operation. Ferrier has recorded instances of cervico-brachial neuralgia resulting from a diseased tooth. The pain of hepatic irritation is very commonly referred to the shoulder, and we have seen as the chief symptom of gastric perforation, with escape of a meal into the abdomen, a mortal agony referred to the root of the neck. Reflex pains should never be spoken of as neuralgias, but as reflex pains. Their relief depends upon the acuteness of the medical attendant in discovering the cause of the irritation and his success in removing it.

Neuralgias are frequently spoken of as being produced by lead and other poisons. Such neuralgias are in great part, probably always, instances of neuritis. In a very large proportion of the cases which are still diagnosed by practitioners as neuralgic, gouty disorder of metabolism is the cause of the suffering. Functional pains, or rather pains from disordered functions, without organic lesions in the nerves or elsewhere, may be the result of anæmia or of a peculiar exhaustion. To such cases the term neuralgia may be well applied.

There is, however, a final group of cases in which neither during life nor after death are we able to detect any cause for the pain. It would seem that there may be a molecular change either in the sensory nerve-centres or in the nerves themselves so fine as to escape our instruments, which predisposes the individual to suffer, so that a change of weather or other untoward influence too slight to be felt by the normal man causes a pain-storm. There appears to be a distinct general condition which may be known as the neuralgic temperament or diathesis. This is often inherited, but may be developed by prolonged bodily exhaustion or other causes. When once acquired it may persist although the original cause has been removed. The pains which come to some persons in malarial anæmia probably are often neuralgic, but when the anæmia has been relieved, if the nervous system has been sufficiently long impressed, the pain-tendency becomes stamped upon it, precisely as the epileptic tendency becomes constitutional in a case of reflex epilepsy and persists after the removal of the original irritation. These neuralgic pains are to be recognized by their persistency, by the absence of cause, and by the excluding of all other sources of pain. The acquired or inherited neuralgic temperament is closely connected with the migraine heredity, as well as with inherited gout; but exactly what the relations between the three conditions are is still obscure. The neuralgic diathesis corresponds with the general neurotic temperament, is more frequent in women than in men, and is often prevalent in dry neurotic climates and in persons free from gouty symptoms. The neuralgic temperament is further allied to hysteria and to neurasthenia, and especially to the neurasthenia which follows severe traumatisms. The nervous headaches spoken of elsewhere (see page 457) should perhaps be classed as neuralgic.

DIAGNOSIS.—The diagnosis of neuralgia is to be reached chiefly by exclusion. When no proper cause can be found for a pain in a nerve-trunk, such pain may be spoken of as a neuralgia. A great majority of cases of so-called neuralgia are really instances of gouty neuritis. Diseases of nerve-centres produce pains which in their character are indistinguishable from true neuralgia except by their greater severity and persistency. Whenever a neuralgia is persistent in a definite nerve territory a centric lesion should be suspected, unless there be a gouty diathesis. The pains of posterior spinal sclerosis may closely simulate neuralgia, and if they precede the development of the more characteristic symptoms may lead to mistaken diagnosis. The occurrence of persistent neuralgic pains in the legs, associated with a single symptom of a spinal sclerosis, such as Argyll-Robertson pupil or loss of the knee-jerk, will afford sufficient ground for a probable diagnosis of the centric disorder.

In the diagnosis of obscure cases of nerve-pain the greatest care should be exercised in searching for a possible distant point of irritation which may produce reflex pain.

TREATMENT.—The treatment of nerve-pain depending upon gout, malaria, organic nerve-disease, etc., is essentially the treatment of the underlying condition. The treatment of ordinary neuralgia, so called, is usually that of a rheumatic neuritis. The treatment of a true neuralgia must have for its basis also the relief of the underlying bodily state. Commonly there is vital depression, often with lowered nutrition, sometimes with anæmia. This state must be met. Thus, if there be failure in the development of fat, cod-liver oil may be of great service; if there be anæmia, iron may be employed, etc. Always, however, great care should be taken not to interfere with digestion by drugs.

In true neuralgia electricity is of no value. Over a neuralgic nerve-storm, such as migraine, or the paroxysms of pain which are said sometimes to replace epilepsy, electricity has no power. In the pain of a neuritis it often brings relief. Local anodynes and mild counter-irritations are sometimes of service in neuralgia. Antipyrin, phenacetin, and other allied coal-tar products will very often control the pain. Whenever it is possible, complete change of scene, with out-door life, should be tried if other measures have failed.

CHAPTER III.

ORGANIC DISEASES OF THE BRAIN AND ITS MEMBRANES.

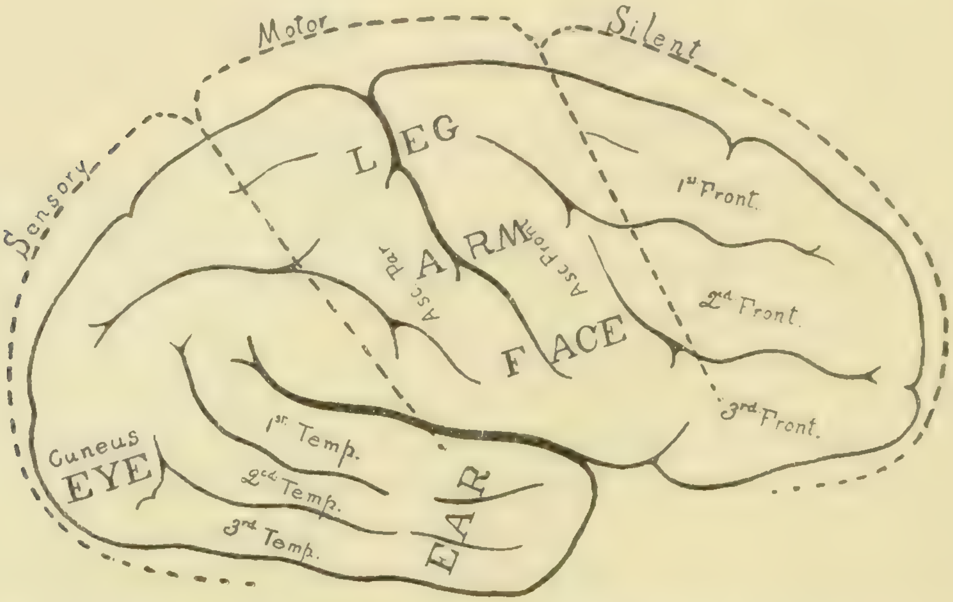
CEREBRAL LOCALIZATION.

THE term cerebral localization is used as a name for the applied science which has grown out of the recognition of the fact that, in the processes of functional evolution and differentiation in the animal kingdom, the human brain has become so specialized that certain functions have been permanently assigned to certain portions of it, so that it is possible to recognize the anatomical seat of a lesion by noting the disturbances of function. In applying our knowledge for the purposes of diagnosis it is essential to remember that it is the seat and not the nature of the lesion which it is the purpose of cerebral localization to discover. A tumor in a certain spot in the brain produces the same disablement whether it be cancerous, tubercular, or of other nature. We can only note that purely destructive lesions paralyze function; that irritative lesions at the same time excite and pervert function; and that many lesions are both destructive and irritative. Thus, a destructive lesion of the portion of the brain connected with vision causes blindness, an irritative lesion causes active disturbance of vision, such as flashes of light: again, a destructive lesion of the motor region of the brain causes paralysis, an irritative lesion causes spasm; whilst a lesion which is both destructive and irritative causes paralysis with spasm which is apt to take the permanent form known as contractures. It may be further noted that the symptoms of irritation are more apt to be produced when the lesion affects the nerve-centres themselves than when the conducting fibres from those centres are attacked.

We have no definite information as to the seat in the human brain of consciousness and the intellectual faculties, or whether, indeed, these functions are confined to certain limited regions or are generalized throughout the whole brain-structure. It is commonly supposed that the frontal lobes of the brain have some especial relation to these functions, but certain it is that a gross lesion of an anterior lobe may exist without registering itself by any characteristic symptom. The function of motion is situated in the region of the cerebral cortex lying about the Rolandic fissure, whilst that pertaining to the special senses is situated in the occipital, temporal, and olfactory lobes: so that we may naturally divide the brain-cortex into the silent, the motor, and the sensory area, as in the diagram on the following page.

In the diagram no notice of speech-centres is taken, because human speech is so complicated in its physiology and so widely distributed in the anatomical location of its centres that it demands the elaborate discussion given at the close of this article under the head of Aphasia.

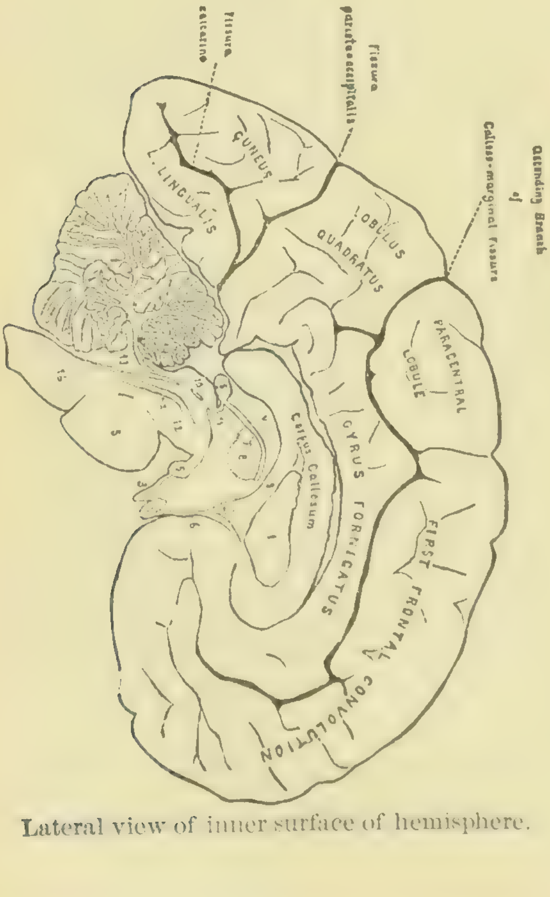
FIG. 3.



External lateral view of brain, showing general arrangement of cerebral centres.

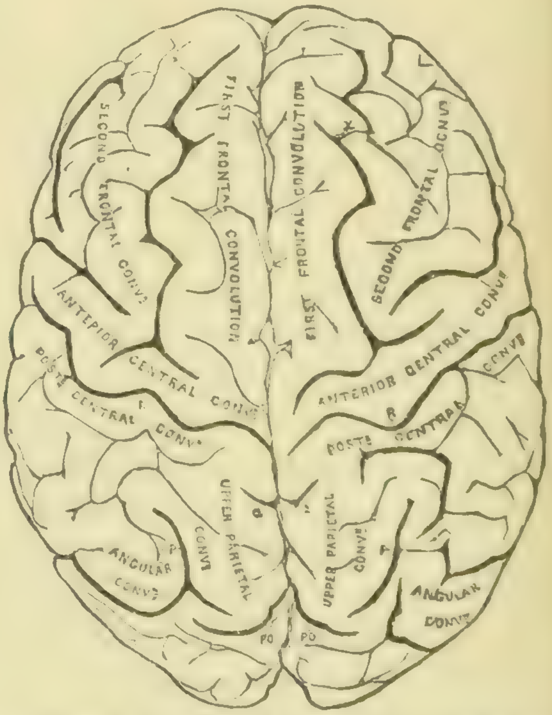
Active nerve-centres always have efferent nerve-fibres forming paths along which run the impulses originating in the ganglionic nerve-cells; from which it follows that in discussing the localization of a brain-func-

FIG. 4.



Lateral view of inner surface of hemisphere.

FIG. 5.



View of brain from above. The anterior and posterior central convolutions are respectively the ascending frontal and ascending parietal convolutions of the nomenclature used in the text.

tion it is first in order to speak of the centres implicated, and then of the nerve-fibres.

FIG. 6.

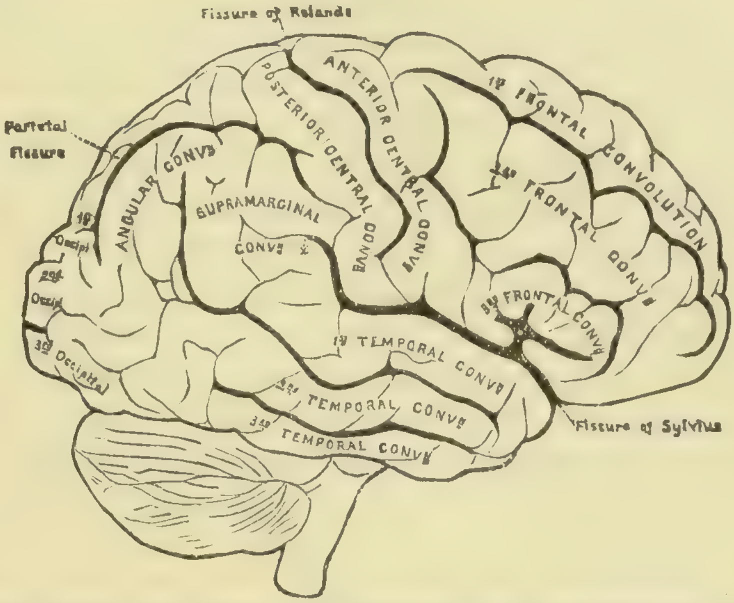


Diagram showing cerebral convolutions. The anterior central convolution and the posterior central convolution are respectively the ascending frontal and the ascending parietal convolution of the text.

MOTION.

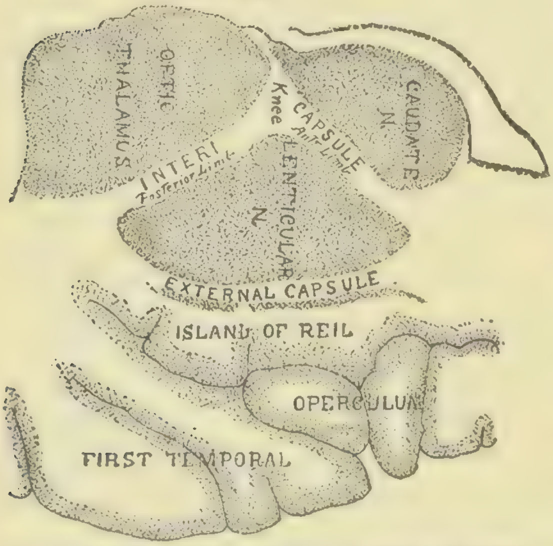
The motor, or, as it is better termed, the psycho-motor area of the brain is the part where thought originates the impulses which finally end in muscular contraction. It comprises the ascending frontal and parietal convolutions, including the upper portion, which is seen on the median surface of the cerebrum, and constitutes the so-called paracentral lobule. The gross arrangement of the centres is shown in the diagram (Fig. 3). It must be understood that these centres are not sharply defined from one another, but overlap. The highest part of the region is occupied by the leg-centres, which certainly also reach into the paracentral lobule. The arm-centres, which occupy the middle third of these convolutions, probably extend higher in the ascending frontal than in the ascending parietal. The lower third of the ascending frontal convolution is the chief centre for the face movement, but there is reason for believing that the corresponding part of the ascending parietal convolution is also involved. The centre of the movements of the angles of the mouth lies at the upper junction of the lower ends of the two convolutions, or, in other words, just below the lower end of the fissure of Rolando; whilst the lips and tongue receive their innervation from the foot of the ascending frontal convolution, the centres probably also extending into the third frontal convolution.

Horsley and Schäfer have found that in the monkey the posterior half of the upper and lower frontal convolutions and the inner aspect of the summit of the ascending frontal convolution (a portion of the paracentral lobule) are respectively connected with the lateral movements of the head and eyes and with the movements of the trunk or body; and it is probable that in man this connection holds. There is reason for believing (see Hemiplegia) that in muscles which absolutely work together so closely as those of the trunk each muscle receives innervation from each side of the brain, a theory which is borne out by the fact that destruction of one of these centres fails to produce permanent paralysis.

The motor fibres* which arise from the psycho-motor area of the brain are at first widely separated, but converge as they pass inward through the centrum ovale, and when they reach the internal capsule are condensed into a very narrow territory. The pathway to the inner capsule is double, one mass of fibres passing between the caudate and the lenticular nuclei, and the other between the lenticular nuclei and the optic thalamus. In the passage of these fibres the course of their direction is changed from vertical to horizontal. The fibres connected with the face are originally the lowest in the cortex, so that their pathway through the centrum ovale lies below that of the fibres connected with the extremities. In the change of their course from the vertical to the horizontal direction, however, they get in front, so that their pathway is through the bend or angle of the internal capsule.

In the internal capsule the fibres from the facial centres are thus placed anteriorly near the so-called knee; next to them come the fibres from the arm-centres; then the leg-fibres, the boundaries between the bands not being sharply defined. Together these motor tracts form about two-thirds of the posterior limb of the internal capsule, the hinder third of the limb being occupied by the sensory pathway. From the internal capsule the motor fibres pass into the crus cerebri, where they occupy the middle two-fifths of the crusta, extending from the surface below almost to the substantia nigra.

FIG. 7.



Transverse horizontal section of brain, showing internal capsule and its relations.

In the crus cerebri the facial nerves begin to leave the main body of fibres, the oculo-motor nerve emerging from the inner surface of the crura. Most of the fibres, however, of the cranial nerves enter the pons, but very shortly after so doing they separate from the band of fibres which go to the body and extremities, and cross over to the opposite side of the pons to enter their various nuclei. The main portion of the motor fibres enters the pons and passes through the crustal portion lying between the superficial and deep layers of the transverse fibres and surrounded by gray matter, with which, however, they have no connection. In this passage through the pons the mass of fibres is broken up into bundles, which a little lower down are again gathered into one string, as which they enter the medulla. In the medulla this string divides, the greater part of it passing over to the opposite side to be continued down the spinal cord as the lateral or crossed pyramidal tract, whilst the smaller portion, failing to decussate, enters the spinal cord upon the side corresponding to that of the cerebral hemisphere in which it originated, to constitute the anterior or direct pyramidal tract. During their passage from the cerebral cortical centres to the spinal cord these white or conducting nerve-fibres are distinct, and apparently without connection with ganglionic cells.

---

* The idea of the construction of the brain and spinal cord in their motor functions is—ganglion-cells in the cerebral cortex, which originate an impulse that passes

In the neighborhood of the internal capsule are the great cerebral ganglia, namely, the optic thalamus and the caudate and lenticular nuclei (corpus striatum). The function of these centres is unknown, the old view that they were connected with the motor fibres going from the cortex having apparently been disproved. The thalamus has close connection with the optic tract and also with the cerebral cortex. The corpus striatum appears to have special functional relations with the cerebellum, with which it has anatomical connections, and by whose development it is influenced, since failure in the development of the cerebellum is always accompanied by a great reduction in the size of the corpus striatum.

In studying the sensory brain-functions the first natural division is that of General Sensation, including the touch, thermic, muscular, and pain senses, and the so-called Special Senses.

SENSATION.

General Sensibility.—We have no knowledge whatever of the localization of the different forms of so-called general sensibility; indeed, the position of the cortical centres which preside over the sense of touch itself has not yet been definitely determined. The only fixed anatomico-physiological point that we have is that the fibres which convey sensory impulses to the perceptive centres in the brain-cortex pass through the posterior third of the internal capsule. According to Flechsig, these fibres pass upward from the capsule towards the cortex through the region which lies beneath the parietal bone,—that is, to the region of the psycho-motor convolutions and the parietal lobe. As the result of ex-

---

down nerve-fibres and exerts an influence upon cells in the spinal ganglia that causes them to discharge force, which in turn produces contraction of the muscles. It will be seen, therefore, that physiologically the spinal system must be considered to commence in the crus cerebri, for here is the first pair of ganglia (that of the oculomotor nerve) which have the power of directly causing muscular movement. The spinal system is then continued through the pons, where certain of the nerves of the head have their nuclei, and through the medulla into the cord itself.

periments upon monkeys, Horsley and Schäfer, however, locate the centres for painful and tactile sensations in the gyrus fornicatus; Ferrier found them in the gyrus hippocampi. Apparently, therefore, the sense of sensation is located in the whole of the falciform lobe. Cases confirming this have been reported by Thomas Savill, who thinks that the posterior part of the gyrus fornicatus is the cerebral centre for tactile sensation in the arm; nevertheless, the view of Flechsig seems to be better borne out by the scanty pathological data which have been accumulated, as in various cases lesions of the parietal region have produced hemianæsthesia, whilst not rarely in legs or arms paralyzed by lesions in the psycho-motor cortex there is blunting of sensibility. The large collection of cases made by Dana indicates that it is especially the posterior half of the psycho-motor area that is concerned with sensation.*

The course of the sensory fibres below the inner capsule is still a matter of much doubt. It is known, however, that in the peduncles the fibres pass through the so-called tegmentum, *i.e.*, through the posterior or superior portions of the crus, separated more or less distinctly from the crusta or motor pathways by the ganglionic mass known as the locus niger, and then continue through the pons without decussation until they reach the spinal cord.

Hearing.—The cortical centre for the perception of auditory impulse appears to be in the posterior half of the first temporal convolution. The loss of hearing due to the destruction of this convolution has, however, been found, both in the lower animals and in man, not to be permanent. There is some reason for suspecting that the cortical auditory centres may be of wider range than that spoken of, and that outlying portions of these centres may take on superactivity when the central parts are destroyed; but it is more probable that each auditory nerve is connected with both hemispheres, although only the connection with the opposite hemisphere is habitually functionally active, and that therefore it is possible for the opposite convolution to assume the function of the destroyed portion. We have no knowledge of the pathway between the cortical auditory centres and the auditory nucleus in the medulla oblongata, except that this pathway passes through the posterior portion of the internal capsule. The auditory nerve is so exposed that in the great majority of cases nervous deafness is peripheral.

Smell.—There are indications from experiment and also from pathology that the centres of smell are in the front portion of the uncinate convolution, to which fibres from the olfactory nerve have been traced; but the subject is still involved in great obscurity.

Taste.—The sense of taste is so much affected by catarrh and other abnormal conditions of the mucous membranes of the mouth that care is sometimes necessary to avoid being led into mistakes of interpretation. Loss of taste, or *ageusia*, may be due to disease of the glosso-pharyngeal nerve, in which case it is confined to the side of the tongue supplied by the affected nerve. In a number of cases it has been produced by disease of the trigeminal nerve, but in other cases it is alleged that taste has been normal though this nerve was very markedly affected. Again, the extreme tip of the tongue seems to depend upon branches of the chorda tympani for the taste-function; hence disease involving the chorda tympani affects taste.

---

* Disturbances of sensibility are very commonly hysterical. Charcot was inclined to believe that loss of muscular sense is always so; but Ransom reported a case in which it followed a traumatism and was relieved by trephining, and Carter Gray one in which it was caused by a sarcomatous tumor in the parietal convolutions.

Loss of taste may be part of a general hemianæsthesia, so that it is probable that the fibres passing from the cortical receptive centres of taste run through the posterior part of the internal capsule; as to their further course we have no knowledge. There is much doubt as to the situation of the receptive centres in the cortex, but cases have been recorded in which tumors occupying the hippocampal region have apparently affected the sense of taste.

In testing any case for a loss of taste, three regions, the front of the tongue, the back of the tongue, and the palate, should be separately examined. The patient should be blindfolded, and quinine or salicin, diluted vinegar or lemon-juice and salt, or other highly sapid matter, employed. In order to prevent confusing smell with taste, the testing substances should be free from marked odor.

Parageusia, perversion of taste, and *hypergeusia*, excessive sensitiveness of taste, are usually of hysterical or psychical origin.

The relations of the eye to the detection of cerebral disease are very complicated: they pertain to the optic disk, to the pupil, and to the function of vision.

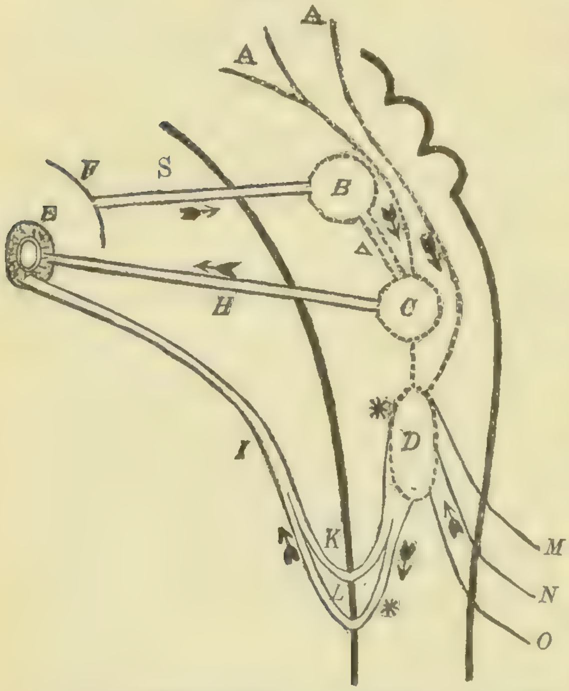
Optic Disk.—It is customary among ophthalmologists, following Von Graefe, to make three alterations of the optic disk connected with disease of the nervous system: the first of these is atrophy, which occurs in various sclerotic diseases of the brain and the spinal cord, or as a secondary result of choked disk; the second is the so-called choked disk; the third is descending neuritis. In *choked disk* there is a projection of the end of the nerve, which is œdematous and opaque, with its margins obliterated and its vessels swollen. In *descending neuritis* the disk is described as less swollen and more red, with tortuous veins and arteries. Practically, choked disk and descending neuritis may be looked upon as one thing, both having similar significance and each ending in atrophy.

The most probable explanation of choked disk is that it is the result of an excessive pressure upon all the lymphatic spaces of the brain, which manifests itself especially in the lymphatics of the optic disk, which are on the surface of a chamber and therefore lack the support of contiguous tissues. Theoretically, choked disk is dependent upon brain-tumor, abscess, meningitis, or other gross lesion which increases cerebral pressure, whilst descending neuritis is the outcome of a basilar meningitis. Practically, however, there is no difference in the significance of the two conditions.

A choked disk may be unilateral, in which case it is usually due to local disease, but may be rheumatic. In rare cases either of the affections under consideration may be the outcome of other causes than brain-tumor or inflammation; the more rapid the development of increased intra-cranial pressure the more positive the ocular manifestation; but when the increase of pressure is slowly developed the choked disk may be absent. It is wanting in about fifteen per cent. of cases of cerebral tumors.

Pupils.—Myosis and mydriasis are not localizing symptoms; inequality of the pupil may or may not be a localizing symptom. The pupil is subject to three forms of movements: first, that produced by light; second, the so-called cutaneous pupillary reflex, by virtue of which pinching the skin causes dilatation of the pupil; third, the changes which accompany accommodation. The Argyll-Robertson pupil is a condition in which no movement of the pupil is produced by pinching the skin or by throwing a bright light into the eye, although the relations between the pupil and the accommodation are preserved, dilatation of the pupil occurring when vision is suddenly taken from a near to a distant object.

Fig. 8.



A, nerve-fibres from the cerebrum; *B*, optic centre; *S*, optic nerve; *E*, pupil; *F*, retina; *H*, oculo-motor nerve; *C*, oculo-motor centre; *D*, ocular centres in the cervical spinal cord (cilio-spinal axis of Budge); *IK* and *IL*, sympathetic nerves; *M*, *N*, and *O*, sensory nerves. (After Erb.)

In the Argyll-Robertson pupil, which occurs chiefly in spinal sclerosis, the failure of the pupil to contract under the stimulus of light shows that there is a lesion in the arc *ECBF*, or, in other words, that either the optic nerve or its centre, or the connection between the optic centre and the oculo-motor centre, or the oculo-motor centre or its nerve, is diseased. The retention of normal vision shows that the optic nerve and its nucleus are perfect; the occurrence of movements during the process of accommodation proves that the oculo-motor nerve and its centre are active: the interruption in the arc *ECBF* must therefore be between the optic and the oculo-motor centre, or, in other words, in the commissural fibres which connect the optic and the oculo-motor centre. The loss of the cutaneous pupillary reflex proves that there is some interruption in the arc *MDL*, this interruption probably being in the spinal cord and due to the lesion which interrupts the continuity of the pathway between the oculo-motor and the optic centre.

Vision.—Amaurosis, or blindness, amblyopia, or partial blindness, hemianopsia, or seeing one-half of the field of vision, scotomata, or blind spots in the field of vision, and contraction of the field of vision, are the chief forms of visual deficiency. In studying the relation of these to cerebral localization it is necessary first to recognize that the visual nerve apparatus consists of perceptive centres situated in the cuneus (occipital lobe), and of conducting fibres which pass from these centres beneath the angular gyrus and through the posterior portion of the internal capsule down to the geniculate bodies and the superior quadrigeminal bodies, whence a flattened band of fibres goes across the upper anterior surface of the cerebral peduncle to the optic chiasm, which is situated upon the olivary eminence of the sphenoid. In the chiasm each nerve divides into two bands, of which the larger or outer band decussates with its fellow, or, in other words, crosses over to enter the optic nerve as it emerges from the opposite side of the chiasm and to be finally distributed to the nasal half of the retina. The inner or smaller band of fibres passes on through to the chiasm, without decussation, to the outer side of the retina of its own eye.

The diagram on page 480 shows diagrammatically the visual paths from the perceptive centres to the retina. It will be seen that each cuneus is connected with the half of each retina corresponding to itself in position,—the left cuneus with the left half of each retina, the right cuneus with the right half. Paralysis of one cuneus, therefore, must produce paralysis of the corresponding half of each retina, the so-called *homonymous hemianopsia*, or *homonymous lateral hemianopsia.* Paralysis of a portion of a cuneus would produce hemianopic scotomata.

Interference with the functions of one optic tract at any position between the cuneus and the commissure will produce the same symptom as does disease of the cuneus, homonymous lateral hemianopsia. A lesion of the optic chiasm will produce disturbances of vision varying according to its situation. If it press backward from the front of the chiasm (*T*.6, Fig. 9) it may produce hemianopsia of the opposite halves of the eyes, *heteronymous hemianopsia.* If it be placed laterally (*T*.3, Fig. 9) it may produce great inequality of vision of the two eyes. Homonymous hemianopsia is spoken of as temporal when both temporal fields are involved; as nasal when both nasal fields are affected. A paralysis of one optic nerve in front of the chiasm will produce amaurosis, or, if not complete, amblyopia, of the eye of the same side.

Horizontal hemianopsia, in which the vision is paralyzed in the upper or lower half of the eye, is almost always due to disease of the eye itself, but has been produced by a tumor pressing from above downward upon the optic chiasm.

Contractions of the field of vision, if not hysterical, are usually due to diseases of the optic nerve. They are not rare in locomotor ataxia and other forms of spinal sclerosis, in which atrophy of the optic nerve-fibres is very apt to occur, and they also accompany optic neuritis.

FIG. 9.

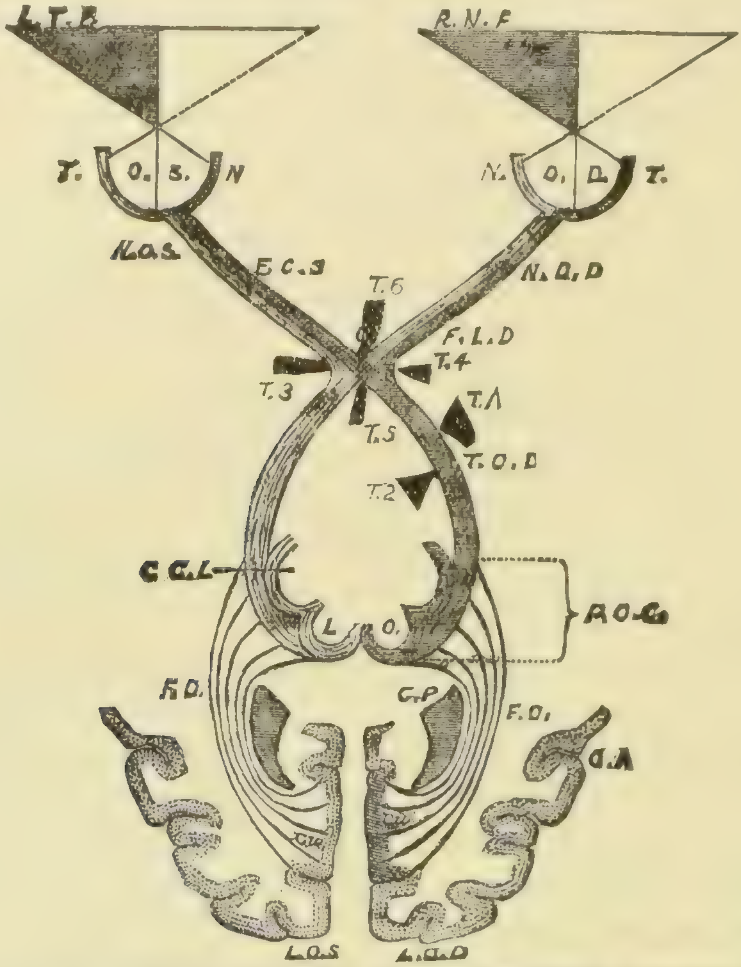


Diagram of visual paths, designed to illustrate specially left lateral hemianopsia from any lesion: *L.T.F.*, left temporal half-field; *R.N.F.*, right nasal half-field; *O.S.*, oculus sin.; *O.D.*, oculus dexter; *N.T.*, nasal and temporal halves of retinæ; *N.O.S.*, nervus opticus sin.; *N.O.D.*, nervus opticus dext.; *F.C.S.*, fasciculus cruciatus sin.; *F.L.D.*, fasciculus lateralis dext.: *C.*, chiasma, or decussation of fasciculi cruciati; *T.O.D.*, tractus opticus dext.; *C.G.L.*, corpus geniculatum laterale; *L.O.*, lobi optici (corpus quad.); *P.O.C.*, primary optic centres, including lobus opticus, corp. genic. lat., and pulvinar of one side; *F.O.*, fasciculus opticus (Gratiolet) in the internal capsule; *C.P.*, cornu posterior; *G.A.*, region of gyrus angularis; *L.O.S.*, lobus occip. sin.; *L.O.D.*, lobus occip. dext.; *Cu.*, cuneus and subjacent gyri constituting the cortical visual centre in man. *The heavy or shaded lines represent parts connected with the right halves of both retinæ.*

Scotomata may occupy any part of the visual field, and be due to disease of the retina. They may be the outcome of a very small cuneal lesion. When symmetrical and central they are in most cases the result of tobacco poisoning.*

---

* In tobacco poisoning the loss of vision is never complete. It is especially marked for the perception of colors, for green and red in particular; the former is usually described by the patient as "white" or "gray," and the latter as "brown" or "no color at all." In most cases the scotoma is smaller than the visual field for central colors, green and red, and hence a zone is present beyond the scotoma in

Diplopia is in the vast majority of cases binocular, and due to a paralysis or spasm of the eye-muscles, which disturbs the coördination of the eyes and throws their axes out of their normal parallelism. It may be crossed or simple. *Crossed diplopia* occurs in cases of divergent squint; the image seen by the left eye lies to the right of that seen by the right eye. *Simple* or *homonymous diplopia* exists in convergent squint. In it the image seen by the left eye lies to the left of the other image. A great aid to the memory in regard to diplopia is afforded by remembering the rule laid down by Gowers in his lectures,—namely, that when the prolonged axes of the eyes would cross, the images are not crossed; whilst when the prolonged axes would not cross, the images are crossed. In other words, convergent squint causes simple diplopia, divergent squint causes crossed diplopia.

Monocular diplopia—i.e., the seeing with one eye of one object as two —is a very extraordinary symptom, which may be present in one or both eyes. It has been noted as the result of protracted use of the eye with the microscope; as due to defects in the eye itself, relieved by spectacles; and as the outcome of cerebral traumatism and of various organic lesions. There appear to be three classes of cases: first, those in which the lesion is of that portion of the eye which transmits light; second, those in which there are troubles of refraction or accommodation; third, those in which there is a cerebral lesion. According to our present knowledge, cerebral monocular diplopia has no localizing significance, as it has been reported in cases of tumors of the right ventricle, of softening of the convolutions of the right posterior hemisphere, of abscess of the temporo-sphenoidal and occipital lobes, and as the result of violent blows upon the head producing no demonstrable local change. Duret in one case of centric diplopia found that the portion of the retina which perceived the double image was paretic. The best but not altogether satisfactory explanation of cerebral monocular diplopia as yet offered is that it is due to disassociation of the two hemispheres, which results in each cuneus having a distinct conscious perception of its own image.

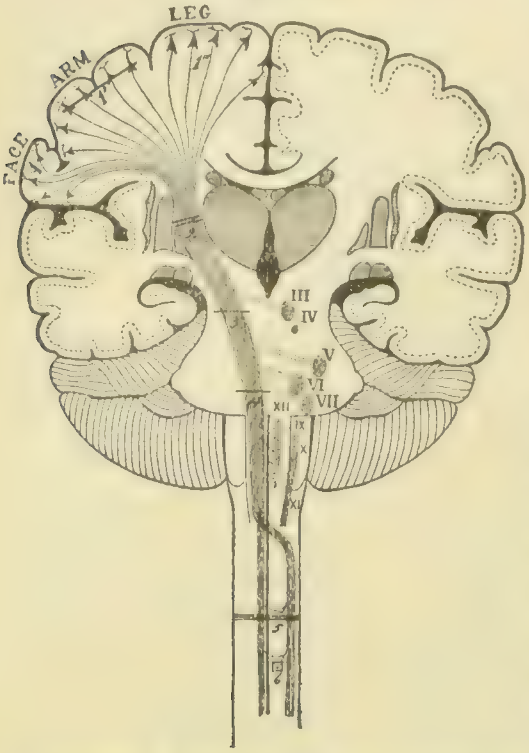
Practical Locating of Lesions.—The following generalizations so evidently follow from what has already been said that they need no comment. *First*, destructive lesions of the ascending frontal or parietal convolution produce monoplegias of the face, arm, or leg, according to the part affected, or, if the lesions be sufficiently wide-spread, cause an association of monoplegias which may amount to a complete hemiplegia, with loss of sensibility by involvement of cortical sensory cells. *Second*, irritative lesions of the convolutions mentioned produce local convulsions, spasms, or choreic movements in the part or parts corresponding to the centres. *Third*, destructive lesions in the centrum ovale, if large,

---

which these colors are observed. This is especially the reason that the patient will recognize the color of a large body and mistake that of a very small one.

cause hemiplegia, but if the lesion be minute a partially distributed hemiplegia,—that is, a hemiplegia without involvement of the face or, it may be, of the leg,—whilst irritative lesions produce, usually, corresponding contractures. Either lesion may be accompanied by a loss of sensibility upon the same side as the motor paralysis, due to division in the corona radiata of the fibres going to the sensory cortical centres. *Fourth,* a destructive lesion in the anterior two-thirds of the posterior limb of the internal capsule will produce a hemiplegia; if, however, the lesion be very small and situated far anteriorly, the face will be most affected, the leg least so, and *vice versa.* *Fifth,* lesions in the middle third of the posterior limb of the internal capsule are prone to influence sensation as well as motion, on account of their tendency to encroach upon the posterior third of the capsule; under these circumstances the hemianæsthesia is upon the same side as the paralysis, because the lesion is above the decussation of both motor and sensory fibres. *Sixth,* if a lesion encroach upon the whole of the posterior third of the capsule the loss of sensibility is complete, affecting not only general sensibility, but also all the special senses, as at this point conducting fibres from each of the senses run together, constituting the so-called "sensory crossway" of Charcot. *Seventh,* permanent irritative lesions of the corona radiata or of the internal capsule may cause tremors or choreic movements (hemichorea, athetosis, post-paralytic chorea). *Eighth,* a destructive lesion in the crus cerebri will produce a hemiplegia or a hemianæsthesia according as it is situated in the crusta or in the tegmentum, or it will produce a hemiplegia with a hemianæsthesia on the same side if it encroach upon both regions; if the lesion be so situated as to interfere with the oculo-motor nerve there will be an oculo-motor palsy opposite to the paralysis of motion and sensation. *Ninth,* a lesion in the pons situated high up may produce a hemiplegia or a hemianæsthesia without involvement of the special senses, according as it is in the motor or in the sensory region, or it may produce a hemianæsthesia and a hemiplegia upon

Fig. 10.



Schematic diagram, after Van Gehuchten, showing motor pathway and paralytic effects of lesions. III, IV, V, VI, etc., on the right represent nuclei of cranial nerves; lesion at 1, 1′, 1″, would cause respectively monoplegic paralysis of face, arm, or leg on opposite side; lesion at 2 would cause complete hemiplegia of opposite side of body; lesion at 3—if large enough to cut descending band of fibres and outgoing nerve—would cause opposite hemiplegia and oculo-motor paralysis on side of lesion (crossed paralysis); lesion at 4 would cause opposite palsy of arm and leg, with, if lesion were large enough, palsy of oculo-motor and facial nerve on side of lesion; lesion at 5 would produce spinal paraplegia.

the same side. If, however, the lesion be not high up,—*i.e.*, not above the decussation of the facial nerve,—it will cause an alternating paralysis in which the opposite sides of the face and body are affected; or if it involve the sensory nerve-fibres it will produce hemianæsthesia of the face upon the side opposite to the motor paralysis of the body,—the so-called alternating hemianæsthesia. *Tenth*, destructive lesions of the optic thalamus and of the striate body are apt to produce hemiplegia, probably by an influence upon the neighboring internal capsule.

Lesion of the corpora quadrigemina produces paralysis of the eye-muscles, with various anomalies of the pupils and lessening of the central vision (anterior pair), or of audition (posterior pair). According to Nothnagel, the union of double ophthalmoplegia with ataxia is characteristic of lesion of the corpora quadrigemina, but L. Bruns reports a case in which these symptoms were present in a tumor of the middle cerebellar lobe. It is probable that in cerebellar cases the ataxia always precedes, in quadrigeminal cases follows, the ophthalmoplegia. Tumors of the corpora quadrigemina are apt to produce an early and severe optic neuritis.

ATHETOSIS. HEMICHOREA.

Athetosis (see page 380) was described as a disease by Hammond, but is certainly only a symptom. In autopsies various lesions have been found; in some instances they have been confined to the cortex, but in the great majority of cases the seat of the disease has been the internal capsule, or the parts in its immediate neighborhood.

Under the name of *double athetosis* Michaïlowski described a disease characterized by peculiar involuntary movements over the whole body, but especially affecting the arms and legs and the two sides of the face, always associated with more or less rigidity of the limbs, and appearing almost always congenitally or in very early childhood in imbeciles, but sometimes developing in early adult life.

As in simple athetosis, so in double athetosis various brain-lesions have been found; there is, therefore, no constant nerve-lesion in double athetosis, so that the latter must be looked upon as a symptom, the outcome of various diseases of the brain which produce similar lesions in both hemispheres and consequently cause athetosis in each side of the body. Double athetosis is commonly associated with imbecility, because sclerotic and other brain diseases which produce athetosis are apt to destroy the mental faculties.

Closely allied to athetosis are other forms of motor disturbance produced by disease of the internal capsule or its neighboring parts. The most important of these are *hemichorea*, *hemi-tremors*, and especially the choreic movements which sometimes precede but more frequently follow cerebral apoplexies, constituting the so-called *pre-hemiplegic* and *post-hemiplegic chorea*. Charcot believed that the lesion of hemichorea was always in the posterior portion of the internal capsule; hence the frequent association of hemichorea with hemianæsthesia. It seems, however, to be proved that any of these various disordered movements may be produced by lesions in the cortex or in almost any part of the motor tract; indeed, Mengui asserts that they may result even from lesions of the spinal cord or of the peripheral nerves, and H. C. Wood's experiments upon choreic dogs certainly proved that both rhythmical and irregular choreic movements may be of spinal origin.

APHASIA.

Human beings communicate with one another by means of written and spoken words, by pictures or hieroglyphs, by signs, and by musical tones. For each form of communication there are necessary an apparatus for expression and one for the purpose of perceiving that which has been expressed by another person. Each apparatus is composed of two parts, one central, the other peripheral.* In the receptive apparatus are a peripheral receptive nerve-mass, a nerve-trunk, and a centre of conscious perception. In the apparatus for expression is a brain-centre in which thought is converted into a word-impulse which, travelling outward, produces the motions which cause words, sounds, speech, gestures, etc. When the peripheral apparatus is paralyzed, communication is correspondingly interfered with, and there results loss of the power of articulation (*anarthria*), or loss of the power of perception,—that is, blindness and deafness.

Cases in which the peripheral apparatus is alone at fault are not included under aphasia, this term being used to represent all forms of interference with human communication by disease of the higher nerve-centres immediately connected with conscious thought and its expression. It is plain that the interference may be with the function of emission or of reception, and there may be *motor* or *ataxic aphasia*, the loss of the power of expression, and *sensory aphasia*, the loss of the power of perception of that which has been expressed.

Ataxic aphasia, as it may invade any form of expression, is divided into *ataxic aphasia* proper, or loss of the power of expression by spoken words; *agraphia*, or loss of the power of expression by written words; *aglyphia*, or loss of the power of picture-making; *ataxic amusia*, or loss of the power of expression by musical tones (singing); *musical agraphia*, or loss of the power of writing music; *ataxic amimia*, or loss of the power of expression by gesture.

Sensory aphasia is divided into word-deafness, or loss of power of recognizing heard words; word-blindness (*alexia*), or loss of power of recognizing seen, written, or printed words; figure-blindness, or loss of power of recognizing drawn or painted figures; *sensory amusia*, or loss of power of recognizing heard musical tones; *sensory amimia,* or loss of power of recognizing gestures. There have been cases in which the patient could sing the tune without words, and even intelligibly express desires by accents and intonations; others in which, with complete aphasia, agraphia, and word-blindness, the patient could sing the melody and also the words; also, cases of word-blindness in which the patient could read musical notes, others in which he could recognize the word by tracing the letters with the finger. The language of emotions and the words in most frequent use are the longest preserved. Thus, aphasics can sometimes swear or pray, and "yes" and "no" are the last words to go.

---

* Peripheral as here used includes the nerves and nerve-centres of the spinal cord or the medulla oblongata, or the special-sense ganglia at the base of the brain.

Any of the various forms of aphasia may be partial in its development. If its lack of completeness is general, it is often expressed by putting the prefix "par" before the name. Thus, partial loss of the power of recognizing musical notes is *paramusia;* partial word-blindness, *paralexia.* Medical literature also recognizes and has given name to various peculiar partial aphasias, which need only be mentioned here. Thus, in ataxic aphasia, when only the word or even a letter is concerned, the case is spoken of as one of *monophasia,* whilst the term *paraphasia* is used to express the condition in which the subject employs wrong words. *Literal agraphia* is the condition in which the power of writing letters is lost; *verbal agraphia* is that in which the power of writing words is alone affected; *paragraphia* is by some restricted to the condition in which wrong words are written. Sensory amusia is by Edgren known as *tongue-deafness,* when the power of recognizing sounds by the ear is alone lost; whilst those cases in which the power of recognizing musical notes by the eye has gone are instances of *note-blindness.*

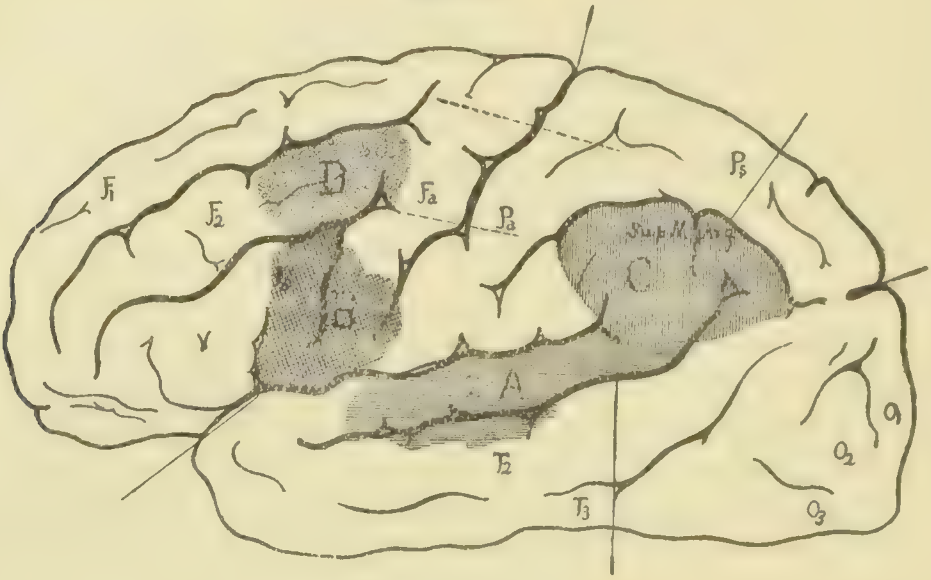
In the majority of cases aphasia is due to organic disease of the brain-cortex or of the conducting nerve-fibres passing from the cortex to the spinal cord. It may, however, be of hysterical origin, or it may be a phenomenon of reflex inhibition produced by irritation of the stomach, intestine, etc. It is sometimes caused by simple brain-exhaustion, and may be one of the symptoms of a violent nerve-storm, such as a migraine. In organic aphasia occurring in right-handed persons the lesion is practically always in the left cerebral hemisphere; in completely left-handed persons it is usually in the right hemisphere. In ambidextrous persons the speech-centre probably follows the use of the hand in writing; or, to speak more accurately, the use of the hand follows and indicates the lesion of the speech-centre. Thus, in a case seen by H. C. Wood, in which the person was right-handed for all acts except writing, a left-handed palsy—that is, of the writing hand—was accompanied by aphasia.

It is probable that the original failure of the right hemisphere to develop its speech-centres is due to habitual disuse not only in the infancy of the individual but through successive generations. In some individuals it is possible to develop the right third frontal convolution after the destruction of the left, so that the power of speech shall be restored.

(Tuke and Fraser, *Journal of Mental Diseases*, 1892.) There is plausibility in the view held by some neurologists that the familiar words "yes" and "no," which often remain in aphasia due to a severe left-sided lesion, have their origin in the right cerebral speech-centre, which has attained sufficient development to compass a few rudimentary expressions.

The cortical localization of aphasia may be summed up in accordance with the elaborate paper of Wylie as follows :

FIG. 11.



The posterior half or three-fourths of the left first temporal convolution is the auditory speech-centre, in which are stored the auditory speech-images (*A*) : paralysis of it causes word-deafness. The third frontal or Broca's convolution is the centre for the motor memories of speech (*B*) : paralysis of it causes motor aphasia. The angular and supramarginal convolutions constitute the centre for the visual images of written and printed speech (*C*) : paralysis of it causes word-blindness. The posterior extremity of the second frontal convolution (*D*)* is the graphic motor centre, in which are stored the motor memories which guide the right hand in the act of writing : paralysis of this centre causes agraphia. Edgren places the centres for tone-deafness in the first and second temporal convolutions of the left side, just in front of the position of word-deafness (*D*).

It is evident that disturbances of the speech-function must result from injuries of nerve-fibres which start in the cortical centres. It is believed that these fibres are of two sorts: first, commissural fibres joining centres; second, main pathway-fibres passing downward to the various lower ganglia: hence there are *transcortical* and *subcortical* aphasias.

---

* It is doubtful, however, whether this centre can be yet definitely fixed. We can write with the left hand, and even with the teeth, which are believed to have no connection with this centre. Wernicke thinks that the visual images are the important ones in writing ; but the congenitally blind, who have no visual images, can be trained to write. We know of no recorded case in which the lesion was strictly limited to the centre *D*.

The theory of transcortical aphasia rests chiefly upon disturbances of speech which have been noted as the results of lesions of the insular lobule in the fissure of Sylvius. It is an established fact that lesions of the left insula in some cases produce an aphasia, in others do not; and it is held by various authorities that the insula is a meeting-place for commissural fibres from the various cortical speech-centres, where it may be that they are all brought in contact with a centre of ideation.

The course of the main pathway downward from the speech-centre is not perfectly known. The most probable view is that in the centrum ovale the impulses travel through the band which lies under the posterior third of the third frontal convolution (*pediculo-frontal band* of Pitres). According to Brissaud, the fibres are associated with those going to the face, both in the internal capsule (in the region of the knee) and in the crus.

It is often important to distinguish between a subcortical and a centric aphasia. In a subcortical aphasia the power of word-thought remains, the motor fibres traversing the pathways mentioned being alone affected, so that the symptoms are those of a pure ataxic aphasia (without sensory aphasia). The conducting fibres from the graphic centres probably pass with the arm-fibres through the internal capsule, so that in a subcortical aphasia there is no agraphia, unless the lesion in the internal capsule is large enough to paralyze the arm and face.

Conditions allied to Aphasia.—There are certain disorders of memory, often the result of organic disease, which are closely allied to aphasia. *Apraxia* is a condition in which the patient has more or less completely forgotten the nature and use of objects about him. For example, he attempts to use a knife for a spoon, to eat a piece of soap, or to write with a fork. All the senses may be involved in this condition, or only one form of perception may fail to awaken old memories. Thus, in object-blindness (*soul-blindness*, *mind-blindness*), which is very often associated with word-blindness, the patient is not able to recognize things or persons by sight, but recognizes them by hearing when they give forth sound. For instance, he will not know his intimate friends on looking at them, but may recognize them at once when they speak to him.

CEREBELLAR LOCALIZATION.

Lesions of the cerebellar peduncles sometimes produce rotatory movements (movements of *manège*) along the long axis of the body. According to Rosenthal, the diagnostic symptoms of tumor of the cerebellar peduncle are headache, vertigo, disorders of the special senses, hemiplegia, unsteady gait with a tendency to fall upon the side, and partial rotation along the vertical axis with lateral rotation of the head. Certainly, however, lesions of the cerebellar peduncles are not always accompanied by rotatory movements.

Titubation is characteristic of disease of the middle lobe of the cerebellum. In it the feet are held well forward and widely separated from each other. If the attempt be made to bring them close together, peculiar movements of extension and flexion will occur in them, and at the same time the body will begin to rock and stagger more and more violently, until, in extreme cases, the subject falls unless he can seize some support. In unusual instances the movements are definite and in one direction, but commonly they are irregular and vary both in direction and in force. The staggering may be so great that the patient is unable to move a step. Very commonly it is impossible for him to turn suddenly without falling. The symptoms may or may not be intensified by darkness or by closing the eyes. The walk resembles that of an intoxicated man. There is a similar staggering, with to-and-fro movements of the whole body, resulting in a zigzag instead of a straightforward progression. In most cases the feet are raised only a short distance from the ground, and are moved with a peculiar irregularity of step.

In rare cases there is a marked tendency to fall or run backward or, it may be, forward. The differentiation of titubation from ataxia is aided by the facts that in cerebellar disease the patient, lying in bed, is able to move the legs with normal promptness and accuracy, and that the arms are never affected.

DISEASES OF THE MEMBRANES OF THE BRAIN.

PACHYMENINGITIS.

DEFINITION.—Inflammation of the dura mater.

Pachymeningitis Externa is an inflammation of the external layer of the dura mater, which is probably always secondary to traumatism, disease of the skull, or septic infection. There are no distinctive symptoms. Headache is usually, but not always, present, and there may be delirium, convulsions, or cortical pressure palsies. The treatment is that of the original disease, with local measures as indicated.

Pachymeningitis Interna, an inflammation of the internal layer of the dura mater, may possibly occur in purulent form as a result of sepsis.

Pachymeningitis Interna Hæmorrhagica (*Hæmatoma of the Dura Mater*) is produced by traumatism, alcoholism, syphilis, sunstroke, and other causes. After death there is found in some cases a subdural hemorrhage with little show of membrane, in other cases a large development of subdural membrane with but little hemorrhage. More commonly there is abundance both of the subdural membrane and of the hemorrhage. Two views are held by authorities as to the relations of the hemorrhage and of the membranes, some teaching that the membranes precede the development of the hemorrhage, others that the hemorrhage precedes the membrane. The probabilities are that the primary change is an inflammatory formation of membrane, but that in some cases the hemorrhage occurs almost at the onset of the disease. In old cases there is often an associated atrophy of the convolution. The symptoms of hemorrhagic pachymeningitis are indefinite: excessive headache with somnolence, and with a history of the causes previously enumerated, should give rise to the strongest suspicion of the disease. Apoplectic attacks, convulsions, optic neuritis, even hemiplegia, may be secondarily produced by hemorrhage from the delicate vessels in the inflamed membrane. Treatment is rarely satisfactory in its results, but severe repeated counter-irritation, especially by the actual cautery, with the continuous administration of minute doses of the mercurials and of the iodides, may be useful in the earliest stages of the disease. Opium may be employed for the relief of pain.

LEPTOMENINGITIS. MENINGITIS. ARACHNITIS.

DEFINITION.—Inflammation of the pia mater and arachnoid.

It is necessary to recognize three forms of leptomeningitis,—acute meningitis, acute tubercular meningitis, and chronic meningitis.

ACUTE MENINGITIS.

DEFINITION.—An acute inflammation of the pia mater and arachnoid.

ETIOLOGY.—Although a bacterial invasion is the probable cause of all acute meningitis, a distinction is to be drawn between primary and secondary varieties, according to the circumstances of the infection. Under the former are included the sporadic and epidemic cases. Secondary forms include those originating in a variety of ways.

Sporadic and epidemic cases probably originate from the same cause or causes, and especial importance is to be attached to the presence of bacteria, particularly the pneumococcus and streptococcus.

The secondary variety is also of probable infectious origin, the bacteria being other than those concerned in the origin of the sporadic and epidemic cases. They enter the membrane from without, probably through blood-vessels or lymph-vessels. Thus, a secondary meningitis may result from wounds of the scalp, erysipelas of the face and scalp, phlegmonous inflammation of these regions, inflammation of the middle ear, chronic nasal catarrh, and operations upon the head. A secondary meningitis may also result from a remote infectious process, as pleurisy, pneumonia, abscess and gangrene of the lung, and the various infectious diseases, also from the amœba coli communis.

An apparently acute meningitis may also be produced by gout, rheumatism, sunstroke, Bright's disease, and syphilis; but in such cases the attack is usually, if not always, an exacerbation of a chronic meningitis whose symptoms may have been so mild as not to have been apprehended.

MORBID ANATOMY.—The anatomical changes in acute leptomeningitis are essentially the same as those found in cerebro-spinal meningitis (see article on cerebro-spinal meningitis), and consist in a sero- and fibrino-purulent infiltration of the meshes of the pia mater, a focal cortical encephalitis, and acute ventricular dropsy.

SYMPTOMATOLOGY.—In a typical case of acute meningitis there is rapidly developed, with or without a chill, an intense headache with paroxysmal exacerbations, intensified by loud sounds or light, soon followed by delirium with high fever, and by stiffness of the neck, due to contractions of the posterior muscles. Vomiting occurs early and repeatedly. General or partial convulsions may take place. In the continuance of the case, strabismus, ptosis, contraction, dilatation, or inequality of the pupil, and disturbances of vision or of hearing, indicate the involvement of the cranial nerves. The pulse is usually rapid unless there is irritation of the pneumogastric centres, when it becomes slow. There is generally pronounced fever, but the range of temperature varies greatly according to the cause of the disease.

Optic neuritis may occur early in the disease; later, coma, paralysis, and other decided symptoms of brain-pressure are developed.

DIAGNOSIS.—When a meningitis has gone so far as to involve the cranial nerves the diagnosis is usually easy, but in the early stages it is often impossible in view of the fact that all the symptoms of the early meningitis may be produced by pneumonia, typhoid fever, or other acute disease, without any organic alteration in the brain-membranes.

PROGNOSIS.—The prognosis of acute meningitis is always grave. It is largely dependent upon the nature of the cause of the attack.

TREATMENT.—The treatment of an acute meningitis usually resolves itself into the treatment of the concomitant condition, along with local blood-letting, persistent application of cold to the head, and persistent use of counter-irritants in the form of blisters over the shaved scalp. Calomel should be given freely to mild salivation unless contra-indicated by sepsis or other general condition. If the disease be due to extension of inflammation from the middle ear, surgical interference should be immediate.

In debilitated or cachectic children under two years of age occurs a variety of acute meningitis which affects chiefly the posterior portion of the base of the brain in the region of the cerebellum (*posterior meningitis*), and often by closing the foramen of Magendie (*occlusive meningitis*) produces an acute hydrocephalus. In most fatal cases the exudation is largely purulent. It is found not only at the base of the brain, but also in the choroid plexuses, the velum, and the ependyma of the third ventricle, whilst the serous fluid frequently distends not only the lateral ventricles, but also the fourth ventricle. The symptoms are especially characterized by the slowness of their development, and by an early, very persistent, and severe retraction of the head (*cervical opisthotonos*).

The case may last for weeks, with slow, irregular fever and great weakness, without other distinctive symptom than that of rigidity of the neck. If death does not occur early, chronic hydrocephalus may result.

TUBERCULAR MENINGITIS.

DEFINITION.—A meningitis associated with tubercles in the brain-membranes, especially in the pia mater, and due to the presence of the tubercle bacillus.

ETIOLOGY.—Tuberculous meningitis is probably always secondary to a tubercular deposit elsewhere, and is often a part of an acute general tuberculosis. Heredity is a strong predisposing cause.

MORBID ANATOMY.—The tubercles are usually in the form of grayish-white granules near the vessels, because of their formation in the perivascular sheaths. They especially affect the branches of the middle cerebral artery, particularly those going to the perforated space. The associated inflammatory exudation is commonly more abundant at the base, and is fibrino-purulent, involving generally the Sylvian fissure, and not rarely giving rise to some distention of the ventricle. The cerebral substance is often superficially infiltrated, and spots of softening may be present.

SYMPTOMATOLOGY.—In the great majority of cases the disease develops insidiously in feeble children, who fail in appetite, become extraordinarily peevish and irritable, and suffer from malaise, headache, constipation, irregular, disturbed sleep, loss of flesh, and wandering pains. Usually after two or three weeks, sometimes after a much longer period, the meningitic symptoms develop suddenly, it may be with a convulsion, followed by fever, violent headache, giddiness, vomiting, and delirium or stupor. Usually the child lies in bed in an apathetic unrest, complaining bitterly of loud sounds, bright lights, or any disturbance. Even during stupor the child utters a peculiar sharp scream (hydrocephalic cry), probably extorted by a pain through the head. During this first period (stage of irritation) the fever may reach 103° F., and there are sudden startings, outbreaks of delirious terror, twitchings of the muscles, and some rigidity of the neck. In the second period (stage of oppression) the stupor becomes more pronounced, and the general muscular relaxation extreme: the pupils are unequal or dilated; the respiration is sighing, often irregular; the pulse is slow and intermittent; the bowels are obstinately constipated. Vomiting is often severe. The fever temperature is irregular, varying from below the norm to 102.5° F., having no regularity of rhythm, rising and falling, it may be, several times in the twenty-four hours. The skin is dry and harsh. If the finger-nail be drawn across the skin, the *tache cérébrale* (a red line) will appear; it is without diagnostic significance. Gradually the patient with developing blindness and deafness sinks into the last stages of paralysis, with complete coma, low delirium, and general or partial convulsions. There are also optic neuritis, strabismus, and ptosis, rapid, irregular, feeble pulse, and all the symptoms of a profound typhoid state. Death occurs ordinarily between the tenth and the thirtieth day. Local paralyses, monoplegia, and even hemiplegia may appear in the later stages of the disease. There may also be diarrhœa or lung-complications as the result of the wide-spread tuberculosis.

In some cases remissions give hope of recovery, but they are usually of short duration. In the last days Cheyne-Stokes breathing is not rare, and a sudden rise of bodily temperature (even to 109° F.), followed by an abrupt fall (even to 94° F.), often precedes death.

DIAGNOSIS.—In the earliest stages the diagnosis may be between typhoid fever, simple meningitis, and tubercular meningitis. The severity of the headache, the vomiting, and the constipation usually distinguish it from typhoid fever, but if there should be abdominal tuberculosis and consequent diarrhœa and tympanitis a mistake is possible, so that it is often well to reserve the opinion until stiffness of the neck, ocular palsies, or involvement of special senses make the case clear. In syphilitic and rachitic children a meningitis occurs which can be distinguished from the tubercular form only by its yielding to treatment.

PROGNOSIS.—The disease probably always ends fatally.

TREATMENT.—There is no specific treatment: the good results which are alleged to have followed the use of calomel have probably been due to the reported cases having been of specific origin. Whenever there is a suspicion of syphilis or of rachitis, appropriate medication should be actively employed.

CHRONIC MENINGITIS.

Chronic meningitis is usually due to sunstroke, to trauma, or to syphilis, but may result from an acute inflammation, and in the majority of cases involves to a greater or less degree the cortex, through its influence on the brain-arteries, which run from the pia mater directly to the cortex. Its treatment varies so much with its cause that the former will be discussed under cerebral syphilis and sunstroke. In traumatic general meningitis the treatment consists of continuous severe counter-irritation and the use of minute doses of mercurials and the iodides for a long time, with abstinence from all mental labor and excitement.

DISORDERS OF THE CEREBRAL CIRCULATION.

CEREBRAL ANÆMIA.

DEFINITION.—Deficiency of blood in the brain.

SYMPTOMATOLOGY.—Acute cerebral anæmia (syncope), as after hemorrhage, produces tinnitus aurium, a sense of general weakness or faintness, disorder of the special senses, cold sweat, loss of consciousness, dilatation of the pupil, general convulsions, and death from failure of the respiration.

Chronic brain-anæmia is affirmed to cause vertigo, apathy, tinnitus aurium, sleeplessness, loss of memory, and hallucinations. Under the name of *spurious hydrocephalus*, Marshall Hall described a condition which in feeble children is especially apt to follow excessive diarrhœa, and has been mistaken for tubercular meningitis, on account of the semi-coma, convulsions, and retraction of the head; but there is no fever, and the pulse is rapid throughout. How far these symptoms are due to lack of blood in the brain and how far to simple exhaustion remains unsettled.

TREATMENT.—In syncope the patient should be placed in a recumbent position, cold water dashed in the face, the nostrils irritated by ammoniacal exhalations, and strong alcoholic drinks administered. In severe, persistent cases strychnine, tincture of digitalis, nitroglycerin, may be given hypodermically; if these fail, a pint of tepid weak saline infusion may be injected into the buttocks, or transfusion may be practised. In chronic brain-anæmia the indication is to cure the anæmia and exhaustion by appropriate measures.

Cerebral œdema or *serous apoplexy* may occur as an acute, severe condition in chronic Bright's disease, and be associated with coma, convulsions, inequality of the pupils, and even partial hemiplegia, so as closely to simulate true apoplexy.

The question how far the œdema is the cause of these symptoms is an exceedingly difficult one to answer. Undoubtedly most if not all of them may be produced by uræmia without œdema, but it is very doubtful whether a pure uræmia will produce localizing symptoms such as inequality of the pupils and partial hemiplegia. In brain-atrophies and in extreme passive hyperæmia there is often an excess of serous fluid in the brain. Local brain-œdemas, probably from local interference with the circulation, occur in the neighborhood of tumors and other cerebral lesions.

CEREBRAL HYPERÆMIA.

The subject of cerebral hyperæmia is one of great obscurity, the diagnosis being continually made by practitioners of medicine as a means of accounting for various obscure cerebral disturbances, such as are seen in cerebral asthenia from overwork. It is possible that weakness of the cerebral vessels from habitual overuse may exist in these cases, but there is no proof of the truth of the supposition. Again, the sleeplessness, headache, giddiness, apathy, somnolence, slight mental confusion, etc., which are often seen in diseases of the heart, in chronic emphysema, in tumors at the root of the neck or in the mediastinum, are attributed by many, with a show of reason, to passive or venous congestion of the brain. In many of these cases, however, the symptoms are probably largely due to the presence of carbonic acid and other poisons in the blood.

The headaches, with red face and throbbing carotids, occasionally met with in persons of full habit, and relieved by a mercurial or other brisk purge, may be ascribed to an acute cerebral congestion. There are undoubtedly rare cases of sudden attacks, with a sense of great fulness in the head, turgid, red face, throbbing carotids, vertigo, and finally, it may be, complete unconsciousness. Such attacks may simulate an apoplexy and be at once relieved by bleeding. The cause of these attacks is entirely obscure: the congestion, so far as can be made out, is primary. It is probable that in some cases of true hemorrhagic apoplexy the attack is not hemorrhagic from the beginning, but simply congestive, a diseased blood-vessel giving way from the strain of a local increased blood-pressure. In apoplectic attacks following violent sudden emotion, a primary cerebral hyperæmia with a secondary cerebral hemorrhage is probably the history.

Rare cases occur in young children of deep sleep or semi-coma without obvious cause, lasting, it may be, for some hours. These have been explained as instances of acute cerebral hyperæmia. The difficulty in all such cases is, first, that there is no proof of the existence of the cerebral hyperæmia; secondly, that at present there seems no way of explaining the occurrence of the cerebral hyperæmia.

TREATMENT.—When cerebral hyperæmia simulates apoplexy a free venesection may be called for. In less severe cases leeches should be applied to the temples, or cups to the back of the neck, and a drastic purgative administered.

DISEASES OF THE BLOOD-VESSELS OF THE BRAIN.

CEREBRAL THROMBOSIS AND EMBOLISM.

DEFINITION.—Arrest of circulation in the brain by embolic or thrombotic occlusion of a vessel.

ETIOLOGY.—The causes of the formation of emboli and of thrombi in the brain are the same as those which act in other portions of the body. The results are more serious, on account of most of the cerebral arteries not anastomosing, so that it is not possible for a collateral circulation to be set up.

MORBID ANATOMY.—Any of the brain-vessels may be involved. It is, however, very unusual for the cerebellar arteries to be affected by an embolus, which, following the direct line of blood-current from the heart, naturally passes up the left carotid into the left middle cerebral artery, or, if it be too large, is arrested in the basilar artery. Sometimes the embolus lodges in the vertebral or the posterior cerebral artery, very rarely in the anterior cerebral.

SYMPTOMATOLOGY.—The symptoms produced are those of embolic or thrombotic arrest of circulation, and vary with the size and situation of the vessel affected. If the middle cerebral or other large artery be suddenly blocked, there will be loss of consciousness, with hemiplegia, and, in some cases, convulsions. The temperature, usually, is not immediately affected, but after some hours begins to rise, though it rarely reaches above 101° F. The temperature disturbance may be very slight and transient or may continue some days. In cases of thrombosis, giddiness, headache, loss of memory, brief attacks of stupor, and other symptoms due to disordered circulation may precede the final closure of the blood-path. Sudden arrest of circulation, even in one of the smaller arteries, may be accompanied by an apoplectic attack, but often the loss of function is the only symptom. In atheromatous thrombosis there is not rarely a creeping hemiplegia: thus, there is a facial palsy, and some hours afterwards the arm of the same side weakens, and then the leg. One or two days may be required for the full development of the symptoms. In some cases temporary palsies occur, probably as the result of the partial formation and the subsequent dissolving of fibrinous thrombotic clots. Cutting off the blood-supply produces a change in the brain-structure, which is accompanied by softening and discoloration,—*red softening* in the cortex and the ganglia, *white softening* in the centrum ovale. The so-called yellow softening is an advanced stage of the red. The softened tissue is composed of disintegrating nerve-fibres, fatty and granular débris, and granular corpuscles. When the softening is of limited extent the tissue may remain in this condition for months, but finally there is absorption, with the deposition of cicatricial tissue, or, if the mass has been too large, the formation of a cyst. There is no regenerative power in softened brain-tissue.

The symptoms of arrested brain-function vary almost indefinitely in accordance with the seat of the artery which is affected. Whole tracts of the brain may be involved, or one centre may be dissected out. Thus, there may be a complete hemianæsthesia and hemiplegia, or there may be a simple hemianopsia or a purely motor hemiplegia. The following schedule shows the result of the occlusion of the more important arteries.

Carotid Artery.—Occlusion of a carotid artery by tying may cause no symptoms, but it may be followed by a hemiplegia, which disappears in a short time by re-establishment of the circulation through anastomosing vessels. If, however, an embolism or a thrombosis forms within the skull, there is commonly a progressively increasing clot, which finally invades the circle of Willis and causes coma and death, or, if recovered from, a hemiplegia or various palsies.

Vertebral Artery.—Occlusion of a vertebral artery usually produces a hemiplegia, which in most of the recorded cases has been upon the same side as the occlusion. The tongue, palate, and larynx are affected, and there is great impairment of swallowing and of articulation. The motor paralysis is sometimes accompanied by partial anæsthesia, and is usually temporary, owing to the free anastomoses between the vertebral and the anterior spinal arteries. In some cases the involvement of the respiratory centres leads to immediate death, and in certain cases there has been a bilateral paralysis, probably due to arterial anomalies.

Basilar Artery.—Thrombus of a basilar artery may cause a foudroyant apoplexy, with rise of temperature and death within twenty-four hours, or with partial recovery and various localizing symptoms due to softening in the medulla and in the pons. The paralysis is often alternating, and if with a basilar artery the posterior connecting artery is affected, there is softening of the cerebral peduncle, which is chiefly supplied by the direct or ascending portion of the posterior cerebral. The artery of the oculo-motor centre, also rising near the origin of the posterior cerebral, may be affected even when the posterior cerebral is not, giving rise to a crossed oculo-motor paralysis.

Anterior Cerebral Artery.—Obstruction of the anterior cerebral artery may give rise to comparatively few symptoms, on account of the free anastomoses between this artery and the middle cerebral. There may be softening of the olfactory bulb, which is supplied by the first branch of the artery, with loss of smell. A monoplegia of the leg might (theoretically) be produced by the softening of the paracentral lobule and the neighboring marginal convolution.

Middle Cerebral Artery.—Occlusion low down causes hemiplegia by softening of the anterior portion of the internal capsule, the internal ganglia also being affected ; and if the left artery be affected there is also aphasia. There is usually some disturbance of sensation, which is commonly not permanent. Occlusion above the arteries going to the centre of the brain gives rise to cortical paralyses and aphasia (left artery) ; occlusion of the first branch causing softening of the third frontal, of the second and third branches softening of the ascending frontal, and of the fourth branch softening about the posterior limb of the fissure of Sylvius with sensory aphasia. Ptosis may occur on the side opposite the hemiplegia. (Gowers.)

Posterior Cerebral Artery.—Obstruction may cause hemianæsthesia from softening of the tegmentum or of the posterior part of the internal capsule, or hemianopsia from softening of the occipital lobe. This artery anastomoses with so much freedom that the paralytic effects are often temporary. Gowers states that sometimes there is complete temporary loss of sight, probably due to reflex inhibition.

DIAGNOSIS.—The diagnosis between a hemorrhagic and a thrombotic or embolic apoplexy or paralytic stroke can rarely be a positive one, except in cases of congestive apoplexy, the symptoms of arrested circulation resembling those of the syncopal form of apoplexy. (See page 501.) If there be cardiac or aneurismal disease capable of furnishing a source for the embolus, a diagnosis of embolism may be made, but will often be erroneous. Slowness of development, failure of rise of temperature, minuteness of paralysis (demonstrating that the lesion is cortical), the occurrence of several attacks at short intervals, each attack plainly involving a small territory of the cortex, point towards thrombosis, but may all coexist in a case of hemorrhage. Spasmodic contractions and pronounced fever are strong indications of hemorrhage.

PROGNOSIS.—The prognosis is always grave, and is in direct proportion to the severity of the symptoms. If the paralysis has lasted some days, improvement is less to be hoped for than in the hemorrhagic disease.

TREATMENT.—There is no known means of dissolving an embolus or of preventing the formation of a thrombus. Even when thrombosis is threatening nothing can be done in the way of prevention, except in syphilitic cases. Depleting measures of any kind are harmful. Where there is threatened heart-failure, digitalis, alcoholic drinks, and other cardiac stimulants may be useful in maintaining the activity of the circulation, but, unless urgently demanded, may do harm by increasing excitement and even forcing the clot onward. In most cases nothing should be done but to enforce quiet, feed carefully with a light, nutritious diet, and administer calmatives and laxatives as needed. When there is syphilis the medication should be actively antispecific. Mercury is usually preferable to potassium iodide, as more active. The treatment of paralyses resulting from thrombosis does not differ from that proper for cases following brain-clot.

CEREBRAL ANEURISMS.

Any of the cerebral vessels may suffer from aneurisms, which occur in two forms, small miliary multiple enlargements (see Cerebral Hemorrhage, page 499) and single large aneurism. The latter lesion may produce symptoms of pressure or of irritation conforming to its position, but it may be entirely latent until it produces an overwhelming apoplexy. The diagnosis of cerebral aneurism is, except in the rarest cases, a matter of grave doubt. Even if a bruit can be heard, it may be only the normal murmur which in many individuals can be heard in the temporal region.

THROMBOSIS OF CEREBRAL SINUSES.

Thrombosis of a brain-sinus may be due to—1st. Phlebitis, caused by infection from general sepsis, erysipelas, furuncle, diphtheria, typhoid fever, etc., or by extension of a neighboring inflammation, as in suppurative otitis, or by traumatism (in one hundred and fifty-one cases collected by Worden this occurred once).

2d. Advanced cachexia (marantic thrombus), as in chronic diarrhœa, chlorosis, phthisis, old age, infantile disease.

3d. Diseases of the venous system, wide-spread degenerations, local involvements in tumors, etc.

SYMPTOMATOLOGY.—The symptoms of thrombosis of the brain-sinus vary greatly. If only one sinus be involved, the affection may be latent, especially in very young infants. Again, the cerebral disturbance may be lost in the general symptoms of a violent pyæmia; usually, however, violent headache, intense fever, vomiting, delirium, coma, ocular palsies or contractions, and optic neuritis, indicate what has occurred. Convulsions may be present. Pulmonary embolism may occur by the breaking off of small portions of the clot. When in young children the longitudinal sinus is attacked, according to Gerhardt and Eichhorst, there is dilatation of the veins of the scalp in the region of the grand fontanelle, whilst the bregma is depressed from the beginning. When the transverse sinus is invaded, the jugular veins of the same side are apt to be affected, and there is painful œdema of the ear and mastoid region from implication of the posterior auricular veins. Thrombosis of the cavernous sinus is shown by the blocking of the ophthalmic vein and the consequent exophthalmos, œdema of the upper eyelid, swelling of the conjunctiva and face, chemosis, and oculo-motor nerve palsy.

DIAGNOSIS.—Diagnosis is always difficult, often impossible until evidences of venous pressure are apparent. When in any case of otorrhœa a chill followed by fever occurs along with occipital headache and stiffness of the neck or retraction of the head, the diagnosis is sufficiently clear for immediate surgical interference.

PROGNOSIS.—Death is almost invariable in the adult unless surgical interference is possible. Remissions occur, but are very deceitful, although infants sometimes get well with hopeless damage to the brain.

TREATMENT.—No medical treatment is of avail. If a positive localizing diagnosis can be reached, early trephining, evacuation of the sinus, disinfection, and drainage are justifiable.

CEREBRAL HEMORRHAGE.

DEFINITION.—A hemorrhage into the brain-substance.

Hemorrhage into the brain-substance is commonly accompanied by great disturbance of the consciousness, constituting the apoplexy* of ordinary language; but an apoplectic attack may occur without hemorrhage. Hemorrhage into the brain may occur without distinct apoplectic symptoms, when it is known in the ordinary parlance of the sick-room as a paralytic "stroke," or, sometimes, simply as a "stroke."

---

* The term apoplexy means a striking from or a stroke, and might be used for any sudden loss of cerebral function, whether followed by paralysis or not. In this meaning of the term an apoplexy may be produced by an embolism, by toxæmia, and even by a traumatism. Hence some writers use two terms: apoplectic stroke, significant of cerebral paralysis accompanied by primary loss of consciousness, and paralytic stroke, indicating a sudden cerebral paralysis not accompanied by primary loss of consciousness. The word apoplexy is not, however, properly confined in its application to the brain, as it is used to signify a sudden escape of blood into an organ by the bursting of a blood-vessel and a consequent momentary paralysis of function. Pulmonary apoplexy may be cited as a familiar example. In strictness, the word cerebral apoplexy should be used in the text; but custom warrants the employment of apoplexy as equivalent to cerebral apoplexy.

Etiology.—Cerebral hemorrhage, not of traumatic origin, or not due to scurvy, leucocythæmia, purpura, and certain infectious diseases, is always the result of previous disease of the blood-vessels. Such disease may be the outcome of carcinoma or other new growth in the brain, but in the vast majority of cases it is an arterio-sclerosis produced by syphilis, gout, renal disease, old age, and other causes. (See Arterio-Sclerosis.) Apoplexy is, therefore, much more frequent after the age of fifty; but as arterial degeneration is possible at any age, so also is cerebral hemorrhage. Heredity plays an important part in the etiology of apoplexy. Not only are there families in which there is an overwhelming impulse towards premature arterio-sclerosis, but there are families in which there is a distinct tendency to local degenerations of the cerebral blood-vessels, and others in which there is proneness to congestive attacks of the brain. The disease is more frequent in men than in women, probably because of the greater frequency in men of arterio-sclerosis. It is not certain that brain-work, even when habitually in excess for years, has any distinct influence in the development. The belief that men having the so-called apoplectic build or habit—*i.e.*, a stout, thick body with a short neck and an habitually red face—are more liable than others to the disease is still wide-spread. Cardiac hypertrophy is probably more active in its influence.

In the great majority of cases no exciting cause for an apoplectic attack can be traced, but a violent emotion or a violent general muscular strain has occasionally precipitated the disease by increasing the cerebral blood-pressure. Thus, it may happen during defecation, childbirth, slight intoxication, etc., and in a number of cases has occurred during coitus, especially in old men in houses of prostitution.*

Morbid Anatomy.—The most important alterations of blood-vessels leading to cerebral hemorrhages are the miliary aneurisms first noted by Cruveilhier in 1836. They appear as red or black enlargements, about the size of a pin's head, scattered along the small blood-vessels. They are the results of a chronic inflammation of the external and internal coats of the vessels, accompanied by atrophy of the middle coat. According to the teachings of Charcot and Bouchard, the primary alteration is a sclerotic periarteritis with proliferation of the cellular elements in the adventitia, which at first becomes enormously thickened. Zenker and also Eichler, however, believe that the alteration begins in the inner coat and is, in fact, a chronic endarteritis. The hemorrhage resulting from rupture of one miliary aneurism is minute, but usually several give way together, with consequent free loss of blood.

---

* Birds, stallions, and probably other animals occasionally die of apoplexy during copulation.

In some cases of cerebral hemorrhage the changes of the arteries are diffused, not aneurismal; while in rare instances it does not seem possible to demonstrate any sufficient change in the vessels.

Intra-cranial hemorrhages are divided according to their seat into meningeal, ventricular, and parenchymatous (into the brain-substance).

Meningeal hemorrhage, usually the result of gross aneurism or of violence, may be between the skull and the dura or more commonly subdural, or between the arachnoid and the pia mater. Intra-cerebral hemorrhage is more common upon the right than upon the left side, and especially affects the region of the corpus striatum and optic thalamus, particularly the outer border of the lenticular body, which is supplied by the striate artery,—the artery of cerebral hemorrhage. (Charcot.) Intra-cerebral hemorrhage, however, may occur in any portion of the brain, and is accompanied by tearing of the tissues, so that a clot more or less mixed with brain-substance occupies an irregular cavity. Ventricular hemorrhage is rare except as the result of the breaking through of an intra-cerebral hemorrhage. Multiple hemorrhages occur, and are often distinctly symmetrical.

The changes which take place in the blood-clot consist in the absorption of the liquid portion, probably also of the fibrin, and the conversion of the hæmoglobin into pigment, reddish-brown hæmatoidin. If the clot is a large one, an irritative inflammation is set up around it, and a cyst with defined walls and fluid contents is finally formed; when the clot is minute, a dark-colored scar is all that is left. In meningeal hemorrhage degenerations of the convolution involved are very common, especially in infants.

As the result of inflammatory action, a clot of sufficient size, affecting motor centres or motor fibres, causes a *secondary degeneration* which follows the motor path downward. Thus, if the clot is cortical, the secondary degeneration passes along the corona radiata, the internal capsule, the crus, the pons, the medulla, and the cord, even to the ganglionic cells, which, however, do not undergo change.

SYMPTOMATOLOGY.—For the purposes of discussion two types of apoplexy may be recognized, although every grade of attack between the types occurs. The apoplectic attack may come on with great suddenness. It may, however, be preceded by prodromes, such as headache, tinnitus aurium, mental confusion, and hemiplegic or monoplegic numbness or weakness, which are in some cases affirmed to have lasted for some hours. Usually without distinct warning the patient drops unconscious, or he may become confused in speech and manner and then suddenly be stricken, or else gradually grow more and more heavy and finally sink into unconsciousness, or the unconsciousness may be partially recovered from and relapse. At the height of the attack the unconsciousness is complete. The pupil is fixed, dilated or contracted, as the case may be. In the congestive form the face and conjunctivæ are intensely suffused, dark purplish red. The breathing is loud, snoring, and stertorous from the paralysis of the palate. The pulse is usually full and bounding. It may be slower or more rapid than normal; occasionally it is small and hard. The surface is warm. In the syncopal form the face is pale and the breathing quiet, or, if stertorous, not loud and harsh in its sound. The surface is coolish, and the pulse rapid and feeble. In either type of apoplexy paralysis may reveal itself by drawing of the mouth or puffing of the lips upon one side more than upon the other, or by absence of motion in one arm or one leg; the general relaxation may, however, be so extreme as completely to mask the paralysis.

Convulsions may occur in any stage of an apoplexy. The apoplexy may end in death, which may take place in a few minutes, but is usually delayed for many hours. The unconsciousness remains complete; the pulse, whether originally strong or feeble, continually falls in force; the respiration grows more and more shallow, or more and more irregular, and may at last gradually die away or suffer sudden arrest. The cerebral reflexes are frequently lost early in a case of severe apoplexy, especially when the basilar region is invaded by the lesion. Thus it is that the power of swallowing is affected. Complete loss of this function is an almost invariably fatal sign. In a favorable case there is usually a gradual return to consciousness, but the awaking may be sudden.

The subject of temperature in apoplexy needs further elucidation. Usually in a severe case the temperature falls immediately, but, according to Dana, in fatal cases it rises during the twenty-four hours to 102° F. or beyond, to undergo a slight fall the next day unless death intervenes. Except early in the attack, the axillary temperature of the paralyzed side is commonly one-half to one degree higher than on the sound side. A very great rise of temperature may occur in uncomplicated fatal cases (to 105° or even 107° F.); when the lesion is in the hemispheres such rise of temperature is of very serious import, and usually ends in death. Lesions in the pons commonly produce high fever, even when recovery takes place.

The initial symptoms of cerebral hemorrhage may vary decidedly from those given. If the clot be in the medulla, the patient may fall unconscious and die from arrest of respiration inside of two or three minutes. If the hemorrhage be small, and be situated in a not very sensitive portion of the brain, the only manifestation may consist of vertigo or sudden headache, followed by very brief loss of consciousness, or even by simple mental confusion, during which nausea and vomiting frequently occur. If such an attack occur at night, the patient may pass into it without wakening, and have no knowledge of his illness until an attempt at movement in the morning reveals the hemiplegia.

A very frequent symptom in apoplexy is that of the so-called conjugate deviation of the eyes and head. In the majority of cases the eyes and head are drawn powerfully towards the lesion,—that is, away from the paralyzed side of the body; but in exceptional cases the deviation is in the opposite direction, the face looking away from the lesion. The law formulated by Vulpian and Prevost, that in lesions of the hemisphere the head is drawn towards the lesion and away from the paralysis, whilst in lesions of the mesencephalon it is drawn away from the lesion and towards the paralysis, certainly has exceptions. Conjugate deviation may occur without the loss of consciousness; it is commonly accompanied by quietude of the eyeballs, but there may be marked nystagmus. It is usually fugitive, in most fatal cases disappearing before death, but it has persisted for months or years. The muscular irritation which shows itself in the symptoms just spoken of may also give rise to a general rigidity of the muscles which is especially apt to occur in the side opposite the hemisphere. During the condition of unconsciousness the knee-jerks and other reflexes are usually abolished.

When the patient survives twenty-four hours in a condition of coma, or even, in exceptional cases, with return to consciousness, a reaction may occur with marked evidences of brain-irritation. At such time the rigidity of the extremities is pronounced, and is often associated with heightened reflexes. It is now that the trophic changes appear, the most serious being the so-called decubitus. This begins usually on the buttock as a patch of erythema, dark red or violet in color, disappearing momentarily upon pressure, but soon giving origin to vesicles or bullæ whose contents are usually opaque and bloody. The bullæ soon pass into ulceration, leaving a raw, bluish or violet surface with swelling and sanguinolent infiltration of the tissues beneath. In the course of a few hours the ulcer becomes blackish, and there is formed a smaller or larger slough, whose separation may lay bare the deeper muscles with the nerve-trunks and arteries, or even the bone. The decubitus is always on the paralyzed side.

It may be that the pulmonic congestion which so commonly complicates apoplexy is the result of trophic irritation, but the fact that it is very prone to be in excess in the lung of the paralyzed side suggests that it may be due to the disordered action of the respiratory muscles.

The motor paralysis which remains behind a cerebral hemorrhage depends for its position and completeness upon the seat and the size of the clot. The ordinary form of the paralysis is a simple hemiplegia, involving the face, arm, and leg. The facial palsy is rarely sufficiently complete to affect the soft palate; the hypoglossal nerve is affected a little less frequently than the facial. The two palsies of face and tongue unite to produce the characteristic thickness of speech of the recent hemiplegic; and whilst, owing to the almost universal escape of the motor root of the trigeminal nerve, there is no loss of the power of mastication, the failure of the cheek-muscles (supplied by the facial) makes mastication difficult from the inability to retain the food between the teeth.

As a rule, the arm is more completely paralyzed than the leg; of the muscles of the trunk the trapezius is the only one which is commonly much affected. The universal escape of the muscles of the eyes, thorax, and abdomen, which are associated in symmetrical movements, is usually explained upon the theory of Broadbent, which is that in the case of muscles which are habitually associated in function there are commissural fibres in active life between the spinal ganglia or nuclei, so that brain-impulses pass over from one nucleus to the other, a right nucleus giving to the left nucleus a portion of the impulse which it has received from the left cortex, and *vice versa*. If this be so, it is evident that when one side of the brain is injured both nuclei may be innervated from the remaining hemisphere. It is possible, however, that the commissural fibres may really exist higher up, uniting the cerebral and not the spinal nuclei, or that these associated muscles may receive nerve-fibres from both the direct and the crossed cerebral tracts, and so be normally innervated from each hemisphere.

Pronounced disturbances of sensation are not common in hemiplegia, but hemianæsthesia, with or without implication of the special senses, may exist. It is evident that, as a cerebral hemorrhage is a focal lesion, instead of there being a hemiplegia there may be a monoplegia; that instead of the paralysis throughout being on one side it may be crossed; or, in other words, that there may be any association of symptoms which is in obedience to the laws of cerebral localization heretofore enunciated.

The after-history of cerebral hemorrhage varies. In favorable cases, with a small clot, the paralysis may disappear in a few days. More commonly partial recovery only is attained. The improvement begins in from a few days to a few weeks, and is commonly greater in the leg than in the arm: so that a hemiplegic often can walk although the arm is perfectly useless. The gait of the hemiplegic is, however, seldom perfect; the leg is dragged, and especially is it prone to be raised from the ground by an elevation of the pelvis rather than of the knee; or both knee and pelvis may be required to raise the foot sufficiently to clear the toes, which droop from the inability of the anterior tibial group to hold them up. In either case the whole limb is usually thrown outward and forward in a semicircle instead of being simply carried forward. The shoulder-joint is commonly the last part of the arm to regain its movements. Owing to the contractures of the muscles, there is in most cases some flexure of the arm at the elbow and at the wrist, whilst the fingers are more or less clenched.

Two forms of muscular rigidity occur in hemiplegia, in each case with heightened reflexes. Early rigidity developing immediately after the formation of the clot appears to be the outcome of inflammation around the clot, and is absent in favorable cases. Late rigidity comes on with the secondary degeneration of the pyramidal tract, and is generally believed to be due to an irritation of the pyramidal fibres similar to that which occurs in lateral sclerosis. This seems to be correct, although the theory that the contractures are produced by the habitual position taken by the paralyzed limb has a certain degree of plausibility. The latter explanation does not, however, account for the increased activity of the reflexes which always accompanies late rigidity. Atrophy of the paralyzed muscles is usual after many years, but is not of a degenerative character, so that the paralyzed muscles long retain their normal relations to electricity. How far the joint-inflammation, especially in the shoulder, and the peculiar enlargements which sometimes occur in the peripheral nerve-trunks of the paralyzed side, should be looked upon as trophic, is uncertain. In rare cases rapid and pronounced atrophy of the muscles follows a cerebral hemorrhage, and may be remarkably out of proportion to the degree of the paralysis. According to Quincke, it may continue after the paralysis has disappeared, and is therefore not an atrophy of inaction. The fact that there is neither qualitative nor quantitative alteration of the electric excitability of the muscles shows that the atrophy is not due to any disturbance of the lower trophic centres of the spinal cord. Quincke believes that the brain contains nerve-fibres which have distinct relations with nutrition, and which pass through the hinder end of the internal capsule.

After cerebral hemorrhage, especially in children, various motor disturbances may occur, many of which have been commonly spoken of as distinct diseases, such as athetosis, tremors, choreiform movements, etc. They are no more diseases than is the late rigidity of the muscles, but the importance of the late results of cerebral hemorrhage in childhood warrants special notice of them. (See page 508.)

A peculiar very slow arthritis, first pointed out by Charcot, may develop in hemiplegia, and is often mistaken for chronic rheumatism, from which it pathologically differs in having a tendency to the formation of purulent effusion and to the destruction of the cartilages and bone. During life the trophic arthritis is to be recognized by the following points: first, the hemiplegic arthritis develops about the time at which late muscular contractures usually come on, and, at least in the early stage, is limited to the paralyzed side; secondly, the pain may be moderate, but the tenderness to touch or to movement is excessive; thirdly, the swelling, which is pronounced, develops rapidly, and is accompanied by distinct œdema, with pitting on pressure.

Diagnosis.—The diagnosis between hemorrhagic and thrombotic apoplexy having been sufficiently dwelt upon (see page 496), in the present paragraph the word apoplexy is used in its widest significance. Whilst in private life, with a full history of the attack, the recognition of an apoplexy is usually easy, in public practice, without a history of the case, mistakes may be easy, especially when drunkenness masks the symptoms of the cerebral lesion. Very careful examination of the head for possible traumatism in such cases should first be instituted. Whenever by shaking, shouting in the ear, etc., a drunken person cannot be aroused, the probabilities of cerebral hemorrhage are strong enough to justify the temporary diagnosis. A very careful examination should be made for evidences of paralysis. Its existence warrants the diagnosis of apoplexy.

Unequal pupils may be produced by poisoning,* but are *prima facie* evidence of apoplexy. Drawing of the face to one side is, of course, decisive, and if in breathing the air comes out with a sort of puff and pulling of one corner of the mouth, the case is one of cerebral hemorrhage. If the patient be restless, the motionlessness of the arm or leg of one side will usually betray a hemiplegia, or upon raising the extremities and allowing them to fall, those of one side will be found to drop much more heavily than do their fellows.

The absence of demonstrable paralysis is not proof that a case is not apoplexy, as a severe hemorrhage into the brain may produce a general muscular relaxation ; if, however, in such case any measure of capability of being aroused remains, the probabilities of apoplexy are very small. Conjugate deviation or any monoplegic or hemiplegic spasm justifies the diagnosis of apoplexy. A general convulsion may readily be of alcoholic or uræmic origin.

It is sometimes impossible to make an immediate diagnosis between the quiet form of uræmia and cerebral hemorrhage, especially since in uræmia there may be hemiplegia. In every case brought into a hospital unconscious the urine should be at once examined; but, as cerebral hemorrhage often occurs in advanced kidney disease, care is necessary in interpreting the results of such examination. A distinct rise of temperature makes the diagnosis of simple uræmia very improbable unless there be a series of very violent convulsions. In doubtful cases the physician should be cautious in making positive statements as to the nature of the attack.

The recognition and localization of a meningeal or of an extra-dural hemorrhage after head-injuries are of great importance and difficulty. Whenever symptoms of cerebral compression develop, and still more imperatively whenever convulsions, paralyses, inequality of the pupil, localized spasms, or other evidences of cortical irritation or loss of power appear, shortly after a blow upon the head, the probabilities of a meningeal hemorrhage are sufficient to demand trephining. The temperature may be subnormal or as high as 104° F. The difficult cases are those with irregular symptoms : thus, the appearance of the hemiplegia has in reported cases been delayed for a week ; further, large clots have been found after death in the membranes although during life there were no motor symptoms. If in any case after a head-injury lost consciousness is regained and after a short time again lost, an operation should be performed at once, whether there be or be not convulsions. When there are no localizing symptoms it may be impossible to determine whether the clot exists at the seat of the blow or upon the opposite side. Under such circumstances trephining should be practised first at the point of violence, then, if nothing be found, upon the opposite side. As the middle meningeal is the artery especially liable to rupture, in a doubtful case it may be the best practice so to operate as to secure it.

---

* I have seen them in opium poisoning. (H. C. Wood.)

TREATMENT.—The treatment of a syncopal case of cerebral hemorrhage is distinctly different from that of the congested or sthenic form, and as these two varieties of attack grade into each other, so must the practitioner in the treatment of individual cases alter and adapt his measures of relief. In either case it may be a matter of importance, as asserted by Bowles, to place the body in a semi-sitting position upon one side, to prevent the entrance of the secretions into the lungs, and also to lessen the danger of the falling back of the tongue and consequent interference with the respiration. In congestive apoplexy, with full, strong pulse, or small, wiry pulse, free venesection should be immediately practised; the blood should be taken, with the patient in a sitting posture, from a large orifice in the vein, as rapidly as possible. The amount should vary with the case, but usually from one to one and a half pints may be abstracted, the flow being permitted until the pulse is distinctly softened. In very strong cases the lancet may be followed by a cautious administration of aconite to keep down any tendency to reaction, with its attendant rise of blood-pressure. In syncopal cases of apoplexy venesection does no good, and, in case the lesion be a thrombus, may possibly do harm by increasing the tendency to clotting of the blood. It is especially in syncopal apoplexy that compressing the carotids, as recommended by Horsley and Spencer, might be tried. We can hardly conceive of a case in which the full operative procedure of tying the carotid with a ligature would be justifiable.

In either form of apoplexy it is customary to attempt to control the hemorrhage by cold to the head—especially in the form of the ice-bag—and by sinapisms to the wrists, calves, and ankles. The efficiency of any of these means is extremely doubtful, but as they represent about all that can be done, and as it is usually essential, for the sake of those who are around the patient, to appear to be doing something, the treatment should be assiduously practised. In all cases of apoplexy a drastic cathartic should be given at once, partly because the symptoms are often aggravated by fæcal retention and partly because there is reason for believing that counter-irritation throughout the alimentary canal has some influence in drawing blood from the brain. Of all the drastics croton oil is the most generally serviceable; from one to two drops should be given in a tablespoonful of water, or be placed upon the tongue if there be difficulty in swallowing.

Stimulants must be used in cases of apoplexy with the greatest reserve; any drug which increases the arterial pressure certainly has in it great potentialities for harm, and it is doubtful whether a stimulant ever does any real good. A cerebral hemorrhage is not distinctly depressing to the heart or to the vital forces unless it be in overwhelming amount, and under any circumstance if the patient would die without stimulants it is almost certain he will die with them. Only when there can be no shadow of doubt as to the failing of the heart should stimulants be given. In all cases of severe apoplexy care should be exercised to prevent, if possible, the formation of decubitus or acute trophic bed-sores. There is always danger that in the hurry and alarm of an apoplectic attack the patient may be severely injured by mustard plasters being allowed to remain on too long, and especially by being burnt with bottles of hot water, etc. In health warning sensations prevent accident; in apoplexy they are absent.

No drug will control the overpowering elevation of temperature which often occurs in mortal apoplexy, but in the slight fever of reaction aconite with antipyrin may be used with advantage. During this reactionary stage the patient, unless very feeble, should be kept for twenty-four or more hours without food, and then be given only milk or animal broths. It is often wise to administer frequent doses of calomel, whose purgative and antiphlogistic actions are alike beneficial. After recovery from the immediate effects of the apoplexy minute doses of corrosive sublimate (one seventy-fifth of a grain three times a day) or of potassium iodide (two grains) may be given continuously for some weeks, in the faint hope of aiding in the absorption of the clot. It is very uncertain how far these alteratives are sorbefacient, but in minute dose they are tonic rather than depressant and afford moral support. During this period the food of the patient should be light; absolute freedom from excitement should be enjoined, and no mental effort whatever allowed. Laxatives are to be given freely. It is essential that neither strychnine nor electricity be used at this time. The gentlest massage may be allowed, and is often comforting. After from six to twelve weeks, however, electrical treatment may be employed, the faradic current being the one always selected, and the application being confined to the paralyzed muscles. The application of electricity to the head itself never does any good, and, if currents sufficiently strong to reach the brain be used, may do harm. The value of electricity at any stage of hemiplegia is very slight; it has no effect upon the real lesion; all that it can accomplish is to maintain the health of the muscles. As, however, the muscles, except in very rare cases, are in hemiplegia still under full trophic influence, the only tendency in them towards wasting is due to inaction, and therefore develops very slowly. If in any case of hemiplegia which presents itself for treatment it is found that the muscles are soft and sluggish in their electrical reactions, distinct improvement under the use of electricity may be expected, for it is possible that at such time the amount of repair to the brain is greater than would appear from the symptoms, because the muscles themselves are out of health. The indefinite use of the faradic current in hemiplegia can at best only amuse the patient, and should be carried out, if at all, by an attendant, and not by the physician. Strychnine is a classic remedy in hemiplegias. It has, however, no distinct specific action, but is useful simply by its tonic influences, or possibly in rare cases it may stimulate the motor ganglia of the cord when insensitive from want of use.

In most cases there is a permanent loss of vital power and in the capability for mental action after a cerebral hemorrhage, so that the patient requires habitually the use of measures tending to the building up of the general health, including abstinence from too much work.

As death frequently occurs in apoplexy from arrest of respiration at a time when the circulatory forces are full of power, life may often be prolonged by artificial respiration. We have seen the vigor of the heart maintained for hours after the total cessation of natural respiration, and when there is a minute hemorrhage into the medulla life may be saved by artificial respiration.*

CEREBRAL PALSY OF CHILDREN. INFANTILE SPASTIC PARALYSIS.

DEFINITION.—Paralysis in children dependent upon organic brain-lesion, usually taking the form of a monoplegia or a hemiplegia, and associated with contractures and heightened reflexes.

ETIOLOGY.—Cases which are here grouped together have as their starting-point various brain-lesions, which are followed in the course of years by pachymeningitis, scleroses, atrophies, and other secondary brain-changes, with consequent symptoms of palsy and irritation. The most frequent of all these primary causes is cerebral hemorrhage, which may occur before birth, is very often induced during hard labors, often by asphyxia or by the use of the forceps (usually meningeal, giving rise to sclerosis of the convolutions), or may happen at any time during early life. A cerebral hemorrhage very often in the child causes a convulsion, whilst a convulsion may induce a hemorrhage : so that cerebral paralysis in the child frequently dates from a convulsion or from the paroxysms of a whooping-cough. *Porencephalia* has been frequently found in autopsies upon old cases of infantile cerebral palsy. This lesion, first described by Henschl, consists in the presence of cortical cysts communicating with the arachnoid spaces and penetrating deeply into the brain, even as far as the ventricles. The nature of the primary attack in such cases is obscure. It may be, as suggested by Strümpell, that it is a *polioencephalitis* allied to poliomyelitis, but attacking the cerebral instead of the spinal cells.

---

* In a case which H. C. Wood saw with Edward Martin, the symptoms were sudden unconsciousness and complete general muscular relaxation, followed in a little while by an entire arrest of respiration, accompanied by dropping of the tongue and jaw. Dr. Martin, being present at the moment of arrest of respiration, at once commenced artificial respiration, and kept it up steadily for three-quarters of an hour, when attempts at breathing began. It was necessary, however, for nearly two days for an attendant to hold forward the jaw and tongue by raising the ramus of the jaw upward and forward, in order to prevent mechanical asphyxia by closing of the glottis. The final recovery was complete.

SYMPTOMATOLOGY.—The symptoms of cerebral paralysis vary with the nature and the seat of the lesion. Idiocy, epilepsy, disorders of sensation, may be present in any degree of severity or may be altogether absent. Athetosis, choreic movements, tremors, nystagmus, often but not always accompany the paralysis. The latter is often hemiplegic, not rarely monoplegic, and in many cases is irregularly or symmetrically diplegic (*i.e.*, on both sides of the body). If the diplegia attacks especially the legs, a spastic paraplegia results, with its typical crossed-leg progression.

The only symptoms which are common to all cases of infantile spastic paralysis are partial or more rarely complete paralysis, with marked contracture of the muscles, heightened reflexes, and preservation of the electro-contractility for a great length of time.

DIAGNOSIS.—The symptoms of spastic paralysis are so characteristic that there can usually be no difficulty in the diagnosis. The pseudo-rigidity of Osler, evidently a form of tetany, is at once distinguished by its development after prolonged illness, rickets, etc., and by the intermittency and lack of permanency of the contractures.

TREATMENT.—There is no specific medicinal treatment for spastic paralysis. The general health and nutrition of the child should be maintained in every possible way, and, when the local symptoms are not very severe, attempts should be made to lessen the disablement by massage, by stretching, by passive movements, and by mechanical and surgical measures for the correction of deformities. In our opinion, surgical operations upon the brain itself are unjustifiable.

DISEASES OF THE BRAIN.

ACUTE HEMORRHAGIC ENCEPHALITIS.

DEFINITION.—An acute inflammation of the brain, attended with numerous minute hemorrhages.

ETIOLOGY.—Acute hemorrhagic encephalitis appears to be always due to some primary infection. It has been especially noted in epidemic influenza, and has been recorded after cerebro-spinal meningitis and ulcerative endocarditis.

MORBID ANATOMY.—The lesion is usually localized. The part affected is swollen, extremely hyperæmic, and somewhat œdematous. The capillaries are intensely congested, and surrounded with microscopic hemorrhages and masses of exuded white corpuscles. The nerve-elements show evidences of rapid degeneration.

SYMPTOMATOLOGY.—In very acute cases, after one or two days of headache, giddiness, and malaise, stupor develops suddenly, and deepens very rapidly into profound coma, with fixed pupils, rapid respiration and pulse, and ascending temperature, ending in death in forty-eight hours. In less severe cases the coma is less profound, and consciousness may return, when various focal symptoms, according to the seat of the lesion, may appear.

PROGNOSIS.—Death is the usual end of the stormy cases; if consciousness return, recovery with more or less impairment of function may take place.

TREATMENT.—In sthenic cases Oppenheim asserts that blood-letting with free after-use of calomel has been followed by recovery. In feeble cases cold may be used to the head and counter-irritation to the nape of the neck and to the lower extremities.

Under the name of *polioencephalitis superior* there has been described by Gayet, Wernicke, and others a form of hemorrhagic encephalitis occurring mostly in chronic alcoholism, sometimes after influenza, and located in the neighborhood of the floor of the third and fourth ventricles and in the gray matter of the upper cervical cord. The prodromes, general symptoms, and course are similar to those of hemorrhagic encephalitis in general, except that the temperature remains near the norm, and paralysis of the eye-muscles appears early and rapidly deepens into a complete ophthalmoplegia, with in most cases an optic neuritis. A chronic form of the disease, with the eye-symptoms of the acute disease and with a disordered gait like cerebellar titubation, has been recorded.

Hypertrophic Encephalitis.—In 1868, Hayem described under the name of *sclerotic encephalitis* a form of brain-inflammation which may be either diffused or localized, and which is characterized by swelling and discoloration of the part affected, accompanied by great engorgement of the blood-vessels, whose perivascular sheaths are also stuffed with leukocytes. Grasset believes this affection to represent simply a stage of hemorrhagic encephalitis, but in view of the cases recorded by Knags and Brown it appears probable that there is an encephalitis distinct from the hemorrhagic variety, in which there is a subacute inflammatory increase of the neuroglia, with engorgement and dilatation of the perivascular spaces, ending in degeneration of the nerve-cells and atrophy of the tissues. In the majority of these cases there appears to be a history of traumatism. The symptoms during life have been prolonged apathy, deepening into a complete torpor of mind and body, loss of flesh, lowered temperature, irregular spastic contractions, and partial paralysis. There is no specific treatment.

SUPPURATIVE ENCEPHALITIS.

DEFINITION.—A suppurative inflammation of the brain.

ETIOLOGY.—Suppurative encephalitis may be of septic origin, and hence occurs as a secondary complication of various suppurative diseases. It also occurs after the various specific fevers, especially influenza. More frequently it is the result of a traumatism, or of the extension of inflammation from a neighboring part. As veins run directly from the brain over the petrous portion of the sphenoid, extension of inflammation by septic phlebitis may occur in suppurative otitis. A propagation of the disease-process through the lymphatics or by direct contact from the bone through the dura mater to the brain is, however, more common.

MORBID ANATOMY.—Suppurative encephalitis may exist with or without suppurative meningitis, and may be diffused or localized in solitary or multiple abscesses. The abscess may be with or without distinct capsule, according to its age, and may contain reddish-white or greenish pus, or in very old cases a yellowish, desiccated, caseous matter. In four-fifths of all cases the abscess is solitary. When it has followed sepsis or traumatism it is commonly situated in the frontal lobe and the centrum ovale; when it is due to ear diseases it is usually either in the temporo-sphenoidal lobe or in the cerebellum.

SYMPTOMATOLOGY.—The brain-abscess, especially if situated in a frontal or lateral cerebellar lobe, may remain latent or give rise to simple headache and other obscure symptoms until it suddenly destroys life by bursting. Usually, however, violent headache, with vomiting, anxiety, mental confusion which in some cases amounts to an acute delirium, and general convulsions, are produced. If the abscess involve localizing centres, disorders of the pupil, of the movements of the eye, hemiplegic or monoplegic paralyses or spasms, disorders of the special senses, and other focal symptoms, may be present in accordance with the seat and the extent of the lesion. Owing to the increase of the pressure, optic neuritis almost always exists if the abscess is large. With these symptoms is usually associated irregular fever, with chills and sweating.

DIAGNOSIS.—In the diagnosis of brain-abscess it is first important to recognize a sufficient cause. An abscess may follow an injury without there being any bone-lesion. The interval is usually from one to two weeks, but may be longer; indeed, authentic fatal cases are on record in which the abscess did not manifest itself for years after the injury. In some cases of simple otitis media symptoms simulating meningitis and even abscess, such as headache, fever, and even double optic neuritis, are said to occur without abscess. Nevertheless, if persistent headache, with vomiting, and optic neuritis or other evidences of brain-pressure develop after a head-injury or during an otitis media or a septicæmia, the probabilities of the existence of a brain-abscess are so strong that they must be acted upon, unless reasons can be found for believing that there is a syphilitic or other form of brain-tumor. In such a case the occurrence of chills, fever, and sweating would strongly corroborate the diagnosis. It is a matter of great practical importance to locate the abscess. Abscesses in a frontal cerebral lobe and in a lateral lobe of the cerebellum produce no localizing symptoms; this is also true of the temporal sphenoidal lobes, a very common seat of abscess, unless the abscess be of sufficient size to involve the motor zone or the centres of speech. Disturbance of equilibration in a case of brain-abscess points to the middle lobe of the cerebellum.

PROGNOSIS.—The prognosis in a case of brain-abscess which cannot be evacuated is absolutely fatal; recovery may occur under surgical treatment.

TREATMENT.—In all cases in which the existence of a brain-abscess is probable, trephining should be at once resorted to. As the procedures to be adopted belong to surgery rather than to medicine, we shall not discuss them.

HYDROCEPHALUS.

DEFINITION.—A condition of the brain characterized by accumulation of liquid in the ventricles.*

ETIOLOGY.—Hydrocephalus may be primary or secondary, due to interference with circulation in the straight sinus or in the venæ Galeni, or to stoppage of the foramen of Magendie or of Monro by exudation or tumor. Primary hydrocephalus may be congenital or developed shortly after birth. It is commonly connected with arrest of brain-development, but is in many cases similar in its origin to hydrocephalus of later life (so-called secondary), the difference being that it is not possible to demonstrate the original lesion in the grossly altered brain. The original causes of primary hydrocephalus are not known.

MORBID ANATOMY.—The ventricles are enormously distended with a colorless liquid (from a pint to eight quarts) containing mucin, fibrin, albumin, succinic acid, urea, cholesterin, etc. (Hilger.) The brain-substance is softened, œdematous, and thinned. The cranium may be very thin.

SYMPTOMATOLOGY.—The characteristic symptom of early hydrocephalus is the enormous enlargement of the head, with distended fontanelles, prominent eyes (exophthalmos), and an, apparently, ludicrously small face. Great muscular weakness, imperfect intellection, nystagmus, paresis of the ocular muscles, contractures, epileptic attacks, and exaggerations of the reflexes, are ordinary symptoms.

PROGNOSIS.—In congenital hydrocephalus death may be expected inside of four years, according to the severity of the symptoms. When disease develops after birth the case is more protracted, and may last into adult life.

DIAGNOSIS.—In rachitic macrocephalus the head is squarer than in hydrocephalus, and has a flattened vertex and non-bulging fontanelles. In simple cephalic hypertrophy the enlargement is chiefly occipital, in hydrocephalus it is chiefly frontal.

TREATMENT.—Repeated minute tappings of the ventricle, with gradual compression by means of broad straps of adhesive plaster, are generally used, but it is doubtful whether surgical interference ever does any good. The wisest course is to struggle only for euthanasia.

---

* Hydrocephalus as here defined is the *hydrocephalus internus* of authors: in cerebral atrophy fluid may accumulate between the dura and the pia mater, constituting the *hydrocephalus externus* of authors.

ACUTE PERIENCEPHALITIS.

DEFINITION.—An acute disease of the brain, attended by stupor, wild delirium, general disturbances of the psychic functions and of the motor functions, and fever; dependent upon acute hyperæmia and subsequent inflammatory changes in the cerebral cortex.

SYNONYMES.—Acute peripheral encephalitis; Phrenitis mania gravis; Typhomania; Acute delirium; Delirium grave; Bell's disease (Luther Bell).

ETIOLOGY.—Idiopathic non-septic periencephalitis may be produced by profound grief, protracted anxiety, especially when accompanied by great overwork, partial starvation combined with the gnawing anxiety of deep poverty, and also, it is affirmed, by sunstroke. It certainly may develop without apparent cause during locomotor ataxia, or may occur as an exacerbation of chronic periencephalitis. It is especially frequent during pregnancy following seduction; and the cases of death from alleged acute hysteria which have taken place after rape and various intense emotional disturbances have probably been instances of the disorder. Those cases of acute periencephalitis which have been reported as produced by acute fevers, traumatism, and various purulent diseases were evidently of septic origin.

MORBID ANATOMY.—The lesion of acute periencephalitis is an acute inflammation, solely or chiefly confined to the cerebral cortex and its membranes, and first appearing as an excessive hyperæmia, which is rapidly followed by escape of the white blood-corpuscles and filling up of the lymph-spaces both of the pia mater and of the cortex by leukocytes, which finally invade also the periganglionic spaces. Not rarely the whole cortex is œdematous, and minute apoplectic hemorrhages may occur. As to the ultimate nature of the inflammation and its cause there has been much discussion, but it seems certain that periencephalitis may be of various origin. It may be septic, as it has occurred in septic subjects, and as various forms of pyogenic organisms have been detected in the inflamed tissues by Braden Kyle, James R. Hunt, and others. Rasori affirms that he has isolated from a case of periencephalitis a hitherto undescribed bacterium, which injected into rabbits produced septicæmic death. On the other hand, periencephalitis may exist without the presence of any micro-organisms in the brain or the general system. It would appear, therefore, that the existence of two varieties must be recognized (with the possibility of a third): first, septic periencephalitis; second, idiopathic periencephalitis, produced by emotional causes; third, and at present very doubtfully, a bacterial periencephalitis, due to a peculiar specific organism.

SYMPTOMATOLOGY.—The onset of acute periencephalitis may be abrupt, but usually there are some prodromes, such as short periods of impaired consciousness, especially upon waking in the morning, or brief nocturnal attacks of wandering delirious restlessness, or mental excitement, or pronounced mental aberration. We have seen during the day a patient warn those about him that he would kill them during the night in his period of delirium. A complete insomnia rapidly develops, and soon the delirium becomes constant: the night and the day are passed in violent excitement, with a perpetual outpouring of incoherent speech and a fury of fighting and destructiveness.

Hallucinations and half-formed delusions are present, and often bear a close relation to the cause of the attack. The abandoned mistress will in her ravings recount her past shame and present agony. The business-man will be perpetually occupied with an incoherent jumble of business transactions. Almost invariably along with the delirium there is great physical restlessness, which grows more intense until it causes the patient to leap from his bed and to attempt to run away. Very commonly violent assaults are made upon the attendants. Convulsions are rare. The delirium may at first be not continuous, occurring only at night, or at least be interrupted by brief intervals of comparative rationality during the daytime. Finally, however, there is persistent intense mania.

During the whole course of the disease the pulse is rapid; if in the beginning it possesses a show of force it is in fact soft and compressible. Food is usually absolutely refused. The fever is pronounced, and the temperature may reach 106° F. According to our observations, the temperature varies with a stormy irregularity which is almost characteristic, rising and falling many degrees many times during the twenty-four hours. Its variations are connected with the mental and physical excitement of the patient, maniacal outbursts producing an immediate rise of the temperature. In advanced stages the temperature may fall much below the norm. The pupils may be contracted, dilated, or normal. In the course of a few hours to several days the second stage of the disorder develops. There are now quiet coma or else muttering delirious unconsciousness, failing pulse, cool skin, and general evidences of collapse. In the early part of this stage, when aroused, the patient may respond incoherently or perhaps give some slight evidences of comprehending what is said to him, but he rapidly sinks lower and lower until he dies from exhaustion. Early in the disorder the skin becomes very harsh, and finally cyanotic; in the later stages irregular desquamation, or even ulceration, may occur. In a case quoted by Spitzka the anæsthesia was so complete that the patient gnawed off a portion of one of his fingers. Pemphigus-like vesicles, phlegmons, decubitus, gangrenous patches of skin, or gangrenous extremities are occasional complications.

DIAGNOSIS.—As it is probable that acute mania and acute periencephalitis simply represent different degrees of one disease, it is apparent that the diagnosis between the two disorders may be not only difficult but impossible. The points upon which such diagnosis rests are the greater intensity of the symptoms and especially the presence of pronounced fever in acute periencephalitis. Ordinarily in acute mania the bodily temperature does not rise more than a degree above the norm, and Krafft-Ebing affirms that in a maniacal case the temperature of 100.5° F. indicates strongly delirium acutum. H. C. Wood, however, has shown that there exists every grade of temperature in maniacal cases, so that no sharp line can be drawn, either as regards the intensity of the symptoms or the height of temperature, separating clinically the two alleged diseases. Again, an acute periencephalitis may be with great difficulty separated from an acute confusional insanity, in which violent delirium with irregular fever and the rapid development of typhoid symptoms make a picture which cannot be at once distinguished from that offered by acute delirium. Even in the most acute form of confusional insanity, however, there are usually in the first days of the attack some of the peculiar confusion, the excess of hallucinations, or other symptoms that mark confusional insanity. The diagnostic difficulties are further intensified by the circumstance that the causes which produce a confusional insanity in one case may be apparently the same as those which cause acute periencephalitis in another. Thus, the so-called *puerperal insanity*, which is commonly spoken of as though it were one form of disease, may be a violent confusional insanity, or it may be a septic periencephalitis, or it may be a non-septic idiopathic periencephalitis.

Carelessness on the part of the practitioner may lead to a symptomatic delirium being considered as acute delirium. This is especially true of the abrupt maniacal outburst which occurs in latent pneumonia. Such outburst, however, almost without exception takes place in young children or in persons broken down by age or by excessive privation or dissipation, so that the character of the subject should put the practitioner on his guard, and physical examination would reveal at least pulmonic percussion dulness.

PROGNOSIS.—The prognosis of acute periencephalitis is highly unfavorable; at least three-fourths of the cases end fatally. When recovery occurs the mind is almost invariably left in a damaged state. The more violent the symptoms the worse the outlook.

TREATMENT.—The early symptoms and the lesions of acute periencephalitis suggest venesection and local blood-letting as a means of resisting the disease. It may be that in some cases of the disorder these measures are indicated, but we have never met with such a case. As the affection is ordinarily seen, the antiphlogistic treatment must be limited to the local application of cold to the head, to the use of blisters, and to the administration of drastic purgatives. Hyoscine hydrobromate should be given in full doses every six hours (one seventy-fifth of a grain) until physiological effects are apparent. With it, in many cases, can be advantageously combined morphine or chloral. Solivetti asserts that he has obtained extraordinary results from hypodermic injections every eight hours of one gramme of ergotin. Certainly the use of ergot would seem to be indicated, and the severity of the disorder thoroughly justifies the risk of any local trouble from hypodermics. A filtered solution of the official extract of ergot in freshly boiled water may be used, ten grains every three hours. In the later stages of the disorder alcoholic and cardiac stimulants may be employed *pro re nata.*

During the whole course of the disease it is essential that the restraint which is usually necessary be applied in such a way as not to increase the excitement. No one should be allowed in the room except the nurses. Non-irritating nutritious foods must be administered in as large amount as possible: milk, raw eggs, strong animal broths and essences, thickened soups and such liquid foods, are to be employed, and in many cases must be given with the stomach-tube.

CHRONIC PERIENCEPHALITIS.

DEFINITION.—A chronic disease, dependent upon a peculiar inflammatory degeneration of the cerebral cortex, which gives rise to change of character, progressive mental deterioration, with delusions of grandeur, emotional exaltation or emotional depression, occasional maniacal outbreaks, and epileptic attacks, with progressive physical deterioration, as shown by irregularity of the pupils, disorder of speech, and loss of control over the movements of the hands and legs,—all symptoms finally being swallowed up in a complete paralysis of intellection and of voluntary motion.

SYNONYMES.—Paretic dementia; General paralysis of the insane; Paresis; Dementia paralytica; Periencephalo-meningitis.

ETIOLOGY.—General paralysis is not a distinctly hereditary disease, is rare in females, and is remarkably frequent in military and naval officers. In civil life the disease is most frequent between forty and fifty years of age, and very rare under thirty or over sixty. According to Mickle, in soldiers and sailors the average age is about thirty-three. These peculiarities, however, are probably simply the outcome of differences in the degrees of exposure to the great causes of the disorder, which are alcoholic and sexual excesses, syphilis, and a long-continued emotional strain, such as that of excessive ambition or excessive anxiety, factors whose influence is increased by overwork. According to Mendel, seventy-five per cent. of the cases of general paralysis have a distinct history of syphilis, whilst in other insanity the percentage is about eighteen. As the disease is not gummous,—*i.e.*, specifically syphilitic,—it is evident that the relations between it and syphilis are parallel to those which exist between locomotor ataxia and syphilis. It is a post-syphilitic affection. It also occurs not rarely as a late complication in locomotor ataxia, and locomotor ataxia or other spinal sclerosis frequently develops in the paralytic dement. Sunstroke and traumatisms are asserted by authorities, with doubtful correctness, to be among the exciting causes of periencephalitis.

MORBID ANATOMY.—Various secondary or complicating alterations of the skull, the brain, and its membranes are found in old cases of periencephalitis. The characteristic lesions are, however, in the cerebral cortex, which is usually discolored, sometimes firmer, sometimes softer, than normal, often containing minute cysts varying in size from that of a pin's point to that of a millet-seed. Microscopic examination reveals degeneration or perhaps complete disappearance of the ganglionic cells and a peculiar alteration of the white fibres, which renders them much more apparent than in the healthy brain, besides pronounced degeneration of the neuroglia and large numbers of peculiar many-processed connective-tissue cells (Deiters's or spider-shaped cells). The blood-vessels are usually injected, and altered in character, with distention of the adventitial lymph-spaces. The spinal cord is very frequently degenerated. Changes in the sympathetic ganglia have also been noted by recent investigators.

In regard to the original nature of the changes which have just been described, there are two distinct views held by alienists: the one which seems to us most probable is that the disease is a diffused interstitial cortical encephalitis, in which the connective tissue and the blood-vessels are primarily affected. In accordance with the second view, however, the process is a diffused parenchymatous inflammation, which begins in the nerve-elements and secondarily involves the neurogliar tissue.

SYMPTOMATOLOGY.—For the purposes of discussion we shall recognize the four stages commonly made by writers on General Paralysis; but it must be understood that these stages in their time-relations vary almost indefinitely, and that in many cases some of them never appear, or at least are so very brief and indistinct that they pass unobserved.

The symptoms of the prodromic stage of chronic periencephalitis may be indistinguishable from those of an ordinary cerebral neurasthenia, consisting of loss of power of fixing the attention, apathy, inability for mental exertion, and some emotional depression. If to these symptoms be added distinct vaso-motor phenomena, such as facial congestion, headache, vertigo, tinnitus aurium, temporary hemianopsia and other disturbances of vision, and a slight alteration in the character of the patient, there is sufficient ground for fear. Krafft-Ebing gives as almost characteristic the peculiar alteration of the relations of the patient to time and space, which renders him exceedingly unpunctual or causes him at times confusedly to lose himself in well-known streets. Although this stage is so often overlooked, yet after the disease has declared itself the books and correspondence of the business-man or the office-histories and records of the professional laborer will, in their loss of accuracy and dignity and in their general evidences of failing power, usually afford a history of a slowly progressive mental degeneration.

In the second stage the mental aberration is pronounced and distinct, but the motor disturbances of the more advanced disorder are wanting. This stage of the disease is usually short, but we have seen it persist for more than a year without the slightest failure of the general physical powers.

The third stage of the disease is that in which the motor symptoms become marked, as shown in inequality of the pupils, flabbiness and loss of expression of the face, disorders of articulation, general loss of endurance, and, it may be, distinct paresis of the extremities.

The fourth stage of the disease is that in which the dementia is complete and the general loss of muscular power very great, the patient being reduced to a feeble automaton.

The mental phenomena of paretic dementia may be considered for the purposes of study as conforming with four types, but every grade of cases exists between these types, and the same individual cases may at different periods represent two or more of them. In the first form of paretic dementia (*vulgo*, softening of the brain) there are no marked emotional disturbances or delusions, but simply a progressive failure of the mental and physical faculties, which ends after a period of childishness in complete dementia. In the second form of the disease there are delusions of grandeur or expansive delirium ; these delusions may be of the most intensely dominating character or so mild that they may be readily overlooked ; they may be replaced by simple emotional exaltation, so that the man is not affected by any depressing circumstances or surroundings, although he may make no assertions of the possession of high faculties or of great power or wealth. Such cases as these may be considered as midway between the first and second types. Maniacal outbursts may occur in any form of paresis, but are especially frequent when there are delusions of grandeur.

The third form of general paralysis is that in which there is emotional depression and even pronounced melancholia with depressive delusions. Not rarely the depressive delusion relates to the person of the patient, who believes himself ill, deformed, or wanting in some member or function. In this way arises the so-called hypochondriacal variety of general paralysis.

The fourth and rarest form of general paralysis is that in which excitement and depression alternate so as to make a periodic or circular insanity. The excitement and depression may abruptly succeed each other, but in some cases are separated by a lucid interval, so that the whole cycle will consist of three periods. According to Mickle, such a cycle differs from that of ordinary circular insanity in the order of sequence, which is—first, excitement ; second, calm ; third, depression.

Major and minor epileptiform convulsions may usher in a general paralysis or may occur at any time during its course. The seizure may consist of a sudden pallor with mental confusion, or of a momentary unconsciousness, or of a dilatation of the pupils with drawing of the head, or of a sudden fixation of the countenance with an outpouring of cold perspiration, or of an automatic repetition of coherent or incoherent phrases. Such an attack occurring in persons who have been subject to the causes of a general paralysis and are of the proper age should always excite alarm.

Not rarely epilepsy in general paralysis takes upon itself the Jacksonian form, the convulsion being limited to isolated groups of muscles. or to one side of the face, one leg, or one arm, or being hemiplegic. Usually the attack begins with an aura, which is especially apt to be vertiginous. Sometimes the convulsion is preceded for several days by excessive restlessness, tinnitus aurium, and great psychical excitation. In other cases it begins with vomiting. In the advanced disease epileptic fits may be frequent and severe, and the observation of Esquirol that a succession of them frequently closes the scene has received abundant confirmation. When the true epileptic status occurs during a general paralysis the successive convulsions are often very diverse, one being complete, the next partial,—in one the head being drawn to the right, in the next to the left, and so on. After the paroxysms, convulsive tremblings frequently persist in single muscles or in groups of muscles for many hours, and are followed by a more or less pronounced partial palsy. To use the words of Nichol, paralysis follows the convulsion or spasm as the shadow follows the body. During the more severe paroxysms consciousness is always lost, but in mild attacks, and especially when the convulsive movements are more or less local, it may be perfectly maintained ; occasionally it is affected as in hysteria. Sometimes the convulsion may be replaced by an apoplectiform attack. The mental condition of the patient is almost always distinctly worse after a severe seizure.

The most characteristic motor symptom of chronic periencephalitis is an incomplete paralysis affecting all the body and commencing with tremors and disorders of coördination. The first manifestation is usually a loss of control over complicated muscular movements of the hands, so that a man although able to lift many pounds may not be able to write his name. In engravers or other persons whose daily vocation requires great technical hand-skill this loss of muscular control may be the first distinct evidence of a general paralysis.

Inequality of the pupil in the early stage of the disorder is a not uncommon, and in the later stage is a constant, phenomenon. It may be associated with mydriasis or myosis. A similar loss of delicacy of movements in the lips and tongue produces in general paralysis a difficulty of pronunciation, which is especially manifested in the speaking of the lingual and labial consonants and in the syllables of long words. Moreover, as mental power fails there is a corresponding failure in the formation of ideas and of the association of the ideas with the spoken words. Through the failure of mind and of tongue there arises a peculiar stuttering or hesitating, somewhat thick, characteristic speech. There is often an elision of syllables, a dropping out of words, and a marked tendency to fall into a rhythmical speech, giving rise to a peculiar utterance like that of a school-boy scanning Latin poetry, and hence it is spoken of as "scanning speech."

The handwriting in general paralysis very early becomes shaky and irregular, with ill-formed, widely separated lines, and often with a disappearance of the finely graded strokes of correct writing in a common, thick, uncertain line. The mental characteristics of the disorder show in the writing as much as in the speech. The dropping out of letters, the omission or repetition of syllables, and the elision or interjection of words and clauses, are almost characteristic. In medico-legal cases letters often afford most effective testimony.

In periencephalitis there is neither headache nor pain, save as a complication, such as may occur from the development of locomotor ataxia, etc. In the advanced stages increasing numbness passing into analgesia is almost invariable, making it especially necessary to provide carefully against possible accidents to the patient. We have known such a patient scalded to death by getting into a too hot bath.

Violent sexual excitement is often one of the earliest symptoms of general paralysis, but in the progress of the disease it gradually gives way to impotence, excessive libidinousness often persisting after the total loss of sexual power.

Disturbances of temperature, especially a tendency to an evening rise and to irregular paroxysmal alterations of temperature without apparent cause, are very frequent in general paralysis. Especially characteristic is a liability to the production of violent fever by very slight cause. The epileptic paroxysm may occur without alteration of temperature, but is usually accompanied by a distinct rise, which frequently precedes by eight or ten hours the convulsion and lasts many hours after it. Both Mendel and Westphal have recorded cases in which a continuing epilepsy was followed by a violent fall of temperature. Usually, however, a decided fall of temperature in an attack of unconsciousness marks the occurrence of a true apoplexy. In advanced dementia the temperature is commonly subnormal, and often different upon the two sides of the body.

DIAGNOSIS.—Although it may not be possible to make a positive diagnosis in the prodromic stage of general paralysis, the recognition of the probable nature of the attack may be of supreme importance for the good of the patient and of his estate. Sudden changes of character, accompanied by a tendency to sexual crimes, or to the formation of large plans beyond the means of the patient, or to excessive expenditure of money, if occurring in a man of middle age with a history of past syphilis, or of abuse of alcohol, or of excessive mental or emotional work, should be sufficient ground for practical action, especially if they be associated with great hilarity, *bien-être*, or emotional or mental exaltation.

The diagnosis between paretic dementia and syphilitic cortical disease is frequently not possible in the beginning of the attack. The occurrence, however, of headache or of localized palsies points strongly to syphilis. (See page 530.) When the mental disorder precedes, as it occasionally does, for several years the distinct physical symptoms, the diagnosis is between a dementia paralytica and a pure insanity. Very often in these cases either irregularity of the pupil or loss of power of executing fine movements, such as those of writing, buttoning, dancing, etc., can be detected if the case is one of periencephalitis. Of almost equal importance is a distinct and progressive failure of memory, which is not a feature of the earlier stages of the pure insanities.

PROGNOSIS.—Usually death occurs in general paralysis in from two to three years. Cases lasting four years are not extraordinary, whilst some have passed the decennium. Very acute cases may terminate in three or four months. The prognosis is always bad. It is probable that in those cases in which recovery is claimed the diagnosis was at fault.

TREATMENT.—From the despairing outlook of general paralysis it is justifiable in the beginning of an attack to use local blood-letting by leeches, the actual cautery, and other counter-irritations to the nape of the neck. Especially should this be done if apparently cortical disease has followed traumatism or sunstroke and is therefore in all probability largely meningeal.

As the early diagnosis between this disease and syphilis of the brain is often not possible, acute antisyphilitic medication in many cases should be essayed. Restraint is in the early acute stages of the disease always necessary, and usually very difficult of enforcement outside of an asylum. Very good results in obtaining remissions have been claimed by alienists from the employment of massive doses of ergot. One to two drachms a day of the official extract should be given continually for weeks, unless coldness of the surface or other physiological effects become apparent. Hyoscine, morphine, and sulphonal are useful for overcoming wakefulness or excitement. Massage, moderate bathing, very carefully restricted out-door exercise, very warm clothing, a non-stimulating but abundant and nutritious diet, the avoidance of physical and mental as well as emotional excitement, and in the later stages of the disease great care to protect the patient from his fecal and urinary discharges and to prevent decubitus, constitute in outline the measures to be employed. As fatal pneumonia has frequently been produced in the advanced stages by particles of food getting into the lungs, a liquid or semi-liquid diet should be given.

DISSEMINATED SCLEROSIS.

DEFINITION.—A disease whose essential lesion is nodules or patches of sclerotic tissue scattered through the nerve-centres.

SYNONYMES.—Multiple sclerosis ; Multiple cerebro-spinal sclerosis.

ETIOLOGY.—In the great majority of cases no cause can be assigned. Rarely have several members of one family been affected, and it is affirmed that the disease is sometimes a sequela of exanthematous fever. The subjects may be of any age, but the period of life most liable to attack is from forty to sixty.

MORBID ANATOMY.—The diseased areas are irregular, from two to thirty millimetres in diameter, on exposure to the air reddish gray (in the brain) to grayish white (in the spinal cord), very firm, consisting of connective tissue, free from nerve-elements, or more commonly containing axis-cylinders deprived of their medullary sheaths. The blood-vessels are usually sclerotic, but may be fatty. Any portion of the nerve-centres may be the seat of nodules, which may even form in nerve-roots, and have been especially noted in the optic or other nerves of special sense. The white matter is usually involved to a greater extent than is the gray.

The patches may exist either in the brain or in the spinal cord alone, or, as in the ordinary type, may be cerebro-spinal. The development of the lesion appears to be—first, increase of the neuroglia, with multiplication and enlargement of the nuclei ; then atrophy of the medullary sheaths, with preservation or even augmented volume of the axis-cylinders ; finally, atrophy of the latter. According to Charcot, the long persistence of the axis-cylinder is characteristic of this form of sclerosis, distinguishing it from that which involves nerve-tracts. Taylor asserts that the ganglionic cells are often deeply pigmented when the nodule is situated in the gray matter.

SYMPTOMATOLOGY.—In the great majority of cases disseminated sclerosis is very insidious in its onset and progress. The chief symptoms are a very slowly increasing loss of memory and of general mental and physical power, with vertigo, volitional tremors, defects of speech, various eye-symptoms, and minor nerve-disturbances, according to the seat of the lesion.

The characteristic of the volitional tremor is its cessation during complete rest, such as is obtained by absolute repose and support of every part of the body when lying down. The oscillations are commonly extensive, rhythmical, and slow, from seven to ten per minute ; they are especially pronounced in, but are not confined to, the part which is in action. They usually develop first in the hands, and for a time may exist solely in the upper extremities. The head is early attacked.

The eye-symptoms are squint, with its consequent diplopia ; various irregularities of the pupils ; nystagmus, the movements being usually horizontal, in rare cases vertical ; inability to move the eyeballs in unison ; and atrophy of the optic nerve, with amblyopia, dyschromatopsia, contraction of field, scotomata, transitory attacks of blindness, etc.

The defects of speech vary : there may be simple tremulousness, or thickness of utterance, or scanning speech like that of general paresis.

The vertigo is a constant and often severe symptom, and is usually a true whirling vertigo; in the advanced stages to it may be added the giddiness of diplopia.

The mental disturbance may be simply a loss of power, but there may be delirium of grandeur and hallucinations; melancholia, hypochondriasis, or almost any of the forms of pure insanity, so called, may be simulated.

Paresis, spasmodic contractions, spastic paralysis, muscular atrophies, diminished or excited reflexes, various disturbances of sensation, girdle-pains, fulgurant pains, as of locomotor ataxia, disturbances of coördination, in a word, any of the symptoms which are producible by sclerotic disease of the spinal cord, may be present, according to the position of the nodules in the cord. Even cerebellar titubation is said to have been noted in cases in which the middle lobe of the cerebellum was especially affected.

Apoplectic seizures and epileptic attacks are common, and may end in death; not rarely they are followed by a hemiplegia in which the muscles are flaccid, rarely contracted. The hemiplegia may be accompanied by aphasia or by crossed or direct facial palsy, but passes off in a few days. If death occur in an apoplectic attack, no relation between the symptoms and the lesions can usually be made out.

DIAGNOSIS.—The diagnosis of a typical case of multiple sclerosis is very easy; in children, however, it may be confounded with Friedreich's ataxia, from which it should be immediately separated by the volitional tremor. The tremors appear to be of cerebral origin, and with the mental symptoms are absent when the nodules are confined to the spinal cord. The symptoms of such a purely spinal multiple sclerosis differ from those of a sclerosis affecting certain tracts, in that they do not conform with the function of any one tract. Thus, fulgurant pains may coexist with increased patella reflexes, because of the sclerotic degeneration being present both in the posterior and in the lateral regions; or the knee-jerks may be different in the two sides of the body; or atrophy may exist in certain muscles, with evidences of spastic paralysis in other muscles, and fulgurant pains in other parts, showing the coexistence of scattered lesions in the lateral, posterior, and central portions of the spinal cord. In spinal cases the diagnosis must rest upon the slowness of development and the demonstration by the diversity of the symptoms that there are multiple lesions.

PROGNOSIS.—The course of multiple sclerosis is essentially slow, many years usually being required for the complete development of the disorder. It is affirmed by authorities that a small proportion of the cases recover, but we are inclined to believe that such cases have been instances of mistaken diagnosis.

TREATMENT.—Zinc phosphide and the double gold and sodium chloride have been recommended by various authorities and may be used.

Arsenic, belladonna, ergot, the whole line of the spinal sedatives, have also been lauded from time to time, but there is no sufficient reason for believing that they have value. Strychnine, it is stated, will sometimes arrest temporarily the tremor, but it seems probable that its general influence would be for harm rather than for good. Solanine, according to Grasset, in the dose of one and a half to two grains a day will distinctly lessen the tremor, and we have found hyoscine sometimes to have such action. Massage, electrical treatment, douches, and suspension may all be tried, as in other forms of spinal sclerosis.

INTRA-CRANIAL TUMORS.

DEFINITION.—New formations within the brain.

ETIOLOGY.—The causes of brain-tumors are the same as those of neoplasms situated in other parts of the body.

MORBID ANATOMY.—The varieties of brain-tumor are Glioma, Syphiloma, Sarcoma, Solitary Tubercle, Carcinoma, Fibroma, and Cystic Disease of the Choroid Plexus, with, as rare phenomena, Lipoma, Psammoma, Cholesteatoma, Osteosarcoma, Dermoid and Echinococcus Cysts, and almost all other known varieties of morbid growth. Of these tumors the syphiloma is the most common; next to it in frequency come in adults the glioma, gumma, or sarcoma, in children the solitary tubercle. The glioma is found especially in the cerebral hemispheres and the cerebellum; the tubercle affects chiefly the pons, the cerebellum, and the cerebral cortex. The sarcoma, glioma, and tubercle are capable of quick development, but often grow slowly, and may remain stationary for an almost indefinite period. By direct pressure and irritation, and especially by interfering with large blood-vessels, tumors give rise to secondary changes in the brain-tissue which may involve distant parts and produce localizing symptoms which draw attention away from the original lesions and lead to mistaken diagnosis as to the seat of the tumor. By direct pressure upon the skull tumors situated in the outer parts of the cerebrum may lead to softening of the skull (*craniotabes*), or even to complete perforation (*osteoporosis*). In rare cases deeply seated tumors not in contact with the bone bring about similar changes.

SYMPTOMATOLOGY.—The symptoms of brain-tumors may be divided into general—*i.e.*, those which evince simply a general cerebral disturbance—and focal,—*i.e.*, those which are dependent upon disorders of function caused by the local action of the tumor.

The general symptoms are headache, vomiting, stupidity, giddiness, slowing of pulse, and optic neuritis. Of all these symptoms headache is the most rarely absent. It is usually intense, unyielding to remedies, constant, with paroxysmal exacerbations, especially at night, and intensified by jarring, straining, coughing, or other acts increasing the blood-pressure. It may be localized in the region of the tumor, but usually it is diffuse. The apathy or stupidity is most marked in advanced stages of tumor, but may be an early symptom. It often gives rise to a peculiar slowness and hesitancy in speech and in act, and may be accompanied by pronounced somnolence, which in turn may pass into profound coma. Distinct mental aberration, with hallucinations, may occur in brain-tumor, but is rare. On the other hand, failure of memory and of the other mental powers is common.

Vomiting is often absent, but may be the first symptom. It is especially severe when the cerebellum is involved. The attacks come on without apparent cause, and without connection with the taking of the food. The nausea is not pronounced. Optic neuritis is present in nearly ninety per cent. of the cases; it is usually double. As time goes by, a very pronounced choked disk commonly gives way to atrophy of the nerve.

Except in cerebellar tumors, giddiness rarely amounts to a violent vertigo or to such a disturbance of coördination that the subject is forced to lie down to prevent falling. It is especially apt to come on when the patient suddenly rises from the supine position, or when acts are performed which increase greatly the blood-pressure. It often occurs with vomiting in the acme of a cephalalgic exacerbation.

Epileptic attacks may take the form of petit mal, but are usually accompanied by a general, often very severe, convulsion. True epileptic automatism or destructive mania is very rare, if it ever occurs. When the tumor is in the motor zone the fit usually begins in, and often is confined to, one extremity (Jacksonian epilepsy). An aura may precede an organic convulsion when a special centre is involved. The aura usually consists of a corresponding subjective sensation.

Slowing of the pulse (to even forty per minute) is especially present when the tumor irritates the pneumogastric nucleus, but is often produced by the general brain-pressure.

Fever may be present in brain-tumor, especially when growth is rapid or the pons is involved, and the local temperature over the tumor may be higher than in the corresponding position on the other side of the head. According to Bruns, a peculiar bruit may be sometimes heard over the tumor.

The focal symptoms of brain-tumors vary in accordance with the seat of the tumor, and are so closely in accordance with the rules before enunciated (page 471) that it appears to be a work of supererogation to enter here upon their discussion. Nevertheless, certain brief statements seem justifiable.

Anterior Lobes.—There are no localizing symptoms, unless the olfactory bulbs are involved, as is especially the case in glioma, when subjective sensations of smell become prominent phenomena, and in advanced stages may be associated with more or less complete loss of the sense.

Psycho-motor Area.—Tumors are especially connected with local, monoplegic, or hemiplegic spasms or paralyses, and with Jacksonian epilepsy.

Commonly the local spasms are attended with tingling and with numbness, and it is especially important to observe in any case the exact locality at which this sensory disturbance first manifests itself, as it usually is more confined in its distribution than is the spasm, and therefore more closely represents the part of the psycho-motor area involved in the lesion; if there be remaining temporary paralysis, its position should correspond to that of the numbness. Aphasia is a localizing symptom, but when it occurs in paroxysms it indicates that the centres are rather indirectly than directly involved.

Parietal Lobes.—There are no localizing symptoms, unless the tumor be sufficiently deep to involve the visual tract,—*i.e.*, the conducting fibres coming from the cuneus,—when it may produce hemianopsia, or be situated in the left angular or supra-marginal gyrus, when it may cause word-blindness.

Occipital Lobes.—The only localizing symptoms are disorders of vision, ranging from slight subjective phenomena to complete hemianopsia due to involvement, less or more complete, of the cuneus or its conducting fibres.

Central Ganglia.—Tumors of the optic thalamus and the striate bodies may exist without localizing symptoms, or may produce disturbances varying from the slightest loss of sensation or of motion to complete hemianæsthesia and hemiplegia, according as they encroach little or much upon the internal capsule.

Temporal Lobe.—The only localizing symptoms are disturbances of hearing, especially word-deafness, which has been noted when the first and second gyri were affected.

Basal Ganglia.—Tumors of the corpora quadrigemina produce ocular symptoms, and especially a very early developed optic neuritis; not rarely hydrocephalus results from the pressure. If the crus be involved there may be hemiplegia with or without disturbances of sensation on the same side, and with or without oculo-motor paralysis on the opposite side.

Pons.—Tumors of the pons produce almost every conceivable form of spasm and of paralysis by involving the cranial nerves and the motor tracts going to the limbs, and also by similar involvement of the sensory tracts. The auditory nerve is not rarely paralyzed, with resultant deafness. Facial paralysis, with opposing conjugate deviation of the eyes, appears to be diagnostic.

Medulla.—Tumors involving the medulla may produce partial hemiplegia with hemianæsthesia on the opposite side, or partial paraplegia, or various disorders of swallowing, of respiration, of the heart's action, and of other functions of the nerves situated in this region. Ataxia is said to be a frequent symptom.

Cerebellum.—Tumors of the lateral lobe of the cerebellum, unless by pressure they affect the central lobe, produce no localizing symptoms, but may be suspected when, without localizing symptoms, the general manifestations of brain-tumors are present, and are accompanied by excessive vomiting and by a very early and intense development of choked disk. Tumors involving the central lobe of the cerebellum usually produce intense headache, excessive nausea and vomiting, and a persistent, distressing giddiness, which may be so severe as to confine the patient to a horizontal position or may chiefly consist of cerebellar titubation. (See page 488.) In some cases there is a distinct tendency to fall uniformly to one side, perhaps more frequently backward, or occasionally forward.

The knee-jerk is probably never affected by a cerebral tumor unless the latter be located in the cerebellum or its neighborhood. Under these circumstances the knee-jerk may be wanting, normal, or exaggerated; and it has happened under observation that the normal knee-jerk has gradually disappeared, and then reappeared and increased in activity until much above the norm. These alterations of the knee-jerk occurring in the course of a few weeks or months are probably diagnostic of tumor in the cerebellum or its neighborhood. The explanation is that in the beginning the tumor irritates Setschenow's centres and produces excessive spinal inhibition with loss of knee-jerk, but that as the tumor grows and invades Setschenow's centres these lose their functional power until all inhibition of the spinal cells is ended, in which case there must be exaggeration of the reflexes.

It is a general rule that in basal brain-tumor optic neuritis, paralysis or spasms of the eye-muscles, irregularities of the pupil, nystagmus, or other symptoms due to irritation or to pressure of the nerves of the base of the skull, are present.

DIAGNOSIS.—Of the non-localizing symptoms the most important is optic neuritis; this may occur in acute encephalitis, in multiple neuritis, and in chronic metallic poisonings, especially lead poisoning; but probably in ninety-eight per cent. of the cases it is the result of meningitis, hydrocephalus, or brain-tumor. In individual cases it may be impossible to decide whether the lesion is a basal meningitis or a basal brain-tumor. In syphiloma the exudation is so wide-spread that often it would be as correct to say that there was a syphilitic meningitis as to say that there was a gumma.

The diagnosis of meningitis can usually be arrived at by a history of the existence of its cause, by the rapidity of its course, and by the age of the patient. Hydrocephalus occurring in infants is at once distinguished by the size and appearance of the head.

A small brain-tumor may exist without causing optic neuritis; but unless it give rise to focal symptoms the diagnosis must usually be long reserved and finally reached chiefly by exclusion,—that is, by the determination that no other explanation can be found for the symptoms which persist indefinitely. When dementia paralytica develops with epileptic attacks, followed by transitory paralysis, an error is possible, but should be prevented by the peculiar alterations of the intelligence and by the absence of severe headache.

In the advanced stages of cerebral tumor, when optic atrophy has occurred, a likeness to multiple sclerosis is possible. In the latter disorder, however, there is little or no headache, very rarely vomiting, and still more rarely slowing of the pulse, whilst there are present the spinal symptoms which are absent in cerebral tumor.

Acute encephalitis with optic neuritis is to be recognized by its rapid development with pronounced fever.

The diagnosis as to the exact seat of the brain-tumor is to be made by carefully applying the principles of cerebral localization. Localizing symptoms which develop late in the course of a case are frequently due to secondary changes at a distance from the original tumor. Especially are tumors which are placed in or about the Sylvian fissure prone by pressure upon blood-vessels to cause distant softening. The diagnosis of the nature of a cerebral tumor can be nothing but a guess, unless such tumor be secondary to tubercular, carcinomatous, or other growths in distant parts of the body.

TREATMENT.—In no form of cerebral tumor, except syphiloma, is medical treatment permanently effective. Removal of the tumor is probably practicable in about three per cent. of the cases. An attempt at it is justifiable only when the tumor is clearly located in the psycho-motor zone and has positively resisted antisyphilitic treatment. When the headache is so atrocious that the patient prefers death to life, after exhaustion of all other methods for relief, trephining for relief of pressure is a defensible procedure.

Cerebral surgery as practised by various surgeons has been very valuable to neurology by affording early post-mortems in cases which otherwise might, after some months, have drifted away from medical watching.

CEREBRAL SYPHILIS.

DEFINITION.—Gummous inflammation or neoplasm in the brain or its membranes.

ETIOLOGY.—Cerebral syphilis may appear within three months after primary infection, but usually is delayed from one to thirty years. It is especially liable to develop when the secondary symptoms have not been severe, and often occurs when both primary and secondary symptoms have been so slight as to escape observation on the part of the victim. Inherited syphilis is less prone to attack the nervous system than is acquired syphilis, but cerebral gummata may develop during intra-uterine life and at any time subsequently; indeed, nervous syphilis may appear after puberty as the first open outbreak of inherited disease.

MORBID ANATOMY.—The cerebral gumma probably always has its origin in the membranes, is usually surrounded by a reddish zone, and does not become so uniformly and completely caseous as does tubercle, from which it is further distinguished by its proneness to cause cerebral softening. In gummous meningitis the exudation forms an extended, shapeless, gelatinous mass, which is in the majority of cases situated at the base of the brain. Microscopically, the cerebral gumma differs from other similar bodies chiefly in the presence of very large, spider-like cells containing an exaggerated nucleus and a granular protoplasm, which extends into the multiple, branching, rigid prolongations.

Under treatment gummata may disappear completely or may leave behind them cicatrices, imperfect cysts, or even calcareous masses. A gumma may involve a blood-vessel, and, extending along its wall, give rise to a thrombus with secondary softening, or it may lead to rupture of the vessel and intra-cerebral hemorrhages. A gummous inflammation commencing in the pia mater may infiltrate a wide extent of the cortex.

Syphilitic sclerosis of the cerebral vessels is not rare, and the arteries of the base are especially prone to suffer from a peculiar destructive specific lesion which renders them whitish, opaque, and hard, and finally almost obliterates their lumen.

SYMPTOMATOLOGY.—Although brain syphilis probably always develops somewhat slowly, yet the symptoms may appear most abruptly and violently, headache, vertigo, or other prodromes having passed by unnoticed. Syphilitic fulminating coma may or may not be accompanied by convulsions, by delirium, or by hemiplegia, monoplegia, or local paralysis. In its general symptoms it may conform to any variety of apoplectic or epileptic attack.

The symptoms of *chronic brain syphilis* are so protean, so varying, that it is almost impossible to reduce them to any order. Malaise, a little brain-failure, a succession of causeless headaches,—these may for a time be all the outcome. After a greater or less continuance of these prodromes, epileptic attacks usually develop, with a hemiplegia or a monoplegia which is almost invariably incomplete and usually progressive; diplopia from weakness of ocular muscles, decided squint, or pronounced oculo-motor palsy may be manifested before the epilepsy. There is almost always distinct failure of the general health, with progressive intellectual deterioration, as shown by loss of memory, failure of the power to fix the attention, mental bewilderment, morbid somnolence, perhaps aphasia, and towards the end of life not rarely dementia. If the case convalesce, the amelioration will be gradual. A fatal ending is usually by a gradual sinking into complete paralysis, or the patient is carried off by an acute inflammatory exacerbation or by a very violent epileptic fit. Death from brain softening around the tumor is not infrequent, but a fatal apoplectic hemorrhage is rare.

It is almost impossible satisfactorily to reduce to any order or types the various forms of cerebral syphilis. Besides those cases which resemble dementia paralytica, Heubner makes two types: (1) psychical disturbances, with epilepsy, incomplete paralysis (seldom of the cranial nerves), and a final comatose condition, usually of short duration; and (2) genuine apoplectic attacks with succeeding hemiplegia, in connection with peculiar somnolent conditions occurring in often-repeated episodes; frequently phenomena of unilateral irritation, and generally at the same time paralyses of the cerebral nerves.

The only conformity of brain syphilis, as we have seen it, with these types is in the fact that when epilepsy is pronounced the basal cranial nerves are not usually paralyzed; and it seems necessary to add two other types of disease,—namely:

(3) Psychical disturbance without complete epileptic convulsions, associated with palsy of the basal nerves and often with partial hemiplegia.

(4) Paraplegia associated with ocular or other symptoms indicative of lesions at the base of the brain.

In nature, however, there are no distinct varieties of cerebral syphilis, all forms grading one into the other, and it is most satisfactory to study the important symptoms separately.

Headache is the most constant, and usually the earliest, of the symptoms of meningeal syphilis, but it may be entirely wanting. It may last for several years without the development of other distinct symptoms, and sometimes disappears when these appear. It has no fixed character, but is usually paroxysmal, and may occur solely in the form of very distinct and very violent paroxysms, accompanied by partial unconsciousness or other marked congestive symptoms. Distinct soreness of the head indicates disease of the skull or its periosteum.

Insomnia is a frequent prodrome of cerebral syphilis, but a peculiar somnolence is much more characteristic. The foudroyant coma has already been described: in the second variety of syphilitic stupor the symptoms develop gradually. The patient sits all day long or lies in bed in a state of semi-stupor, indifferent to everything, but capable of being aroused, answering questions slowly, imperfectly, and without complaint, but in an instant dropping off again into his quietude. In other cases the sufferer may still be able to work, but often falls asleep while at his tasks, and especially towards evening has an irresistible desire to slumber. This state of partial sleep may precede that of the more continuous stupor or may pass off when an attack of hemiplegia seems to divert the symptoms. The mental phenomena in the more severe cases of somnolency are peculiar. The patient can be aroused,—indeed, in many instances he exists in a state of torpor rather than of sleep; when stirred up he thinks with extreme slowness, and may appear to have a form of aphasia, yet at intervals he may be endowed with a peculiar automatic activity, especially at night. Getting out of bed; wandering aimlessly and seemingly without knowledge of where he is, and unable to find his own bed; passing his excretions in a corner of the room or in some other similar place, not because he is unable to control his bladder and bowels, but because he believes that he is in a proper place for such acts,—he seems a restless nocturnal automaton rather than a man. Apathy and indifference are the characteristics of the somnolent state, yet the patient will sometimes show excessive irritability when aroused, and will at other times complain bitterly of pain in his head, or will groan as though suffering severely in the midst of his stupor,—at a time, too, when he is not able to recognize the seat of the pain. We have seen a man with a vacant, apathetic face, almost complete aphasia, and persistent heaviness and stupor, arouse himself when the stir in the ward told him that the attending physician was present, and come forward in a dazed, highly pathetic manner, by signs and broken utterances begging for something to relieve his head. Heubner speaks of cases in which the irritability was such that the patient fought vigorously when aroused.

After some days of excessive somnolence and progressive deepening of the stupor, or sometimes more rapidly, the victim of cerebral syphilis may pass into a condition of profound coma, out of which he cannot be aroused, and during which his fæces and urine are either not passed at all or are voided involuntarily. This condition of coma may end in death, but even when the symptoms seem most serious the patient may gradually recover, slowly emerging from coma into stupor, and from stupor into wakefulness and normal life.

Motor paralysis sometimes develops gradually, but it may appear suddenly, with or without the occurrence of an apoplectic or epileptic fit. A paralysis which on the first return to consciousness is complete is usually due to clot or thrombus.

The characteristic syphilitic palsy is progressive and incomplete. Any portion of the body may be involved, but the syphilitic exudation especially haunts the base of the brain, and a rapidly but not abruptly appearing strabismus, ptosis, dilated pupil, or any other paralytic eye-symptom not readily accounted for in the adult is, in the majority of cases, syphilitic. The specific palsy is often temporary, transient, and shifting. Sensory palsies are less frequent than motor palsies, but may occur in any form. Special-sense palsies are sometimes present, whilst specific aphasia is common. It may be incomplete, transitory, and paroxysmal, but is more apt to be complete and to have permanency than are motor paralyses. Owing to the tendency of syphilis to produce multiple lesions, a lack of apparent agreement between the palsy and the aphasia is almost characteristic. Thus, Tarnowsky found that out of thirty-two cases of syphilitic aphasia with hemiplegia in fourteen the paralysis was on the left side. Polyuria and true saccharine diabetes occur in cerebral syphilis, probably as the result of vaso-motor disturbances.

Epileptiform convulsions are most characteristic: an intense and protracted headache, followed by an epileptic fit, in an adult should excite the greatest suspicion. Our experience is in accord with that of Fournier, that epileptiform convulsions not due to alcoholism or uræmia, and not appearing until after thirty years of age, are in nine cases out of ten specific. The aura is rarely present; the symptoms may be unilateral or even monoplegic, but any variety of epileptiform convulsions may be simulated. Furious attacks of local spasms also occur without loss of consciousness. Then, again, the movements may be continuous and distinctly choreic.

Apathy, somnolence, loss of memory, and general mental failure are the most frequent and characteristic mental symptoms of meningeal syphilis; but almost any form of insanity—mania, melancholia, erotic mania, delirium of grandeur, etc.—may be of specific origin. Usually, sooner or later, distinct symptoms of organic lesion appear. Especially common is a loss of mental and physical power similar to that which occurs in dementia paralytica.

DIAGNOSIS.—In the diagnosis of cerebral syphilis too much weight should not be attached to the history of the case, as non-syphilitic, organic brain disease may occur in persons who have had syphilis, and cerebral gummata may develop in persons who are unconscious of ever having been infected.

The prodromes of foudroyant cerebral syphilis are worthy of the most careful study on account of their diagnostic value and of their habitually being overlooked. Persistent headache, slight failure of memory, unwonted slowness of speech, general lassitude and disinclination to mental exertion, sleeplessness or excessive somnolence, attacks of momentary giddiness, vertiginous feelings when straining at stool, yelling or in any way disturbing the cerebral circulation, alteration of disposition,—any of these (and *a fortiori* several of them) occurring in a syphilitic subject should be the immediate signal for alarm. Of these prodromic symptoms the most important and characteristic are headache and somnolence. Slight and shifting localized weaknesses sometimes precede an acute attack, but are more characteristic of the disease at a later stage. A momentary weakness of one arm; a slight drawing of the face, disappearing in a few hours; a temporary dragging of the toes; a partial aphasia which appears and reappears; a squint which to-morrow leaves no trace,—all or any of these may be due to a non-syphilitic brain-tumor, to miliary cerebral aneurisms, or to some other non-specific affection; but in the majority of cases, when these phenomena occur repeatedly in a patient who is not suffering from hysteria they are the result of syphilis.

In a doubtful case of sudden coma other ordinary causes must be eliminated: a pronounced rise of temperature or a pronounced conjugate deviation of the head and eyes tells strongly against, whilst decided ocular palsy or a partial paralysis of any character argues in favor of, a specific origin.

Headache occurring with palsy or with a history of attack of partial monoplegia or hemiplegia, vertigo, petit mal, epileptoid convulsions, disturbances of consciousness, attacks of unilateral or localized spasms, ocular palsies, epileptic forms of attacks occurring after thirty years of age, morbid somnolence,—any of these, even when existing alone, should put the practitioner upon his guard. Any apparent causelessness, severity, and persistency of headache should arouse suspicion, to be much increased by a tendency to nocturnal exacerbations or by the occurrence of mental disturbance or of giddiness at the crises of the paroxysms. Not rarely there are very early in these cases curious, almost indefinable, disturbances of cerebral functions which may be easily overlooked, such as temporary and partial failure of memory, word-stumbling, fleeting feelings of numbness or weakness, and alterations of disposition. In the absence of hysteria, any indefinite and apparently disconnected series of nerve-accidents is of very urgent import. To use the words of Hughlings-Jackson, "A random association or a random succession of nervous symptoms is very strong warrant for a diagnosis of a syphilitic disease of the nervous system." Cerebral syphilis occurring in an hysterical subject may be readily overlooked until fatal mischief is done.

The age of the patient must also be taken into consideration. Apoplexy occurs most frequently in persons over fifty years of age, while congestive syphilitic attacks are most common before that age. The course of a case for the first six or ten hours after the commencement of the acute paroxysm is sufficiently different in the two affections to be worthy of the closest study. A hemorrhagic or embolic apoplexy which is sufficiently severe to keep up pronounced disturbance of consciousness for some hours is almost invariably accompanied by a complete hemiplegia, or more rarely by some other form of complete palsy; whilst in the syphilitic attack the paralysis is often absent, and probably never complete. Unless the clot has been a very large one, the return to consciousness after hemorrhagic apoplexy is usually much more rapid than it ordinarily is in syphilitic cases. Headache after an apoplexy is rare, whilst it is very frequent after a severe syphilitic congestive attack.

The peculiarities in the symptoms of cerebral syphilis are chiefly due to the fact that the lesions are apt to be multiple or wide-spread, to be rapidly developed at an age when other organic diseases are rare, and to be situated in the cerebral cortex or at the base of the brain. Hence multiple local or partial palsies are frequent, whilst the symptoms of the basal chronic meningitis in the non-tubercular adult are in the majority of cases the outcome of syphilis. Homonymous hemianopsia is very rare, because the occipital lobes are seldom invaded. Optic neuritis may occur in specific as in other brain diseases: it usually develops with moderate rapidity.

The diagnosis of cerebral syphilis during life is always a matter of inference. When, however, the symptoms disappear under antisyphilitic treatment, for practical purposes the diagnosis may be considered as fixed. The therapeutic test is therefore a matter of the gravest importance. The old belief of syphilographers that tolerance of the iodides warrants the diagnosis of syphilis has been in recent times strongly combated, but we still think that, whilst it is not a positive sign, the tolerance strongly increases the probability of the existence of specific disease.

PROGNOSIS.—Although death may occur during a syphilitic convulsion, yet the prognosis of an acute attack of cerebral congestion or inflammation due to syphilis is on the whole favorable, although it should be somewhat guarded. In chronic brain syphilis the prognosis is favorable for more or less complete recovery unless the symptoms indicate an absolute destruction of brain-tissue. Whenever amendment of the symptoms occurs under antispecific medication, more or less complete recovery becomes probable. As, however, unexpected accidents occasionally happen, it is best not to make the prognosis too absolute.

TREATMENT.—The treatment of cerebral syphilis is best studied under two heads: first, the treatment of the accidents which occur in the course of the disease; second, the general treatment of the disease itself.

In the accidents of cerebral syphilis the treatment should be that which is adapted to the relief of the same symptoms when dependent upon other than specific causes. Thus, in foudroyant coma, if there be pronounced arterial excitement, or if the patient's strength be good, venesection should be resorted to at once. We have seen life saved by the abstraction of nearly a quart of blood, whilst in other cases that of a few ounces has sufficed. Care must be exercised not to mistake a simple epileptiform convulsion for a pronounced congestion of the brain, but if there be epileptic status with repeated convulsions, or if there be violent delirious excitement, venesection may be resorted to if the patient's general condition permit. In severe cases the bleeding should be as rapid as possible from a large orifice, and be continued until a distinct impression is made upon the pulse. When the heart's action continues violent after venesection, the hypodermic injection of tincture of aconite root (two to four drops) may be given every half-hour until physiological effects are manifest. In feeble cases cupping to the back of the neck, stimulating injections, sinapisms to the extremities, cold to the head, croton oil as a derivative, and other classical remedial measures for brain congestion may be used.

In chronic cerebral syphilis remedial measures looking to the relief of symptoms may occasionally be employed with temporary advantage, but are of comparatively little importance.

The first therapeutic question to be decided is usually as to the choice between mercurials and the iodides. Cerebral gummata may develop in persons showing marked evidences of cachexia, but in the great majority of cases cerebral syphilis appears at a time when there is no general breaking down of the tissues or of the general system. The choice between the remedies should rest upon the existing symptoms, and not upon the time which has elapsed between the primary infection and the outbreak. When cachexia contra-indicates the free use of mercurials, or even of iodides, tincture of iron and corrosive sublimate may be given together. (Formula 10.)

The slowness of action of the iodides may be serious. Death from an epileptic fit may be the penalty of delay. When there is no cachexia and no history of recent mercurialization, mercury should be given at once to slight salivation, and a mercurial impression, just below the line of slight tenderness of the gums, should be maintained for some days or weeks, *pro re nata.*

The method of administering mercury should be suited to the exigencies of the individual case. If mercurials by the mouth are well borne, they should be so administered. If the symptoms are extremely urgent, the mercury may be given both by the mouth and by inunctions. When there is a tendency to diarrhœa, the mercurial inunction should be used alone. The oleate is not preferable to the old blue ointment : from a half-drachm to three drachms of either may be used at once. An excellent plan is to give a hot bath late in the afternoon, or better in the evening, and use the inunction on going to bed, ordering the patient to rub the ointment on Sunday night into the left axilla, on Monday night into the left flank, on Tuesday night into the inside of the left thigh, on Wednesday night into the right axilla, on Thursday night into the right flank, on Friday night into the right thigh, and on Saturday night into the region of the umbilicus ; after this recommencing with the left axilla.

If the patient is willing to endure the local pain, the hypodermic injection of corrosive sublimate is sometimes extraordinarily efficacious. From one-twelfth to one-sixth of a grain dissolved in a drachm of distilled water should be injected deeply into the muscles of the back daily or every other day, according to circumstances.

After a prolonged mercurial course potassium iodide should always be given, in order to secure elimination of the mercury as well as to relieve the syphilis. The dose of the iodide must be suited to the individual case. The beginning daily dose should be thirty grains, rapidly increased to four drachms unless iodism results. There are cases of cerebral syphilis in which not more than five or ten grains a day of the iodide can be tolerated, and in which such small dose accomplishes as much good as does the large dose in the ordinary case. It is often best to give the iodide in milk, or compound syrup of sarsaparilla may be used as the vehicle. The number of daily doses may be from four to six. The older preparations of the "woods," so called, seem sometimes to have special value : thus, Zitmann's decoction occasionally is successful in very old cases after the failure of the other mercurials. A fair imitation of this older prescription may be made by using as a vehicle for the iodide a mixture of equal parts of compound fluid extract and compound syrup of sarsaparilla. (Formula 11.)

CHAPTER IV.

DISEASES OF THE MEDULLA OBLONGATA.

For a comprehension of the diseases of the medulla oblongata it is essential that it be fully recognized that the medulla belongs not to the brain but to the spinal cord, both in its anatomy and in its physiology. The spinal cord entering the skull bends forward, so that the posterior portion of the cord becomes the upper surface of the medulla. The first change in structure is an opening of the posterior commissure of the spinal cord into the central canal: by this splitting process is formed the floor of the fourth ventricle. Then the lateral columns decussate obliquely, each crossing over to the opposite side of the medulla. During its passage across the medulla each column cuts the gray matter into three parts: out of each anterior horn of the gray matter of the spinal cord are formed two isolated parts, one representing the central portion of the gray matter and situated in the floor of the fourth ventricle, the other representing the distal parts of the anterior horn of the spinal gray matter and situated deeply in the anterior portion of the medulla. The posterior horns of the spinal cord also undergo bisection, each being separated like the anterior into two tracts, of which one, representing the central part of the cord, is in the floor of the fourth ventricle, and the other, representing the distal gray matter of the cord, is deep in the medulla.

Fig. 12.

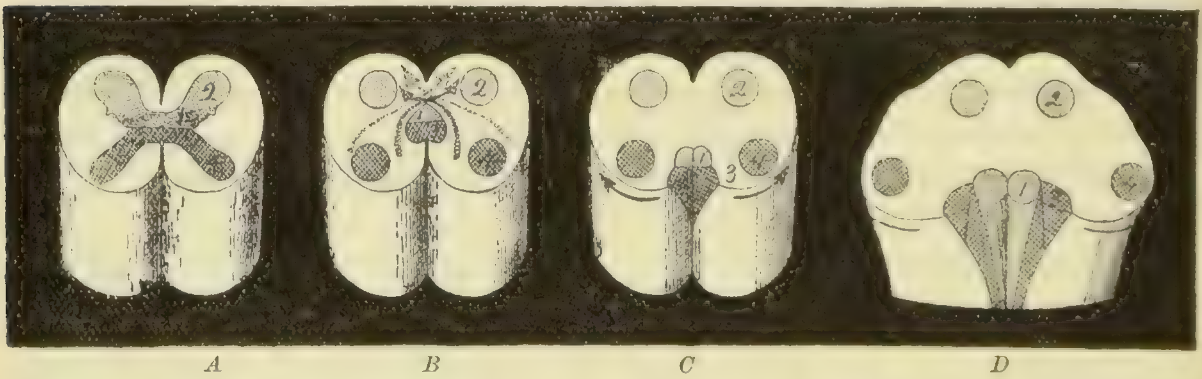


Diagram showing modification of spinal cord into medulla.—*A*, spinal cord: *D*, medulla. 1, base of anterior horn; 2, apex of anterior horn; 3, base of posterior horn; 4, apex of posterior horn. (After Grasset.)

It is plain that the nuclear mass situated in the floor of the fourth ventricle represents the whole of the central portion of the gray matter of the spinal cord, from which have been cut off the ends of both the anterior and the posterior horns. In the central ganglionic mass—*i.e.*, the floor of the fourth ventricle—are the pneumogastric and respiratory centres and the nuclei of the abducent, facial, auditory, and glosso-pharyngeal nerves; from it also go sensory fibres to the trigeminal and other mixed nerves.

Out of the deep-seated gray matter in the medulla which represents the extreme posterior horns come sensory fibres for the glosso-pharyngeal and pneumogastric nerves; whilst in the deeply situated gray substance which represents the extreme anterior horns of the spinal cord are nuclei of the spinal, pneumogastric, and hypoglossal nerves.

In the gray matter of the spinal cord are situated the trophic cells of tributary muscles, and the gray masses in the medulla which represent isolated portions of the spinal gray matter preserve the functions of the latter, and are the trophic centres of the various nerves which originate in the medulla and are supplied to the muscles of the head and neck.

When the anatomy and physiology of the medulla oblongata are comprehended it must be apparent that its diseases are those of the spinal cord, the only differences being in the distribution of the various motor and sensory disturbances induced. Owing, however, to the greater activity of its circulation, the medulla suffers more frequently from hemorrhages into it than do the strictly spinal centres. It is, in its turn, much less frequently attacked than is the brain. The results of bulbar hemorrhages have been sufficiently treated under the general head of apoplexy. (See page 501.) Foreign growths in the medulla oblongata produce localizing symptoms by disturbances of the nerves which come from the medulla, and also cause a more or less pronounced hemiplegia or general paralysis by pressing upon or otherwise interfering with the function of the descending pyramidal tract or tracts.

Any lesion which irritates the pneumogastric centre will produce slowing of the heart, whilst a lesion which paralyzes the pneumogastric centres will register itself in rapidity of heart-action. Lesions of the respiratory centre produce most serious and often fatal disturbances of the respiration.

In multiple cerebro-spinal sclerosis the formation of the nodule in the medulla oblongata is followed by symptoms of irritation or paralysis corresponding to the seat of the nodule. For a detailed description of the symptoms which follow paralysis of the various nerves having origin in the medulla oblongata, see article on Local Paralyses.

GLOSSO-LABIAL PARALYSIS. BULBAR PALSY.

DEFINITION.—A poliomyelitis affecting the motor centres in the medulla oblongata, with consequent paralysis and trophic changes in the tributary muscles.

Glosso-labial paralysis is in no proper sense a distinct disease; it is only a form of chronic poliomyelitis, and may or may not be associated with similar lesions in other portions of the spinal system and the consequent symptoms of so-called progressive muscular atrophy in the affected parts. We retain the distinct heading simply out of deference to a custom which originated before the nature of the disease was understood. Its etiology and pathology are those of poliomyelitis.

SYMPTOMATOLOGY.—The chief symptoms of this disease are a progressive loss of power in the tongue, lips, palate, and muscles of the throat, associated with wasting and fibrillary contractions in the affected muscles. The tongue is protruded more slowly and imperfectly than is normal, and becomes more and more tremulous. Owing to loss of control over it, the pronùnciation of the lingual vowels and of the dental consonants is imperfect. The weakness of the lips shows itself by the failure in articulation of the labial consonants, by the inability to whistle, by tremulousness, and, finally, by the loss of the power of the mouth to retain the saliva, which dribbles constantly. As the disease is almost always symmetrical, the mouth is not drawn to one side, but the wasting of the parts about it may be sufficient to make the orifice appear much larger than normal and to confuse the naso-labial folds. Sometimes the lips during laughter separate, but are incapable of spontaneously returning to their natural position, so that the patient is forced to replace them with the fingers. If the palate is markedly affected, the voice becomes nasal.

Deglutition may be affected early or late in the disorder, and, as the loss of power of swallowing is paralytic, liquids are swallowed with much difficulty and are apt to be returned through the nose. In some instances the larynx is attacked and the voice becomes almost inaudible, without, however, being completely lost. In those cases in which the nuclei of the respiratory nerves are implicated the respiratory muscles undergo wasting and the respiration is much affected. Any attempt at violent movement, or, later in the disease, even ordinary walking, may cause a severe attack of dyspnœa. At last these cyanotic crises come on spontaneously in furious paroxysms, which may occur either by day or by night. A peculiar symptom which especially characterizes this dyspnœa is a sensation of excessive fulness of the chest, which is probably produced by the feebleness of the muscles preventing them from thoroughly emptying the lungs. In some cases the nuclei of the cardiac nerves appear to be attacked, and cardiac crises become violent and alarming. These are especially apt to be present in those persons in whom the respiration is affected, but may occur without the respiratory muscles suffering. The pulse in the cardiac crises is very feeble, irregular, intermittent, and at last may be imperceptible. The face is exceedingly pale and anxious, and there is habitually an intense terror, with a sense of impending death. The ocular muscles may be affected in bulbar palsy, although they usually escape.

PROGNOSIS.—Death always results,—in rare cases in a few months, more commonly in from one to three years; usually from paralytic interference with swallowing or breathing.

DIAGNOSIS.—Glosso-labial paralysis may be confounded with the very rare instances in which multiple lesions in the cerebrum influence especially the muscles of the mouth, tongue, and throat. There should, however, be no difficulty in recognizing the true nature of the pseudobulbar paralysis, because the paralyzed muscles do not undergo atrophy, and because almost of necessity there must be associated with the bulbar symptoms hemiplegic or other forms of motor paralysis or spasm, and also disturbances of intellection or of the special senses.

A number of very curious cases have been reported by Goldflams, Erb, and others, in which the symptoms closely resembled those of bulbar paralysis, but in which careful studies of the medulla made after death failed to detect any lesions. According to Oppenheim, these cases agree in having, besides the throat and mouth symptoms, ptosis, and weakness of the muscles of the rump and of the extremities; and, further, in the fact that there is no wasting of the affected muscles. The nature of the lesion is entirely obscure; it may be that the cases represent a hitherto undescribed disease, but it is more probable that they are instances of neuritic atrophy (see page 615) especially affecting the medulla oblongata. In a rapid case of apparently bulbar paralysis, which was atypical in the presence of bilateral complete facial paralysis and of trigeminal neuralgia, and which ended after four weeks in death from dyspnœa and collapse, Eisenlohr found that the lesion was a multiple neuritis.

TREATMENT.—There is no known specific effective treatment of glosso-labial paralysis. As the power of swallowing becomes impaired, great care to prevent choking should be exercised. Crises must be treated symptomatically.

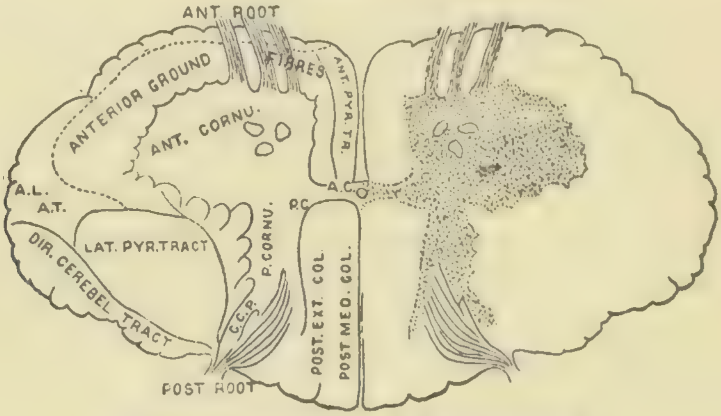
CHAPTER V.

ORGANIC DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

SPINAL LOCALIZATION.

Localization of spinal disease is twofold: it relates disease, in the first place, to the anatomico-physiological tracts of the spinal cord, and, in the second place, to the vertical position of the lesion in the cord. The white matter of the spinal cord is divided into certain so-called longitudinal tracts or columns, which are set forth in the accompanying diagram.

Fig. 13.



Diagrammatic section of the spinal cord.

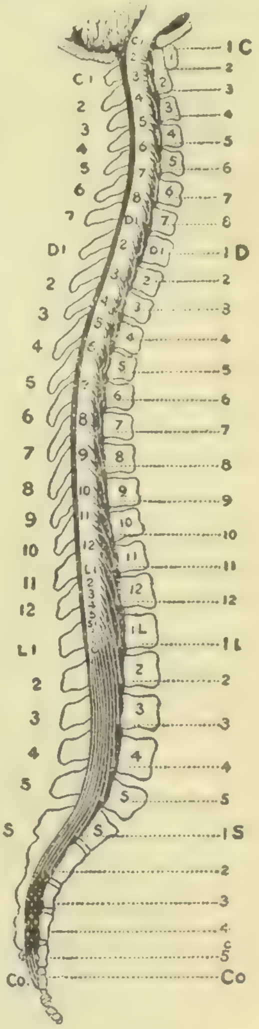
The posterior median column, or the column of Goll, lies immediately in contact with the posterior fissure of the cord. It is composed chiefly of fibres which enter through the posterior nerve-roots and pass upward. The increase in the size of the column of Goll from below upward does not seem to be sufficient for the accommodation of all the fibres that enter the column, supposing that these fibres continuously travel upward to the brain; and, further, the function of the column of Goll still remains in doubt: it is, indeed, probable that some or possibly all of the fibres escape from the column before reaching its summit, but how or where such escape is made remains as yet uncertain.

Next to the column of Goll lies the postero-external column, the column of Burdach, or the posterior root-zone, chiefly composed of vertical fibres, whose function is at present unknown.

The direct cerebellar tract seems to be chiefly composed of fibres which enter it through the lateral column from the gray substance and pass upward. It seems to have the function of conducting impulses upward, and, according to Flechsig, it probably carries impressions from the muscles of the trunk.

Both the lateral pyramidal tract, "crossed cerebral tract," and the anterior pyramidal tract, "direct cerebral tract," or column of Türck, are composed of fibres whose course is downward from the pyramids of the medulla. At the decussation of the pyramids about three-fourths of the fibres cross to form the lateral tract, whilst the remaining fourth of the fibres enter the cord without decussation and constitute the anterior tract. The fibres of both pyramidal tracts finally pass through the gray matter of the spinal cord into the anterior horns, and, although they have not been traced, almost certainly end in the processes of the motor cells. Their function is to conduct from above downward, and they probably constitute the pathway by which impulses from the motor regions of the cerebral cortex reach the motor ganglionic cells of the spinal cord, whose answering discharges provoke the final muscular contraction.

FIG. 14.



Anatomy of the spinal cord (after Gowers).—C, cervical; D, dorsal; L, lumbar; S, sacral; Co, coccyx.

The remaining portion of the white matter of the spinal cord is composed, first, of the so-called antero-lateral ascending tract (A.L., A.T.), which forms the periphery of the anterior portion of the cord; second, of the mass of white fibres marked in the diagram as the anterior ground-fibres. The functions of these portions of the spinal cord have not been made out. Flechsig also anatomically separates the little patch of white matter between the lateral pyramidal tract and the gray matter, known as the lateral limiting layer. The separation of the antero-lateral tract from the so-called anterior ground-fibres does not seem to be at present warranted, either on anatomical or on physiological grounds. Indeed, it is doubtful whether there is sufficient continuity, either of structure or of function, for any of the parts of the spinal cord spoken of in this paragraph to be considered as distinct columns or tracts; also whether any degeneration ever follows the course of these regions.

The gray matter of the spinal cord, besides numerous conducting fibres, contains ganglionic cells whose processes are prolonged into nerve-fibres composed solely of the axis-cylinders. The ganglionic cells are arranged in groups which vary in different portions of the cord, and probably in the same portion of the cord in different individuals. The most readily recognized of the groups are the small inner or medial group, situated in the inner anterior angle of the horns; the large anterior group, placed near the anterior edge of the horns, in the middle or a little to the outer side of the middle of the margin; the anterior lateral group, situated in the outer extremity of the front of the horns (the last two groups frequently consolidate); and the external or postero-lateral group, which is usually the largest and is extended in the posterior outer angle of the cord.

The most condensed statement of the facts necessary for the practitioner to know for the purpose of locating vertical lesions of the spinal cord is in the table of Starr. By means of this table and Fig. 14 the vertical position of almost any spinal lesion can be determined. In studying the diagram and table it must be remembered that the cervical cord is divided into eight segments, and that "II. and III. C." in the first column of the table refers to second and third cervical segments; that the dorsal cord is divided into twelve segments, "I. D.," and so on; that the lumbar cord is divided into five segments, and the sacral also into five segments; thus, "III. to V. S." means third to fifth sacral segments.

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
II. and III. C.	Sterno-mastoid.	Hypochondrium (?).	Back of head to vertex.
	Trapezius.	Sudden inspiration produced by sudden pressure beneath lower border of ribs.	Neck.
	Scaleni and neck. Diaphragm.		
IV. C.	Diaphragm.	Pupil. Fourth to seventh cervical.	Neck.
	Deltoid.	Dilatation of the pupil produced by irritation of neck.	Upper shoulder.
	Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.		Outer arm.
V. C.	Deltoid.	Scapular.	
	Biceps.	Fifth cervical to first dorsal.	Back of shoulder and arm.
	Coraco-brachialis.		Outer side of arm and fore-arm, front and back.
	Brachialis anticus.	Irritation of skin over the scapula produces contraction of the scapular muscles.	
	Supinator longus. Supinator brevis.		
	Rhomboid.	Supinator longus.	
	Teres minor.	Tapping its tendon in wrist produces flexion of forearm.	
	Pectoralis (clavicular part). Serratus magnus.		
VI. C.	Biceps.	Triceps.	Outer side of forearm, front and back.
	Brachialis anticus.	Fifth to sixth cervical.	
	Pectoralis (clavicular part).	Tapping elbow tendon produces extension of forearm.	Outer half of hand.
	Serratus magnus.		
	Triceps.	Posterior wrist.	
	Extensors of wrist and fingers.	Sixth to eighth cervical.	
	Pronators.	Tapping tendons causes extension of hand.	
VII. C.	Triceps (long head).	Anterior wrist.	Inner side and back of arm and forearm.
	Extensors of wrist and fingers.	Seventh to eighth cervical.	
	Pronators of wrist.	Tapping anterior tendons causes flexion of wrist.	Radial half of hand.
	Flexors of wrist. Subscapular.	Palmar; seventh cervical to first dorsal.	
	Pectoralis (costal part). Latissimus dorsi.	Stroking palm causes closure of fingers.	
	Teres major.		

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
VIII. C.	Flexors of wrist and fingers.	. . . .	Forearm and hand, inner half.
	Intrinsic muscles of hand.		
I. D.	Extensors of thumb.	. . . .	Forearm, inner half.
	Intrinsic hand-muscles.	. . . .	Ulnar distribution to hand.
	Thenar and hypothenar eminences.		
II. to XII. D.	Muscles of back and abdomen.	Epigastric, fourth to seventh dorsal.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves.
	Erectores spinæ.	Tickling mammary region causes retraction of the epigastrium.	Upper gluteal region.
		Abdominal seventh to eleventh dorsal.	
		Stroking side of abdomen causes retraction of belly.	
I. L.	Ilio-psoas.	Cremasteric, first to third lumbar.	Skin over groin and front of scrotum.
I. L.	Sartorius.		
	Muscles of abdomen.	Stroking inner thigh causes retraction of scrotum.	
II. L.	Ilio-psoas.	Patellar tendon.	Outer side of thigh.
	Sartorius.		
	Flexors of knee (Remak).	Striking tendon causes extension of leg.	
	Quadriceps femoris.		
III.	Quadriceps femoris.	. . . .	Front and inner side of thigh.
	Inner rotators of thigh.		
	Abductors of thigh.		
IV. L.	Abductors of thigh.	Gluteal.	Inner side of thigh and leg to ankle.
	Abductors of thigh.	Fourth to fifth lumbar.	
	Flexors of knee (Ferrier).	Stroking buttock causes dimpling in fold of buttock.	Inner side of foot.
	Tibialis anticus.		
V. L.	Outward rotators of thigh.	. . . .	Back of thigh, back of leg, and outer part of foot.
	Flexors of knee (Ferrier).		
	Flexors of ankle.		
	Extensors of toes.		
I. to II. S.	Flexors of ankle.	Plantar.	Back of thigh.
	Long flexor of toes.	Tickling sole of foot causes flexion of toes and retraction of leg.	Leg and foot, outer side.
	Peronei.		
	Intrinsic muscles of foot.		
III. to V. S.	Perineal muscles.	Foot reflex.	Skin over sacrum.
		Achilles tendon.	Anus.
		Over-extension of foot causes rapid flexion ; ankle-clonus.	Perineum.
		Bladder and rectal centres.	Genitals.

SPINAL HEMORRHAGE.

Hemorrhage may take place into the spinal cord itself, constituting the so-called *hæmatomyelia* or *spinal apoplexy;* or it may occur into the membranes of the spinal cord, when it is known as *hæmatorrhachis.*

Hæmatomyelia is almost always secondary to various diseases of the spinal cord ; theoretically, however, it may occur without preceding obvious organic disease of the spinal cord as the result of degeneration of the coats of the blood-vessels. Hæmatorrhachis may be secondary to diseases or traumatism of the membranes, but is not very rare as a consequence of syphilitic or other disease of the blood-vessels.

In hæmatomyelia, with or without loss of consciousness, there is a sudden motor and sensory paralysis of all the parts below the seat of the hemorrhage. Pain is very rarely severe, and is often practically wanting. The bladder and rectum are immediately implicated. Myelitis is usually rapidly developed, with loss of reflexes, muscular atrophy, decubitus, etc. The prognosis is absolutely grave, and no treatment is of distinct value. The greatest care should be taken to prevent the development of bedsores.

In hæmatorrhachis the paralysis comes on rapidly, but not abruptly, requiring from a few minutes to some hours for its completion. It is accompanied with great pain in the back and tearing "tiger-claw" or burning pains in the extremity, which are due to the blood as it forces its way along the membranes, tearing or stretching the sensory nerve-roots. Muscular spasms may occur from irritation of the anterior roots. The anæsthesia is usually not as complete as in spinal hemorrhage, nor does it at its upper limits cease as abruptly. The bladder and rectum are paralyzed, unless the hemorrhage is a very small one, when all the symptoms are imperfectly developed.

In most cases the diagnosis between hæmatomyelia and hæmatorrhachis is easy. If, however, a very large vessel bursts in the spinal membranes, the symptoms, except in the presence of severe pain, may closely simulate those of an intra-spinal hemorrhage. The prognosis of hæmatorrhachis is very serious, most of the cases ending fatally. When the hemorrhage is very small, however, recovery with some impairment of function is possible. There is no specific treatment. In a robust subject venesection would be justifiable.

SPINAL EMBOLISM AND THROMBOSIS.

Embolic and thrombotic arrest of circulation, except as secondary complications of previously existing diseases in the spinal cord, are among the rarest of clinical phenomena. If a vessel of any size were affected the symptoms would resemble those of intra-spinal hemorrhage; and as a secondary result of such arrest of circulation there would be a softening of the spinal cord similar to the softening of the brain. Such pathological condition of the spinal cord has been recognized, and is usually spoken of as due to a myelitis. True softening is, however, always necrobiotic, and even if it be associated with a neoplasm or a localized inflammation of the spinal cord is not inflammatory, but is due to interference with the circulation by the neoplastic or inflammatory products. It is also probable that in some cases wide-spread degeneration of the vessel-walls may lead, without absolute arrest of circulation, to softening of the spinal cord. The symptoms which accompany softening are progressive paralysis of the functions of the part affected, without fever, spasm, or other evidences of irritation. The prognosis is hopeless; treatment without avail.

SPINAL ANÆMIA.

Under the name of spinal anæmia is described by various authorities a hysteroidal condition (see page 409) which, so far as we have any definite information, has no connection or relation with anæmia of the spinal cord. We have little knowledge concerning true spinal anæmia, but it is certain that a violent hemorrhage, or a violent purgative, will occasionally produce a partial paraplegia which is recovered from in due time; and it is natural to explain such paraplegia by the theory of loss of power in the spinal centres from failure of proper blood-supply. In profound essential anæmia tingling and numbness, with some loss of muscular power in the legs and arms, are sometimes seen, and may be due to lack of blood in the cord. Degenerations of the cord certainly occur, however, in pernicious anæmia, and if during anæmia a complete paraplegia develops it is probably always hysterical or organic. The loss of functional activity of the spinal cord from bloodlessness is to be treated by absolute rest, highly nutritious diet, iron, strychnine, bitter tonics, etc.

HYPERÆMIA OF THE SPINAL CORD.

Concerning hyperæmia of the cord itself we have no definite knowledge. It is possible that various neurasthenic symptoms are due to loss of power in the blood-vessels of the cord and consequent local congestion. Hemorrhage into the cord is said to have been produced by excessive coitus, and probably in sexual exhaustion local weakness of the blood-vessels in the lumbar cord from frequent excessive distention is an influential factor.

It is further probable that there is a form of ascending paralysis, so called (see Landry's paralysis), in which the lesion is an intense congestion of the enormous plexus of veins surrounding the spinal cord and of the vessels within the cord. Certainly we have seen cases in which, after excessive exertion with exposure, numbness, partial paraplegia, and lessening of reflex activity have rapidly developed, and increased and ascended until death from paralysis of respiration has occurred; and at the autopsies we have found great engorgement of the vessels with serous exudation, or, in other words, a condition of the cord similar to so-called serous apoplexy in the brain.

Whenever, after violent exertion, violent sexual excitement with repeated coitus, or the like, symptoms suggesting congestion of the spinal cord are present, if the patient be robust, general blood-letting and wet and dry cupping along the vertebral column may be practised. If numbness and weakness persist, absolute rest in bed on the side (not on the back) should be prescribed, and ergot freely given (a drachm a day of the extract). In the very beginning of such an attack large hypodermic injections of the extract of ergot might advantageously be given.

ACUTE SPINAL MENINGITIS.

DEFINITION.—An acute inflammation of the spinal membranes, not syphilitic.

Some authorities distinguish between acute *spinal pachymeningitis*, or inflammation of the dura mater, and *acute leptomeningitis*, or inflammation of the arachnoid or pia mater. Almost invariably, however, all the membranes are affected, though it is alleged that septic and traumatic meningitis may be confined to the dura mater.

ETIOLOGY.—Acute spinal meningitis is affirmed to be sometimes produced by severe exposure to heat or cold, but in the great majority of cases it is due to infection. It occurs most frequently in young males.

PATHOLOGY.—In its pathological anatomy spinal meningitis is completely parallel to cerebral meningitis.

SYMPTOMATOLOGY.—Acute spinal meningitis usually begins with a febrile disturbance, accompanied by severe pain along the whole back, soon spreading throughout the body and limbs. The spinous processes are not tender, but motion of the body or limbs usually increases the pain. Violent tonic spasms come on in a very few hours, spreading from the back muscles to the whole body, and result in opisthonotos or other forced positions of the trunk and rigid flexion of the limbs. The spasm and pain produced by attempts at motion give an appearance of paralysis, but until very late in the disorder there is little true loss of muscular power. The reflexes are grossly exaggerated ; retention of urine and constipation commonly develop early ; hyperæsthesia is in most cases an early symptom, but may finally give place to anæsthesia, which is apt to occur in patches. Consciousness and intellection are not primarily affected, but there may be delirium and even coma before death.

DIAGNOSIS.—The only disease with which spinal meningitis can readily be confounded is rheumatism. The rapid development of the symptoms, the universality of the pain and spasm, the wide-spread hyperæsthesia, the exaggeration of the reflexes, and the general severity of the attack characterize the spinal disease.

PROGNOSIS.—Death may occur in two days, but if the patient survive a week, recovery, with more or less permanent disablement from contractures and paralyses, is the ordinary result. The more severe the symptoms the more guarded should be the prognosis. A sudden great rise of temperature or a serious interference with respiration or with deglutition usually presages death.

TREATMENT.—Blood-letting, general or local, the rapid induction of ptyalism by mercurial inunction, and the use of very active counter-irritation over the back, find their justification in the seriousness of the local disease in cases of sthenic type with no apparent infection ; in infectious cases mild counter-irritation constitutes about the whole direct treatment.

Absolute quiet and rest should be enjoined. The food at first should be liquid, non-stimulating, and moderately nutritive, but afterwards should be both nutritive and stimulating. During convalescence absolute care must be enjoined to protect from any chilling of the surface or any fatigue of the nervous or the muscular system.

CHRONIC SPINAL MENINGITIS.

DEFINITION.—A chronic inflammation of the spinal membranes.

Chronic spinal meningitis may be localized or general.

The most important local form is the *cervical pachymeningitis* of Charcot, which usually, if not always, is of syphilitic origin. After death the cervical spinal membranes are found enormously thickened and accompanied by secondary changes in the cord and nerve-roots, due to pressure. Two stages of the disease are recognized: first, that of irritation; second, that of paralysis; but the separation of these two stages is artificial. Pain in the back of the neck, extending into the head and along the arms, associated with stiffness and muscular weakness of the parts and increased by movement, constitutes the chief symptom of the first stage. Vesicular or other trophic skin-lesions due to inflammation of the nerve-roots are often present. The second or paralytic period is characterized by loss of muscular power, with muscular atrophy especially affecting the domain of the ulnar and median nerves, and followed by contractures which extend the hand and the forearm and flex the fingers into a claw-like position. The disease may finally ascend upward and downward and give rise to widespread symptoms of chronic meningitis. The treatment of cervical pachymeningitis should be actively antisyphilitic until success or failure is reached. Long-continued and severe counter-irritation (with the actual cautery) is also indicated. In the advanced disease no treatment is of avail.

Generalized chronic spinal meningitis is almost always alcoholic, specific, or traumatic. Its most important symptoms are pain in the back and limbs, increased by active or passive movements; hyperæsthesia, perhaps associated with spots of anæsthesia; heightening of the reflexes, or in advanced stages loss of the reflexes; muscular contractures, followed by loss of power and wasting of the muscles. Mercurials and iodides should be administered, absolute rest should be enjoined, and the actual cautery or other counter-irritant should be freely and repeatedly used, unless failure to improve shows that the case is hopeless. Analgesics, hypnotics, and similar remedies may be given *pro re nata*.

SPINAL ABSCESS.

Abscess of the spinal cord is probably always infectious: it may be suspected when severe spinal symptoms rapidly develop during a septicæmia. It ends in a destructive myelitis, which is almost invariably fatal.

SPINAL TUMOR.

Almost any form of neoplasm may be formed inside of the spinal column, producing symptoms which vary according to the size, position, and rapidity of growth of the tumor. The symptoms can best be studied as cord-symptoms and root-symptoms.

The cord-symptoms are sharply limited,—loss of motion and sensation, with heightening of the reflexes, and without trophic changes. If the tumor be of very slight growth, one side of the spinal cord may be so pressed upon as to lose its functional power much earlier than the other part; indeed, one function of one-half of the cord may be primarily affected. In this way a sensory or a motor monoplegia or a coexistent sensory and motor monoplegia may result from a tumor.

The root-symptoms are most marked when the tumor is of such character that it spreads into and involves the nerve-roots. They consist of pain, spasm, and motor and sensory paralysis, followed, it may be, by atrophy of the affected muscles, with reaction of degeneration.

The pain of a spinal tumor may be slight or atrocious. It may consist of a heavy localized ache deep in the back, but usually is a burning, lancinating, tearing pain, following the course of the nerves and girdling the body in an agony, or more rarely affecting the arms or legs. Complete anæsthesia may finally be produced without abatement of the pain, constituting a form of anæsthesia dolorosa which is almost characteristic of cancer.

Diagnosis.—The recognition of a spinal tumor depends upon the slow simultaneous development of sensory and motor paralysis, without trophic changes or loss of reflexes, and the abrupt limitation of this paralysis by a narrow zone of partial palsy. In other words, the connection between the cord and the brain is severed, and an abrupt line of motor and sensory paralysis marks the seat of the separation of conduction. Trophic changes can occur only as the result of secondary lesions of the cord. A tumor is distinguished from the transverse myelitis of spinal caries by the slowness of its development and by the lack of tenderness upon direct or indirect pressure upon the vertebræ. Atrocious pain is characteristic of the cancerous tumor, but the caries which produces a transverse myelitis may also be attended with great pain, due to a secondary inflammation of the sensory nerve-roots.

Treatment.—There is no effective medicinal treatment for a non-specific spinal tumor. If the tumor have been accurately located, and be not cancerous or syphilitic, surgical interference will be justifiable.

ACUTE ASCENDING PARALYSIS. LANDRY'S PARALYSIS.

Definition.—An acute disease of uncertain pathology, characterized by the rapid and progressive development of motor paralysis, usually commencing in the lower extremities, and finally involving the whole muscular system; without trophic alterations, or pronounced disturbances of sensation, but with enlargement of the spleen and lymphatic glands.

ETIOLOGY.—Acute ascending paralysis has in various cases been attributed to exposure or recorded as a sequel of an infectious disease, but is still of very doubtful etiological relations, especially in view of the fact that the recorded cases probably represent several different diseases.

MORBID ANATOMY.—Focal myelitis and multiple neuritis have been recorded as the essential lesions of cases of ascending paralysis, but do not belong to the disease. Typical ascending paralysis may result in death without a lesion either of the nerve-centres or of the nerve-trunks that can be recognized by our present methods. The excessive venous congestion and increase in the cerebro-spinal liquid found after death in some of these cases suggest that the disease is an acute congestion of the spinal cord with œdema; but, on the other hand, in some of the cases there is no such congestion, so that it would seem either that there are two distinct diseases, one congestion of the cord, one something else having the same symptoms, or else that the congestion is a secondary result of the original disease, and not the cause of the symptoms. Under these circumstances two theories of the disease naturally confront us: the one that the lesion is an acute inflammatory hyperæmia, which may disappear at death or may produce changes which we can recognize; the other that the symptoms of Landry's paralysis are due to a toxæmia. The second of these theories seems at present the more probable. The enlargement of the spleen and of the lymphatic glands strongly indicates that the disease belongs to the infectious class and is due to the presence of micro-organisms. In corroboration of this, Baumgarten and Curschmann assert that they have found bacteria in the enlarged glands; but Westphal, Kahler and Pick, and others have looked for them in these places without success. Centanni found in a typical case of Landry's paralysis a peculiar bacillus, which existed in moderate numbers in the spinal cord, but in great numbers in the peripheral nerves, where it formed colonies which had resulted in structural alterations of the nerve-fibres, not of the nature of neuritis, but of a neuromycosis. This discovery of Centanni's has been confirmed by Eisenlohr, who in two cases found a wide-spread, partially interstitial, partially parenchymatous alteration of the peripheral nerves extending to the extreme end-filaments of the nerves, caused by the presence of various forms of micrococci, which micrococci also existed to some extent in the spinal cord, where they appeared to have set up an acute myelitic process.

Further, a series of symptoms like those of Landry's paralysis have been produced by inoculations of microbes from typhoid-fever spleen and other sources. Oettinger and Maresco in a Landry's paralysis after variola detected streptococci in the cells and blood-vessels of the spinal cord. Albu failed to find any organisms in a case of alleged ascending paralysis, in which, however, there was no enlargement of the spleen. It is most probable that Landry's paralysis comprises several diseases, and among them an infectious myelitis in which death may occur before the changes of the spinal cord have gone far enough to be recognized.

SYMPTOMATOLOGY.—Ascending paralysis may come on abruptly during apparent health, or be preceded by malaise and paræsthesia. The first distinct manifestation is a feeling of numbness and weight in the feet, soon followed by loss of power, which with the numbness rapidly increases until in a few hours the subject cannot stand. The paralysis continues to increase and to mount higher, involving the muscles of the trunk and of the upper extremities. Dyspnœa develops from the loss of power in the diaphragm and the respiratory muscles ; deglutition becomes difficult or impossible ; the voice grows feeble and inarticulate, or it may be suppressed ; and death results from paralytic asphyxia in two or three days. As the paralysis ascends it involves all the muscles in its course, but the muscles of the face and eyes are rarely attacked, probably because death occurs before they are reached. Intelligence and consciousness are preserved until the last. The sensory symptoms consist of a slight feeling of numbness, with perhaps dulling of sensation. The sphincters are not affected, unless it be very late in the disorder. The reflexes suffer with the motor paralysis. The muscles do not undergo changes in bulk or in their relations to electrical currents.

Since enlargement of the spleen was first noted by Westphal it has been frequently found, and it is probably a constant symptom in one form of Landry's paralysis. Enlargement of the lymphatic glands is also present in a majority of cases. Absence of marked fever appears to be a characteristic of the disease, although distinct elevation of temperature has been noted. Cases have been reported of alleged Landry's paralysis in which the medulla was the first part of the cord to be attacked, so that speech, deglutition, and respiration were primarily affected. It is, however, very doubtful whether these cases should be considered as instances of the disease.

DIAGNOSIS.—From acute central myelitis, and also from ordinary multiple neuritis, Landry's paralysis is distinguished by the absence of disturbances of sensibility, of trophic changes, of alterations of the electro-muscular contractility, of high fever, and of early paralysis of the sphincters, as well as by the rapid loss of the reflexes. Cases have been reported in which a neuritis confined to the motor roots has been said to have caused symptoms scarcely to be distinguished from those of ascending paralysis.

The enlargement of the spleen and of the lymphatic glands should always be carefully examined for. If both are absent, the case probably represents some form of the disease different from that in which these parts are enlarged. It is possible that various poisons may induce similar spinal symptoms, and there is plausibility in the theory that ascending paralysis following severe exposure is of rheumatic origin.

Prognosis.—The prognosis is always very grave. In rare cases arrest of the ascent takes place and recovery results.

Treatment.—Absolute rest, careful feeding, symptomatic treatment, and the free use of the extract of ergot in the hope of diminishing spinal congestion, constitute about all that can be safely done in Landry's paralysis. If a case should present itself having arterial excitement, venesection might possibly be justified; while the very free use of salicylates would be called for by a strong suspicion of rheumatic origin.

ACUTE MYELITIS.

Definition.—An acute inflammatory affection, involving the whole thickness of the cord and producing paralysis of motion and sensation, with trophic changes.

Etiology.—Acute myelitis is most frequent between the twentieth and the fortieth year of life, and occurs more often in men than in women. It may be of infectious origin, and, probably in this way, occasionally develops in chronic tuberculosis, in chronic syphilis, and in the puerperal state. It may be induced by traumatism and by inflammatory diseases of the parts immediately adjacent to the cord. Sexual excesses, unnatural coitus, and violent bodily exertion are assigned causes. That the combination of excessive physical exertion with exposure will produce it is indicated by the large number of cases which have occurred among soldiers during winter campaigns.

Morbid Anatomy.—The myelitic cord may be completely diffluent, or it may be soft, yielding a reddish-yellow or brownish and, to the naked eye, structureless section. Any portion of the cord may be attacked, but the dorsal cord is especially liable. The whole thickness may be completely disorganized, but the gray matter in most cases is the centre of attack: hence the term *central myelitis*. Not rarely the disease takes the form of small foci scattered through the cord, constituting *insular* or *disseminated myelitis*. Rupture and necrosis of capillaries, and even of large blood-vessels, give varying brownish or reddish tints to the tissue, or give origin to small blood-clots (*hemorrhagic myelitis*). The line between sound and diseased tissue is never abrupt, the latter always shading into the former. When, however, the acute disease passes into the chronic condition the neurogliar tissue around the focus of inflammation undergoes hyperplasia and sclerosis, so that the focal débris is shut off and may finally be absorbed, leaving the sclerosed tissue as a thick-walled cyst. The nerve elements in acute myelitis suffer rapid degeneration. The multipolar cells become irregularly swollen, with their processes broken and shrunken, their structure coarsely granular, or in later stages formless, and finally break up into débris. The nerve-filaments first enlarge and have their axis-cylinders especially thickened, then they become moniliform, and finally they break up. In the end the myelitic tissue consists of débris, with remains of nerve cells and filaments mixed with drops and masses of myelin, large granular corpuscles, pigmented granules, altered blood-corpuscles, etc. *Gray myelitis*, so called, is the condition in which there has been an attempt at recovery with absorption of débris, and the formation of connective tissue passing into sclerosis. Under no circumstances is there any repair of damaged nerve-filaments.

The general myelitic process is commonly viewed as inflammatory, and as consisting of three stages: first, hyperæmia and exudation (*red softening*); second, fatty degeneration and resorption (*yellow softening*); third, terminal stage (cysts, sclerosis, etc.). Some authorities, however, deny that myelitis is in truth an inflammation; while Spitzka affirms that there are no records in literature to show that there is a primary stage of congestion and leukocytal inflammation.

SYMPTOMATOLOGY.—Fever usually ushers in an attack of acute myelitis, but may be entirely absent even in a fatal case. It also may rapidly subside, but usually persists without the temperature rising above 101° or 102° F. In some cases there are irregular paroxysms of intense fever, whilst a great rise of temperature is not a rare precursor of death. The spinal symptoms are: first, those of irritation; second, those of paralysis. The symptoms of irritation belong to the earliest stages of the disease, and are usually soon lost, in some cases to reappear when partial convalescence develops. They consist of twitching of the muscles, tonic or clonic contractures, exaggeration of the reflexes, tingling, numbness, violent formication, shooting pains, excessive distress during micturition and defecation; even after a complete abolition of sensibility violent pain may be left, constituting a true *anæsthesia dolorosa.* The suffering may be intense, with bitter complaints of a burning girdle of molten iron, a thrusting of heated needles through the limbs, a drawing or tearing of muscles from the body, etc. Excessive sensitiveness of the spinous processes, especially to hot or cold applications, is not rare. True hyperæsthesia is not common, but a peculiar, diffused, painful, vibrating sensation may in the early stages be produced by touching a part (*dysæsthesia* of Charcot). True sexual excitement is never present, but painful priapism may last even into the paralytic stage. Complete motor and sensory paralysis, with flaccid muscles and loss of all reflexes and of power over the sphincters, marks the paralytic stage. Usually, but not always, the paralysis is in the form of a paraplegia. The upper portion of the cord may be in the first stage whilst the lower is in the second stage of the disease, so that the symptoms of irritation will be present in the upper portion of the body whilst below all is paralyzed. Vaso-motor paralysis may show itself in a temporary rise of temperature in the paralyzed parts, but the affected extremities soon grow cold and bluish, and are often swollen by a diffused œdema. The excretions rapidly become abnormal; even after two days the urine may be highly alkaline, bloody, muco-purulent, and loaded with the crystals of triple phosphates, whilst the perspiration is excessive, irregular, and altered in quality. Muscular atrophy, with reaction of degeneration and finally complete loss of electro-contractility, appears very early. The trophic bed-sore, *decubitus acutus*, usually attacks the sacro-gluteal region, but occasionally appears in the heels or other portions of the body. The first warning consists of one or several dark-red or violet erythematous patches, variable in extent and irregular in shape. Within twenty-four or forty-eight hours reddish or brownish vesicles or bullæ form in the central portions of the erythema. In rare cases, under careful management, the blebs wither and disappear without further symptoms. Usually, however, the elevated epidermis drops off, leaving a bright-red surface with bluish or violet points or patches, and with swelling and sanguinolent infiltration of the surrounding tissue. Quickly the reddish surface becomes blackened, and a slough of variable extent forms. The whole buttock may thus melt down in the course of a few hours. Sometimes the process is arrested and the slough separates, but oftener the process continues, and, unless the patient dies too quickly, the deeper muscles, with the nerve-trunks and arterial branches, are laid bare, and finally the bones themselves are exposed.

In acute myelitis the vision is not usually affected, but when the disease is situated very high up in the cord irregularities of the pupil and even strabismus may be produced; whilst contraction of the field of vision, amblyopia, and amaurosis have been recorded as results of complicating optic neuritis.

Acute myelitis varies indefinitely in the rapidity of its course, but for the purposes of discussion we may recognize three types, with the understanding that they grade one into the other.

Foudroyant or *explosive myelitis* (*myelitis centralis*) commences abruptly, reaching in a few moments or hours the stage of paralysis, with complete anæsthesia, loss of motor power, abolition of reflexes, and trophic changes. This form of myelitis may occur without violent constitutional symptoms, especially when there is hemorrhage into the cord (*hæmatomyelitis*) with its almost abrupt paralysis. Usually, however, there are more or less intense fever, delirium, coma, and even convulsions. Death may occur in one or two days from paralytic asphyxia, or the fatal termination may be reached in from one to two weeks from sepsis due to decomposing urine and sloughing bed-sores.

In the type of acute myelitis may be placed those cases in which the paralysis requires from one to two weeks to become complete. Disturbances of cerebration are much less common than in the explosive disease, and death from septic fever and exhaustion may be delayed from one to several months, or occasionally an imperfect recovery may be secured.

In the type of subacute myelitis may be placed those cases in which more than two weeks are required for a full development of the paralysis, or in which the palsy never becomes complete. These cases may end in death, but more frequently they pass into chronic myelitis, with partial paraplegia, which may last for years or be even imperfectly recovered from.

Varieties of myelitis are sometimes described corresponding to the seat of the disease. Of these we shall notice only *myelitis cervicalis*, in which the muscular atrophy is confined to the arms, whilst the legs are in a condition of spastic paralysis, both arms and legs being anæsthetic. Oculomotor symptoms are also common, whilst dyspnœa and rapid death may result from paralysis of the respiratory muscles. When the cervical myelitis is produced by a traumatism the patellar reflexes are primarily lost, but, according to the general teaching of authorities, return. Bastian, however, affirms that this is incorrect, and that if in any case the reflexes are not entirely and permanently lost it may be certainly diagnosed that the injury to the cord is not transversely complete.

A myelitis may be transversely incomplete, when the anæsthesia and to a less degree the motor paralysis will also be incomplete.

In a form of myelitis which follows small-pox, measles, and other infectious diseases, in which the lesion consists of very minute foci scattered through the whole of the nerve-centres from the pons to the cauda equina, the chief symptoms are disturbances of speech, tremors, and weakness, with an ataxia so pronounced that the affection has been spoken of as an *acute ataxia*.

DIAGNOSIS.—For the diagnosis between myelitis and Landry's paralysis, see page 550. Myelitis is distinguished from acute poliomyelitis by the presence of pain, by the muscular spasms in the early stages, and by the trophic changes in the skin and cellular tissue. From peripheral neuritis myelitis is to be distinguished by the intensity and the rapidity of development of its paralytic phenomena, and by the muscular atrophy and other changes, as well as by the absence of tenderness of the nerve-trunks. In subacute myelitis trophic changes may occur very slowly, but the nerves are not tender, and the severe, continuing, localized pains of neuritis are wanting.

PROGNOSIS.—The course of myelitis is almost uniformly in proportion to the original severity and extent of the symptoms. Even the mildest case is, however, very serious, and usually ends in some disability.

TREATMENT.—If the theory be correct that acute myelitis is an inflammation, the strenuous use of venesection, cold, and other antiphlogistic measures is justified by the danger of the disease. There seems to be, however, no weighty clinical evidence to prove that these measures have distinct influence upon the development of the disorder. Nevertheless, if the general constitutional condition be good, blood may be drawn from the arm, and active blood-letting by means of leeches or dry cups be practised, as is advised by many authorities. Ergot may be employed for the purpose of diminishing congestion, and, if not effective, certainly is harmless. In the beginning it may be given hypodermically, and afterwards from ten to fifteen grains of the official extract should be administered every three hours until gastric disturbance, ergotic coldness of the surface, or the continued progress of the disease indicates its withdrawal. The production of diaphoresis by the hot bath or the hot pack is especially recommended by Erb when the premonitory signs of myelitis appear directly after exposure to cold; but the reputation of these measures probably largely rests upon the relief which has been obtained in cases in which rheumatic pains and general muscular soreness have been mistaken for the precursors of myelitis.

During an attack of myelitis the warm bath is very grateful to most patients, and should always be tried. In employing it, absolute precautions must be taken that the patient himself make no effort whatever, a sufficient number of nurses to lift him being provided. The temperature of the bath should be in the beginning 90° F., to be increased later if it be found advisable. The duration of the bath should at first be about ten minutes, but it should be rapidly increased almost indefinitely, according as it is found to agree with the individual case. The bath may be given once, twice, or three times in the twenty-four hours.

The very active administration of mercury, as recommended by some writers, seems to us of doubtful advisability, and, whilst it may be well to give calomel in small, repeated doses, the practitioner should content himself with the slightest ptyalism. Strychnine, which has been recommended by authorities, has in our experience increased the symptoms; and on theoretical grounds it does not seem to be indicated. There is not the slightest reason for supposing that belladonna, derivation to the intestines, or the production of diuresis by means of the ingestion of large quantities of alkaline waters, as recommended by Erb, is of any service whatever. Of course, if excretion fails from want of nerve-influence, care should be exercised to see that the emunctories are kept active. The local application to the spine of long, thin rubber bags containing ice may be of service, and probably is never injurious. Counter-irritation by means of the actual cautery or the blister has been largely practised, and is strongly commended by some writers. The grave danger, however, of developing ulcers and wide-spread gangrene attends the use of remedies of this class, and certainly no counter-irritants should be applied to the skin which is already distinctly anæsthetic, or to a part which may be exposed to continuous pressure. Spitzka, on theoretic grounds, believes that counter-irritants applied to the lower legs and the feet are of much more service than when applied to the back. The use of the galvanic current, as occasionally practised, seems to be an outcome of a childish credulity.

The nursing during myelitis should be of the most careful character. From the very beginning, so soon as there is any reason to suspect the existence of the disease, rest in bed of the most absolute character should be prescribed. The feeding, the making of the personal toilet, etc., should be done by an attendant. A total abstinence from muscular movements should also be enjoined, even after symptoms of convalescence have appeared. At such time even the least muscular exertion may produce a relapse. If recovery occur, the avoidance of muscular fatigue and of sexual intercourse for one or two years after the attack should be strictly enjoined.

As the dorsal decubitus is believed by some authorities to increase congestion of the spinal cord, the patient should be kept as much as can be upon the side, or, better, if he can be made comfortable, upon the face, so as to remove all pressure from those portions of the body which are most prone to the development of gangrenous lesions. The greatest care must be exercised to prevent, if possible, bed-sores: hence the buttocks and the heels must be guarded from pressure, and the surfaces must be kept perfectly dry. The patient should always be put upon a water-bed, so covered with one or more heavy woollen blankets as to avoid any chilling of the body. All irritating applications to bed-sores must be avoided, and antisepsis should be carried out as thoroughly as may be without too much interference with the part.

From the first hour of a myelitis the strictest attention must be paid to the bladder, as urinary retention, with its concurrent cystitis and pyelitis, is a most serious complication. In most cases continuous catheterization and irrigation of the bladder once in the twenty-four hours with a solution of boric acid or other weak antiseptic solution may be practised. In all handling of the parts, as in catheterization, strict attention should be paid to antisepsis. The soft, flexible rubber catheter should be employed. It may be retained by adhesive straps, or preferably, as suggested by Spitzka, by using a perforated condom fixed to the catheter and then fastened to an inguinal bandage. To the catheter should be attached a soft rubber tube ending in a urinal. The condition of the bowels should always be attended to, mild laxatives being employed and aided occasionally by stimulating injections.

CHRONIC MYELITIS.

Definition.—Chronic inflammation of the spinal cord, involving to a greater or less degree its transverse section for a considerable length of the cord.

Etiology.—Chronic myelitis may originate in an acute myelitis or be chronic from the beginning. The causes indicated are traumatism, exposure to cold, sexual excesses, syphilis, propagation of irritation from peripheral nerves, and diseases of the blood-vessels.

Morbid Anatomy.—In chronic myelitis the cord is usually somewhat hardened. There appear to be two distinct forms of structural alteration. In rare cases the whole body of the cord is filled with moderately large neuroglia-cells, pressing upon and destroying the nerve-elements, but showing little or no tendency to the formation of fibres.

Such cords in our experience yield a section which is reddish to the naked eye.

A more usual change is a sclerosis which yields a smooth, grayish, or yellowish-gray section, constituting one of the conditions which have been called gray degeneration of the cord. Such a cord contains an excess of neuroglia-cells, many of them enlarged and furnished with proliferated nuclei and numerous processes (the so-called Deiters's cells), but it is especially composed of wavy, fibrillated bundles of fibres. The nerve-fibres are swollen, often irregularly so, with sheaths and axis-cylinders abundantly and irregularly enlarged, or they are atrophied, with destruction at first of the medullary sheath and afterwards of the naked axis-cylinder. The ganglia-cells are variously altered, clouded and swollen, or more frequently atrophied, shrunken, indurated, strongly pigmented, or finally changed into irregular, unrecognizable structures. Among the nerve-elements can usually be seen granular corpuscles, corpora amylacea, and pigment granules, whilst the walls of all the blood-vessels are enormously thickened and the perivascular spaces crowded with cells and exudate.

The membranes are commonly in a state of chronic inflammation, and the nerve-roots are frequently in a condition of neuritic atrophy.

SYMPTOMATOLOGY.—In primary chronic myelitis the onset is usually very insidious and marked by fluctuations. Slight sensory disturbances, paræsthesia, partial anæsthesia, girdle sensation, loss of endurance, especially in walking, and uncertainty of gait may develop so slowly and with so many remissions that the subject scarcely knows from what time to date the beginning of his disorder. Constipation, loss of sexual power, and vesical weakness may be among the earliest symptoms.

In the fully developed disease the chief complaint is usually a loss of power, depression of function predominating over irritation. Violent pains and muscular spasms are not common, although very frequently the legs draw up in the bed or suffer from vibratile contractures, especially at night. The muscles are usually rather stiff than relaxed, and occasionally when the patient can walk the spastic gait of lateral sclerosis is present in a moderate degree. The reflexes are in the early stages of the disease almost invariably exaggerated. Often this exaggeration is very marked, the slightest touch upon the patellar tendon, tickling of the soles of the feet, or even stroking of the thighs, provoking not only local muscular contractions, but also general wide-spread movement. Ankle-clonus may be present. In the later stages of the affection the reflexes may be diminished or even entirely lost, but this rarely happens until the gray matter of the cord is disorganized, so that loss of the knee-jerk is almost invariably associated with atrophy of the muscles or other trophic change which belongs to the last stages of the disease.

Vesical weakness or paralysis, with retention or dribbling of the urine, is almost universal, and is very liable to produce a paralytic cystitis, which, creeping up the ureter and involving first the pelvis and then the secreting structure of the kidney, may end in a fatal renal degeneration.

DIAGNOSIS.—Chronic myelitis is distinguished from the subacute disorder chiefly by the slowness of its development. Locomotor ataxia and other spinal tract diseases develop more slowly than does chronic myelitis, and are further distinguished by their not involving simultaneously all the functions of the cord, as does myelitis.

PROGNOSIS.—During the very slow, prolonged course of chronic myelitis the general health and the bodily nutrition of the patient may be well preserved, provided cystitis or renal complications can be avoided. Complete recovery is rare, but arrest of the disease and partial recovery may happen.

TREATMENT.—We do not believe that drugs have any direct influence on the progress of chronic myelitis. Silver nitrate, at one time much used, has been in our experience inefficient. Potassium iodide we have never seen do good. Large doses of mercury are not to be thought of, but the long-continued administration of the tonic dose of corrosive sublimate (one-sixtieth of a grain three times a day) may be useful. Counter-irritation is of doubtful value; to have any influence it must be severe. The actual cautery may be applied over a considerable extent of the affected cord, with such light touches as only to destroy the epidermis. If the part have been previously frozen, the pain of the application will be trifling; whilst the after-pain is usually not so severe as that of the blister. The Paquelin cautery may in this way be used every ten days, or as often as the part heals. Hot baths or hot packs at short intervals, especially hot packs to the legs, are thought to be of service. Electricity is rarely, if ever, directly useful, but may sometimes be used locally to prevent wasting of the muscles.

The hygienic treatment is exceedingly important, and by change of air, careful selection of diet, and all other means, the general health should be improved as much as possible. Mental depression, over-exertion, and fatigue are to be sedulously avoided, and as favorable a view of the case as possible should be given to the patient. Rest on the bed or couch is often of the greatest service, and when conjoined with daily use of massage may be maintained for a length of time without endangering the general health or producing muscular relaxation. When circumstances favor it, the patient may with great advantage spend a large portion of his time on the bed, couch, or lounge in the open air.

COMPRESSION MYELITIS.

DEFINITION.—A myelitis due to pressure from a tumor or from displaced or diseased vertebræ.

ETIOLOGY.—Compression myelitis may be produced by any form of tumor or by traumatic injury to the vertebral column, but in the great majority of cases is the result of Pott's disease.

MORBID ANATOMY.—After death the affected part of the cord is found sometimes softened, sometimes hardened, with changes in it similar to those which have been described in acute and chronic myelitis, but localized in a very small segment of the cord. It is asserted of this form of myelitis that it is entirely possible for nerve-tubules which have undergone more or less complete destruction to be restored. Certain it is that after the paralysis has been complete or nearly so for many months, or even, as in one case recorded by Charcot, for two years, the spinal cord has recovered its functional power, and also that in such cases the cord has been found reduced to about one-third of its normal volume at the point of compression, although containing an abundance of perfect nerve-tubules. Babinski has proved that compression of the spinal cord may give rise to a paraplegia very intense and even complete, lasting for months, without causing any appreciable lesion in the cord.

As it does not seem probable that spinal nerve-fibres once destroyed can be regenerated, the most plausible explanation of the cases of recovery is that the paralysis has been largely due to compression of nerve-fibres still intact, and that although many fibres may have been destroyed by the myelitis, yet enough have been left to carry on the function of the cord when the pressure was removed.

SYMPTOMATOLOGY.—The symptomatology of compression myelitis may be divided into those symptoms which are due to the disease producing the compression and those which are due to the myelitis. In many cases, especially in Pott's disease, nerve-roots become involved in the original disease, so that violent neuritic pains, trophic changes, anæsthesia, etc., form a part of the symptoms, not of the myelitis, but of the complicating neuritis. These pains usually precede the onset of the myelitis. The symptoms due to the myelitis itself are progressive paralysis of sensibility and of motion in the parts below the lesion. If the original lesion be of such character as first to involve one side of the cord more than the other, the paralytic symptoms may be to some degree unilateral. The patellar reflexes are usually preserved, or even exaggerated. Weakness of the bladder deepens into complete paralysis, while a similar condition of the intestines and rectum is often a very troublesome symptom.

DIAGNOSIS.—Compression myelitis is to be especially distinguished from an ordinary myelitis by noting its localized character and the presence of the compressing lesion. In view of the results obtained by Babinski, it seems impossible to state early in a case with certainty whether the loss of function is produced by a severe myelitis or simply by compression. The more profound and persistent the symptoms the greater the probability of serious inflammatory changes in the cord.

PROGNOSIS.—The prognosis of compression myelitis depends entirely upon the nature of the lesion which produces it. When this lesion is removable the prognosis is usually good. It is remarkable how the spinal cord in cases of Pott's disease will so adapt itself to the narrow channel finally formed for it that, generally, if the bone-disease be recovered from, the myelitis can be cured, even though the deformity be great.

TREATMENT.—The treatment of compression myelitis is largely the treatment of its cause. In cases of myelitis due to Pott's disease the systematic use of the cautery is advised by Charcot. The head of a Paquelin cautery should be applied as a series of points on each side of the gibbosity of the back, the application being repeated as soon as the parts have healed.

In recent cases of fracture, when there is reason to suspect pressure from dislocated portions of the bone, spinal trephining is a proper remedy. In most cases of Pott's disease it appears to us to be an unjustifiable procedure. In cancerous tumors surgical interference is improper, but where a morbid growth is neither cancerous, tubercular, nor syphilitic, and can be accurately located, an attempt at removal may be made.

ACUTE POLIOMYELITIS.

DEFINITION.—An acute degeneration of the ganglionic cells in the anterior horns of the spinal cord, characterized by paralysis and trophic wasting of the affected muscles.

ETIOLOGY.—Acute poliomyelitis may occur in an adult, but is essentially a disease of childhood, five-sixths of the cases developing in children under ten years of age. It is not hereditary; indeed, it appears not to occur with abnormal frequency in neuropathic families. It attacks males more often than females. According to Wharton Sinkler, it is more frequent in summer than in winter, at least in the Middle United States. The attacks appear in many cases to have been precipitated by over-exertion, especially by over-walking in very young children. Traumatism, difficult dentition, and acute exanthematous diseases are all credited as occasional exciting causes. The frequency of the disease in childhood seems to depend upon the fact that at this period of life the trophic centres are forced not only to maintain the nutrition of the muscles, but also to develop their structure, so that they (the trophic centres) are continually in a state of hyperfunctional activity, with a consequent hyperæmia, and are therefore easily thrown over the line of health into an inflammatory condition.

MORBID ANATOMY.—Degeneration of the multipolar ganglion-cells of the anterior cornua of the spinal cord is the essential lesion of anterior poliomyelitis. Two theories are in vogue as to the nature of these lesions: one attributes the changes to a primary idiopathic atrophy of the ganglionic cells; the other teaches that the cells are not affected primarily and apart from the other gray matter, but are involved in a limited central and focal myelitis.

The rarity of death in the very early stages makes the post-mortems but few; nevertheless, Drummond, Riesler, Kahlden, and Goldscheider have reported cases in which death occurred within a few hours or a few days.

Usually the parts immediately around the ganglionic cells have been affected, but in two cases very pronounced alteration has been found in the ganglionic cells with a nearly normal condition of the interstitial tissue. It has been further noted that the degree of change in the ganglionic cells is not proportionate to the amount of alteration in the surrounding tissue: so that, whilst it may be considered settled that in most cases the neuroglia is attacked, it would seem clear that the primary and chief lesions are in the cells themselves, and that therefore the disease is essentially an acute poliomyelitis, in which the tissue in the neighborhood of the ganglion-cells is usually but not always involved. The first change in the cells is a granular opacity, which may be accompanied by pigmentation. This is followed by disappearance of the processes and shrinking of the cells, with in many cases a change of the protoplasm into a clear substance which refuses stains; at the same time the nuclei become pale and disappear. More and more the cells become shrunken, irregular, ball-like, and finally they cannot be recognized. The neurogliar tissue is usually congested, the blood-vessels dilated, with their lymphatic sheaths infiltrated with leukocytes or surrounded by minute extravasations of blood. Round granular cells also occur in the neuroglia, nerve-tubes break up, myelin escapes, and finally there may be such general disintegration as to cause minute patches of red softening. The cells are attacked in foci ranging in length from one-hundredth of an inch to more than an inch. In a focus the destruction may be limited to certain groups of cells, or may attack all the groups.

In old cases of poliomyelitis the affected portion of the spinal cord is often shrunken, with the ganglionic cells entirely absent, and the nerve-tubules in the white columns wasted, stripped of their myelin, often without sheaths, surrounded by hyperplastic neuroglia or by amyloid bodies. The anterior nerve-roots suffer from an atrophy similar to that which follows section of the peripheral nerve. The motor nerve-trunks undergo a change, which, as shown by Leyden, may consist of a degenerative atrophy or of a neuritis. Changes in the white matter of the cord and also those in the nerve-roots are either trophic or due to a propagation of the inflammation by physiological or anatomical continuity of structure. As certain metallic poisons, such as lead and arsenic, are capable of producing an isolated neuritis or an isolated poliomyelitis, or a combination of the two diseases, it would appear that either poliomyelitis or neuritis may exist by itself, but that in some cases both affections are simultaneously developed. The walls of the arterioles may be so thickened as almost to obliterate their lumina, the change extending into the comparatively normal portion of the cord.

SYMPTOMATOLOGY.—Acute poliomyelitis is rarely ushered in by prodromes, and may be not only sudden in its onset, but without constitutional symptoms, the child awaking after a good night's rest paralyzed, or with almost apoplectiform abruptness developing weakness during the daytime. In most cases, however, there is a primary fever of moderate intensity, lasting from a few hours to three or four weeks, and in rare instances reaching a maximum temperature of 104° F. There is great variability in the cerebral condition; there may be no disturbance, or only an apathy which in a series of cases grades through stupor, then into coma; whilst the restlessness or isolated spasms present in some cases pass in others into convulsive twitchings, increasing in intensity to the fiercest of general convulsions. Pains in the back and in the limbs are usual, but are seldom intense. Anæsthesia and hyperæsthesia are so rare that their existence challenges the diagnosis. Vomiting may be present, and in some cases is very violent and intractable. The fever rarely lingers after the development of the lesion, but ends in an abrupt defervescence.

Although the paralysis is in most cases complete before it is recognized, it probably always takes some hours for its development, as a rapidly progressive paresis has been frequently noted, and as still more often a paralysis already complete in one limb has under observation spread to other parts. The paralysis varies indefinitely in its extent, but the face, the intercostal muscles, and the diaphragm almost invariably escape. During the period of acute constitutional disturbance there is often incontinence or more rarely retention of the urine, but true permanent paralysis of the bladder never occurs.

The subsidence of the constitutional disorder and the development of the paralysis are followed by a period of quiescence, which after from one to six weeks is succeeded by a peculiar, almost pathognomonic, regression of the paralytic symptoms. The extent of this regression varies so much that there is little relation between the final result and the amount of original paralysis. The improvement occasionally ends in complete recovery, but in the majority of cases after two or three months spontaneous amelioration ceases and some of the muscles settle into permanent paralysis.

During the second stage of the disease there is complete relaxation and loss of the reflexes in the affected muscles, with a rapidly progressive atrophy which is especially pronounced in those muscles which are to remain paralyzed, and is accompanied by changes in the relations of the muscles to electricity similar to those which follow division of a nerve. At first the change is simply *modal,—i.e.*, the muscle responds more sluggishly to galvanic currents than it normally does. Very soon, however, qualitative as well as quantitative changes appear. In order to detect these changes the current must be brought in direct contact with the muscles, for if the electrode be applied to the nerve-trunk it will be found that the electrical reaction is diminished in quantity but not altered in quality. If the negative pole (*cathode*) of a weak battery be placed over a normal muscle, but not over its motor point, a strong contraction occurs at the closure of the circuit; when, however, the positive pole (*anode*) is placed over the normal muscle, the contraction is much less: in neither case is there any contraction when the circuit is broken. In other words, with the normal muscle and a feeble current we obtain good cathodal closing contraction, slight anodal closing contraction, and no motion whatever at either cathodal or anodal opening. When a current of sufficient power is used, opening contractions are produced, and the anodal contraction is greater than the cathodal. The "reaction of degeneration" consists merely in a more or less perfect reversal of the above formula. The anodal (positive pole) closure then causes a stronger contraction than does the cathodal (negative pole) closure. When there is only a slight degree of degeneration present there is a correspondingly slight increase of anodal closing over cathodal closing contraction. A minimum degeneration would be indicated by an equality of the two closing contractions.

These changes expressed by symbols are as follows: An Cl C represents anodal closing contraction; An O C represents anodal opening contraction; Ca Cl C represents cathodal closing contraction; Ca O C represents cathodal opening contraction: $<$ represents is less than; $>$ represents is more than (the point of the $<$ being towards the lesser quantity).

Then the formulas are:

$$\left.\begin{array}{l} \text{An Cl C} < \text{Ca Cl C} \\ \text{An O C} > \text{Ca O C} \end{array}\right\} \text{muscle normal.}$$

$$\left.\begin{array}{l} \text{An Cl C} = \text{Ca Cl C} \\ \text{An O C} = \text{Ca O C} \end{array}\right\} \text{muscle in first stage of degeneration.}$$

$$\left.\begin{array}{l} \text{An Cl C} > \text{Ca Cl C} \\ \text{An O C} < \text{Ca O C} \end{array}\right\} \text{muscle in more advanced stage of degeneration.*}$$

After the reaction of degeneration has been established the galvanic irritability gradually fades out.

The fourth stage of poliomyelitis of many authors, that of permanent paralysis, is not a stage of the disease, but is a condition of wreck left by the disease. The distribution of the permanent loss of power varies indefinitely, but the paralysis is often in the upper extremities and is rarely symmetrical. Monoplegia is the most ordinary form; paraplegia is rare; crossed palsies and hemiplegias are still more infrequent. The affected part is limp or rigid, bluish, habitually weak, and without reflexes. When the destruction of the trophic centres is complete the muscles waste to a fibrous band, incapable of responding to any electrical current, whilst the development of the whole limb is retarded, so that in the growing child the extremity becomes shorter as well as smaller than its fellow. The trophic changes in the bone are not necessarily in direct proportion to those of the muscles, and the growth of the limb may be permanently arrested although the paralysis entirely disappears.

---

* In children it is often almost impossible to make practical test as to the reaction of degeneration. The failure of response first to the rapidly and later to the slowly interrupted faradic current is direct proof that trophic changes are taking place in the muscle, whether the action of degeneration can be brought out or not.

Owing to the failure of the tendons and muscles to support the joints, these become more and more relaxed, until at last the head of the bone may be entirely out of its socket. Neither during the acute stage nor in the chronic after-condition do trophic inflammations or destructive lesions of the skin occur.

As the years progress, various deformities arise in the affected limbs, caused by contractures, which may appear as early as four weeks after the beginning of the attack, but are usually late phenomena, and are situated chiefly in the muscles which have escaped entirely or in part. At the same time there is reason for believing that the interstitial development of fibrous tissue in the remains of muscles sometimes plays a part in the fixation of a joint. The original theory of Delpech, that the deformities are the outcome of contractions of sound muscles which have shortened on account of the failure of their natural opposition by antagonists, accounts for most of the deformities; but the influence of weight upon joints from which has been withdrawn their natural support of muscles and ligaments is not without effect. Thus, the weight of the body pressing on the arch of the foot, which has lost its natural stays, gradually displaces the bones from their normal relations until the arch is entirely flattened or the whole extremity distorted into some form of club-foot. The fact that pes calcaneus is very rare whilst talipes equinus is very common after infantile paralysis indicates that contractures are dominant factors in causing deformities, the talipes being due to the circumstance that the calf-muscles are much less frequently paralyzed than are the anterior tibial groups.

The deformities of poliomyelitis may affect any portion of the body. All varieties of club-foot, knock-knee and inverted knee, rigid flexion of the knees, kyphosis, lordosis, extraordinary scoliosis, subluxation of the thighs or of the humerus, claw-like distortions of the hands,—any of these may result, or the withered, shrunken limb, mobile almost as a rubber tube, may dangle from the trunk, an untoward memory of the past.

Acute poliomyelitis is in the adult very rare. When it does occur the symptoms are similar to those seen in the child, except that vomiting is more common and more severe and cerebral disturbances are less pronounced, and that in the after-time there is much less tendency to the production of deformities.

DIAGNOSIS.—The positive recognition of the true nature of an incipient poliomyelitis with grave constitutional disorder may be impossible, but suspicion should be excited whenever an ephemeral fever in a young child does not conform in its history or in any of its manifestations with any exanthem. The moment paresis is detected the diagnosis becomes plain.

The completeness of the palsy and the rapid alteration of the electrical relations of the muscles, together with the absence of nerve-pains and nerve-tenderness, demonstrate that the case is not one of peripheral neuritis, whilst the course of the paralysis and the occurrence of febrile and of trophic disturbances separate the affection from Landry's paralysis. Moreover, the latter disease is extremely infrequent in children, whilst acute poliomyelitis is extremely infrequent in adults.

PROGNOSIS.—Death in the first stage of poliomyelitis is exceedingly rare, so that in regard to immediate danger the prognosis is most favorable unless there be intense vital failure or implication of the muscles of respiration or of deglutition. No opinion should be given at this stage as to the probable extent and completeness of the final palsy, since there is no relation between the severity of the primary constitutional storm and the gravity and extent of the permanent disablement. Even in the second stage, when the paralysis has reached its maximum, the prognosis must be guarded, because a seemingly mild case will sometimes turn out most unfortunately, and a very wide-spread paralysis may clear up entirely. There is usually, however, in the second stage some direct relation between the present and the final condition of the patient.

The electrical condition of the muscles now becomes a very important factor in presaging the future. The earlier the change occurs the more serious the prospect; and, *vice versa*, if after three weeks the muscles still respond well to the faradic current, the recovery will be rapid and complete. When in an advanced stage the muscles are unable to respond to any electrical current, the case is almost hopeless. When the power of responding to the direct or galvanic current is retained, although the faradic current produces no effect, the prognosis becomes hopeful in direct proportion to the length of time during which the paralysis has lasted. The longer the period that has elapsed the better is the outlook, because the preservation of the galvanic contractility proves that the trophic spinal cells have still some power, and affords ground for the hope that, although unable to stimulate the muscular nutrition to recover that which has been lost, they may still be able to hold up a muscle whose nutrition has been artificially restored.

TREATMENT.—In a very robust child poliomyelitis, ushered in with violent constitutional disturbance, may be actively treated antiphlogistically, even venesection being allowable. Usually, however, the local abstraction of blood is the most that should be thought of, and after paralysis has appeared even this measure should be practised with great caution.

In the second stage of the disorder authorities recommend for the purpose of diminishing spinal congestion ventral decubitus, the continuous application of cold by means of ice-bags along the spinal column, the administration of ergot, potassium iodide, and mercury, and the use of the actual cautery or other violent irritant; in a word, the treatment of an acute myelitis. Erb and some other authorities recommend that the galvanic current should be applied without interruption for several hours daily, the positive pole being placed at the nape of the neck, the negative upon the lower end of the spinal cord or upon the affected muscle. There is, however, no good reason for believing that a galvanic current so applied reaches the spinal cord, and there is still less reason for believing that if it did reach the spinal cord it would do any good. In some cases the application may have a salutary mental effect upon the little patient and the parent.

The treatment of the second stages of infantile paralysis should be chiefly expectant, but extract of ergot may be given in as large doses as the stomach will bear, and calomel cautiously administered; the actual cautery may also be lightly but freely applied, provided the patient be old enough and intelligent enough for it to be used without causing spasms of terror. In the very young or timid, if it be decided to employ the cautery, ether anæsthesia should be induced without the patient knowing what is to be done.

During the stage of regression medicinal treatment should be limited to the use of tonics and the persistent administration of very minute doses of corrosive sublimate, whilst the health of the patient should be built up in all possible ways and the nutrition of the muscles maintained by the use of electricity, massage, etc.

In the fourth or permanent condition strychnine and phosphorus may be administered, in the hope of stimulating ganglionic repair. Tendencies to the development of deformities are to be mechanically combated and the muscles locally treated. In some instances hypodermic injection of the strychnine salts into the paralyzed muscle has seemed to do good.

In the local treatment of the muscles three distinct measures are available:

Mechanical vibratile treatment, combined with the application of heat (and perhaps also of a Junod's boot), by means of Zander's or some other similarly acting mechanism, certainly stimulates the capillary circulation and may be useful.

Massage and passive gymnastics have the same aims as the mechanical treatment just spoken of, and are to be used when they can be commanded: to accomplish anything, however, they must be employed very persistently as well as skilfully. It should be remembered that rubbing the skin by an untrained person is not massage, and does not, like that procedure, reach the deeper circulation: what is wanted is kneading of the paralyzed muscles.

Electricity has a limited value in the treatment of poliomyelitis. There is not the slightest reason for supposing that at any stage of the disorder it can affect the spinal cord or the nerve-trunk for good; and its influence upon the muscles themselves amounts to nothing if the trophic centres have been destroyed. An improvement of the muscles is of no avail unless the spinal cord recover its power; but the effect of partial rehabilitation of the ganglionic cells is greatly increased by maintaining the sensitiveness of the muscles. The application of electricity to the muscles may, therefore, be begun as soon as paralysis is detected, but the greatest caution is at this time necessary to avoid producing muscular fatigue or any reflex irritation of the nerve-centres. The daily séances should be short, and the current used just sufficient to produce feeble muscular contractions. For practical purposes the law formulated in H. C. Wood's "Therapeutics" some years since, that the current to be employed in cases of paralysis is that which will produce the greatest muscular contraction with the least pain, is sufficient as a guide in the selection of the current in any stage of the disease. Usually the faradic current fails entirely, so that the direct voltaic or galvanic current must be employed. In the advanced stage of the paralysis, if electricity has not been used, there is always hope that the amount of paralysis is greater than is necessitated by the condition of the cord: if the muscles respond at all to the electrical current, careful treatment should be instituted. Even if the muscles seem at first entirely dead, two or three weeks' trial of treatment should be made, as such muscles have been awakened by electricity.

The muscles should be immediately acted upon, one electrode being placed over the insertion and the other over the origin of the muscle, and from time to time one electrode being put on the motor point. The galvanic current may be slowly interrupted, but the effect is much greater if by mechanical arrangement instead of simple interruption there is a reversion of the current. If after eight weeks of electrical treatment no gain is achieved, nothing is to be hoped for.

In all cases of infantile paralysis it is essential to prevent, as far as may be, the development of deformities. Contractures are to be overcome, if possible, whilst forming, by thoroughly stretching the muscles morning and evening with the hand. When the contracture persistently increases, section of the tendons should be resorted to. The operation is simple and without danger, and experience shows that the relief to the limb has a distinct effect upon the nutrition of the muscles. So true is this that after such section a renewed attempt to develop the muscles by electrical treatment should always be made. The application of braces or other appliances to the legs to aid in locomotion is often imperatively demanded. It is very much better for the child to exercise the limb even partially than that there should be added to the failing nutrition of spinal disease the depressing influence of loss of use.

Ascending Poliomyelitis.—In 1849 Duchenne described a peculiar paralytic affection whose pathology is not yet elucidated, but is probably that of a myelitis affecting especially the gray matter. The symptoms consist of a rapidly developed paralysis beginning in the legs and extending upward, associated with complete muscular flaccidity, loss of reflexes, and rapidly progressive atrophy of the muscles with reaction of degeneration. The affection is distinguished from simple poliomyelitis by its ascending progressive course, and by the absence of the stages of general stationary paralysis and of regression. From Landry's paralysis it differs in the presence of muscular atrophy with changes in electro-contractility. From neuritis it is distinguished by the absence of nerve pain and tenderness. From progressive muscular atrophy it is separated by its rapid course and by the fact that the paralysis precedes the atrophy and is attended early in the case with well-marked reaction of degeneration. When the disease continues to ascend, and implicates the muscles of deglutition and of respiration, it may cause death; but in the majority of cases the patient recovers with more or less damage to the muscles.

The treatment may be that of chronic myelitis, with the addition of local electrical treatment for the maintenance of the nutrition of the muscles. The resemblance of this affection to certain cases (see below) of chronic metallic poisoning suggests very strongly that it is due to a toxæmia, and that strychnine should be used.

Metallic Poliomyelitis.—A paralysis with muscular atrophy may be produced by various metallic poisons, especially arsenic and lead. In the majority of cases it is the result of a peripheral neuritis, but it may occur without pain or nerve-tenderness, and probably, therefore, be due to a lesion of the cells in the anterior horns. The recognition of this variety of poliomyelitis is a matter of grave importance, because it involves the treatment. The character of a case can usually be recognized by attending to the following points: first, the patient is an adult; second, the attack is without fever, and the paralysis is much more wide-spread and more slowly and progressively developed than in ordinary mild acute poliomyelitis; third, the sphincters, the bladder, and the respiratory muscles, which are usually not affected in true poliomyelitis, are almost invariably attacked; fourth, there is usually but not always disturbance of sensation; fifth, suspicion being aroused, evidences of metallic poison can be obtained from the history, from the presence of the blue line on the gums, or by finding metal in the urine. This form of metallic poisoning may resemble Landry's paralysis in the progressiveness of its course, but is distinguished by the trophic changes which occur in the muscles. Its treatment is that of the metallic poisoning, added to the local use of electricity and of massage upon the muscles, and the administration of massive doses of strychnine, which alkaloid we have seen when pushed to its furthest limit act most promptly and effectually.

CHRONIC POLIOMYELITIS. PROGRESSIVE MUSCULAR ATROPHY.

DEFINITION.—A disease characterized by a degeneration of the trophic spinal cells, accompanied by progressive muscular atrophy with loss of power.

ETIOLOGY.—Progressive muscular atrophy is more frequent in males than in females, and usually develops between the ages of twenty-five and fifty. Heredity, and especially indirect neuropathic heredity, seems to have etiological influence in a majority of cases. Overwork, mental distress, exposure, traumatisms, and syphilis are all assigned causes, but their action is very obscure.

MORBID ANATOMY.—The affected muscles are pale in color, with various alterations in their fibres. Four of these changes seem to be well defined: first, narrowing of the fibres, with some indistinctness of the striation; second, fatty degeneration, in which the striæ become granular and the granules increase in size until at last the sarcolemma is replaced by fat-globules; third, hyaline degeneration, in which the muscle-sheaths contain only a clear, homogeneous, striated material with embedded globules; fourth, an apparently longitudinal splitting of the fibres, with loss of the striation, or with an appearance of striation much finer than normal, followed by fatty degeneration. Of all these processes the ultimate end is an empty sheath, shrunken, but clearly distinguished from the interstitial fibrous tissue.

Nerve-degeneration seems to begin in the anterior roots, and probably is confined to the motor fibres, so that a peripheral nerve will in the end contain many empty sheaths. The posterior roots remain normal.

The primary lesion of the disease is a slow wasting of the ganglionic cells of the spinal cord, by which they lose their processes, become globular or irregular in form, and, shrinking into angular masses, finally disappear, leaving foci of the gray matter in which there is no trace of cells. To this lesion is added, in old cases, a well-developed sclerosis of both direct and crossed pyramidal tracts. The nerve-fibrillæ of the cord, which are the prolongations of the ganglionic cell processes, have usually disappeared from the affected tissues, and probably suffer very early in the disease. The larger blood-vessels are often enlarged, the minute blood-vessels not much changed. The degeneration of the gray matter is in typical cases confined to the anterior horns, the posterior remaining normal.

SYMPTOMATOLOGY.—Progressive muscular atrophy is always a very insidious and slow disease; in most cases the first symptom to attract attention is a loss of endurance, or even a loss of momentary power; the muscle is found softer and more flaccid than normal, and in a short time the atrophy is apparent. Sometimes before the atrophy, always sooner or later, fibrillary contractions occur. They consist of irregular twitchings of the muscle-fibres, which produce no effect except a movement of the skin over the contraction. They may be slow and irregular, or may amount to stormy peristaltic movements hurrying through the muscle in rapid succession. Usually their severity is in direct relation to the activity of the disease process. *Wasting palsy* usually attacks groups of muscles which are more or less isolated and separated from one another; in the majority of cases it is somewhat symmetrical, although it is not exactly the same muscles on the opposite sides of the body that are affected. The degree of the paralysis of the muscles is in direct proportion to the loss of muscular substance, which is evidently the cause of the weakness.

The hands are in most cases the first portion of the body to be attacked, and frequently the symptoms are more severe in the right than in the left hand. According to Eulenberg, the interosseous muscles commonly suffer first, whilst Roberts, Wachsmuth, and Friedreich state that the ball of the thumb is usually implicated before the interosseous muscles. The first external interosseous is said to be the first to feel the influence of the disease, whilst the opponens and the adductor pollicis are more apt to suffer than the extensors, the abductors, and the flexors of the thumb. In the few cases in which we have had an opportunity to see the disease in its earliest stage the interosseous muscles were the first affected. The loss of power in the interosseous muscles is especially apparent in abduction of the index finger; whilst the wasting shows itself in the flattening of the thenar eminence and the falling in of the interosseous spaces.

Progressive muscular atrophy sometimes makes its first appearance in the deltoid, in the pectoralis major, in the serratus magnus, or even in the lumbar muscles. The legs are the most infrequently affected, but do not always escape. The disease process may spread to the medulla, and indeed the medulla may be primarily attacked, constituting glosso-labial paralysis.

The "ophthalmoplegia externa" of Hutchinson (see page 595) may also be a symptom of progressive muscular atrophy.

Disturbances of sensation are never severe in a pure muscular atrophy, and in most cases there is either no or only very slight paræsthesia.

The loss of power in the diseased muscles and the secondary contractures which occur in their antagonists produce in some patients extraordinary deformities. In one case under our care, in which the disease commenced, or at least very early was most pronounced, in the neck, the head perpetually fell forward, the chin resting upon the breast. The most characteristic of the deformities is the clawed hand, caused by the permanent flexion of the last two phalanges of the fingers, which are extended at the metacarpal joint. As was shown by Duchenne, this deformity is the result of atrophy of the internal and external interosseous muscles with the preservation of power by the extensors and flexors of the fingers. It must be remembered that this deformity is really pathognomonic of paralysis of the interosseous muscles, and is characteristic of progressive muscular atrophy only for the reason that loss of power of the interosseous muscle is rare from other causes. If, however, from local disease of the nerves the interosseous muscles be paralyzed, the clawed hand will be developed. If only one hand be clawed, local disease should be suspected. Subluxation of the shoulder-joint is common in cases in which the muscles of the part are especially paralyzed.

The electro-muscular contractility is remarkably preserved, evidently because the destruction of the trophic cells in the cord involves individual cells one after the other, and consequently compromises trophically individual bundles of muscle-fibres, one after the other, so that the muscle loses power not *en masse*, but fibre by fibre, that portion of the muscle which retains its functional activity remaining in health and preserving its normal electrical reaction. According to Eulenberg, in the later periods of the disease qualitative alteration in the muscular reaction may be shown by an increased reaction under anodic closure, and less commonly by an increase under cathodic opening. We have never been able to demonstrate this. Remak's diplegic contractions are also rarely to be demonstrated in progressive muscular atrophy.*

There is sometimes a heightened irritability of the muscular fibres in progressive muscular atrophy which causes the muscle to react more actively than normal to the faradic current. This condition we have noted especially when the fibrillary contractions were very severe. In some very slow cases of muscular atrophy with deposition of fatty material in the muscles the response to the faradic current is sluggish.

DIAGNOSIS.—The slow progression of the symptoms, the occurrence of atrophy before paralysis, the preservation of the electrical relations of the muscles, the absence of distinct disturbances of sensation and of pronounced tenderness, make the recognition of progressive muscular atrophy usually very easy. The only disease with which it can be confounded is pseudo-hypertrophic paralysis. (See Pseudo-Hypertrophic Paralysis.)

PROGNOSIS.—The course of the disease usually spreads over many years. Remissions sometimes occur, and it is not very rare for the paralysis to be long located in a few groups of muscles.

---

* "Remak found that the contractions could be produced in the atrophied muscles of the arm when the positive electrode was placed in an 'irritable zone,' which extends from the first to the fifth cervical vertebra, or, still better, in the carotid fossa or the triangle between the lower jaw and the external ear, while the negative was put below the fifth cervical vertebra. The contractions were always on the side opposite to the anode, but when the electrodes were applied in the median line they occurred on both sides. If the current was very weak they were limited to the muscles most severely affected. Remak regarded these as reflex contractions originating from the superior cervical ganglion of the sympathetic, and especially as the patient perceived a sensation behind the ball of the eye when the current wás closed." (Eulenberg.)

TREATMENT.—In our experience the treatment of progressive muscular atrophy has been without good result. The effect of electricity and massage on the muscles has not been perceptible. Gowers, however, asserts that arsenic and strychnine are very useful, and that he has known the progress of the disease to be arrested by the hypodermic injection of strychnine, although the drug had been given by the mouth without avail. The dose which he employs is very small, one injection a day of one-eightieth of a grain, gradually increased to one-fortieth, never beyond. The greatest care should be taken during the whole course of the disease to avoid muscular fatigue, which has a decided deleterious influence. Prolonged rest in bed would seem to be indicated.

SYRINGOMYELIA.

DEFINITION.—A chronic disease characterized by the presence of cavities in the spinal cord, and by peculiar alterations in the sensibility, associated with motor paralysis and various trophic disturbances.

ETIOLOGY.—Concerning the causes of syringomyelia we have no definite knowledge. The disease usually begins between fifteen and thirty-five years of age; it is more frequent in men than in women, and in laborers and artisans than in brain-workers; it does not appear to be distinctly hereditary, although there is reason for believing that it depends upon some embryological affection of the cord which diminishes the power of the nerve-elements to resist the hyperplastic tendency inherent in neurogliar tissues. It may exist as a primary affection, but has been associated with lateral sclerosis, poliomyelitis, chronic periencephalitis, and other organic diseases of the brain or the spinal cord.

MORBID ANATOMY.—The principal lesion in syringomyelia is spinal, with secondary trophic changes in the muscles, the bones, the cellular tissues, the skin, and the peripheral nerves. To macroscopic examination the cord may present the appearance of a large blood-vessel empty and collapsed. It is irregularly increased in size, deformed, soft and fluctuating to the touch, or giving the sensation of a hard, firm, rigid cord, and contains one, two, or rarely three cavities, situated in the horns of the gray matter. The size, length, shape, and cross-dimensions of the cavity vary almost indefinitely. Its liquid or gelatinous contents are enclosed by a smooth yellowish coating.

Hydromyelia—that is, dilatation of the spinal cord with excess of fluid—has long been known to the profession as a pathological condition which may exist without producing symptoms during life; it is probably the analogue of hydrocephalus internus, and is caused by a stopping up of the spinal canal. It is, however, rather an accidental than an essential part of syringomyelia, whose primary histological lesion is looked upon by the majority of investigators as a neoplastic hyperplasia of the neuroglia of the gray matter, but is believed by others to be a hyperplastic myelitis. According to the late researches of Hoffmann, the disease commences in proliferation of the epithelial lining of the spinal canal, and in many cases proceeds from a congenital anomaly,—viz., nests of embryonal tissue. The result of this proliferation is a mass, the so-called *gliosis*, which closes the central canal and after a time undergoes regressive metamorphosis, with destruction of the tissue and formation of a cavity. The gliotic tissue is composed chiefly of mono- or bi-nucleated, spider-like cells with long anastomosing processes, between which are granular elements and pigment-granules. The lining of the cavity is a dense fibrillary felting, which is not sclerotic. During the process of growth and degeneration secondary inflammations, hemorrhages, and wide-spread sclerosis are produced in the surrounding tissue. The peripheral nerves are usually found altered, suffering from parenchymatous neuritis, or perhaps from atrophy. The muscles themselves undergo atrophy, and Dejerine found that the intra-muscular nerves were normal or atrophic according as their muscles were normal or atrophic. The changes in the nerve and in the muscle are, however, but secondary lesions.

SYMPTOMATOLOGY.—Syringomyelia commences insidiously, with weakness and disorder of sensation in the upper extremities, followed after a time by muscular atrophy, with increase in the sensory symptoms; then by spinal curvature in the form of scoliosis; and finally by motor palsy in the lower limbs. Vaso-motor and trophic changes in the skin, in the subcutaneous cellular tissues, and even in the joints and bones, soon follow upon the muscular atrophy. In some cases the shoulder muscles, more rarely those of the lower extremities, are first attacked, and the medulla oblongata may be the part primarily affected.

The most characteristic symptom of syringomyelia is a peculiar dissociation of ordinary sensation. Commonly the sense of pain and the power of recognizing heat and cold are diminished or lost, although sensibility to touch and the muscular and special senses are intact. Rarely it is some form of ordinary sensibility which is first lost, and cases have been recorded in which pain and thermic sensibility have been increased, ordinary sensibility remaining normal. Further, thermic sense perversion may be produced, so that hot bodies feel cold and cold bodies hot. The thermic anæsthesia may be so slight that it can scarcely be made out, or so complete that a patient can be burned without being aware of it.

The thermo-anæsthesia and the analgesia do not necessarily coincide in degree or position, and may from time to time vary in the same case. They usually occupy considerable zones, and affect both mucous membranes and skin. In some cases there are burning or freezing subjective pains. Loss of motor power in the arms is a common primary symptom. Usually motor disturbances in the legs are secondary, and develop late in the disorder. They commonly consist of spasmodic paraplegia with exaggerated patellar reflexes, but may be simply ataxic incoördination with abolished reflexes. Loss of motion is followed in the upper extremities by muscular atrophy, secondary contractures, and a claw-like hand resembling that of poliomyelitis. Paralysis and trophic disturbances are extremely common in the muscles of the neck, so that scoliosis is an almost constant and may be an early symptom; it is said to be almost universally dorso-lumbar, with the convexity to the left. The atrophic muscles may give the reaction of degeneration; more commonly, however, there is a simple diminution of electrical excitability.

As the disease progresses, the paralysis extends up the arm, and finally involves the shoulder and even the face. Indeed, altered trigeminal sensations, nystagmus, inequalities of the pupil, disturbances of the taste, paralysis of the vocal cords, dyspnœa, and rapid heart action, any or all of these may develop as the result of serious implication of the medulla.

The trophic changes in syringomyelia are very marked. The skin may become glossy or be covered with a thick epidermis or with bullous, eczematous, or herpetic eruptions. Perforating ulcers have been described, and in rare cases there has been a trophic gangrene of the skin, followed by loss of substance and by a whitish cicatrix. The distorted, thickened, often furrowed nails sometimes fall out. The subcutaneous cellular tissues may be œdematous or the seat of abscesses and especially of whitlows. The bones and joints sometimes undergo arthropathic changes similar to those seen in locomotor ataxia; and acromegalia, coinciding with, if not dependent upon, syringomyelia, has been reported.

The implication of the vaso-motor and secretory systems is shown by irregular or exaggerated sweating, by blueness and coldness, or by swelling, scarlet color, and excessive heat of the extremities. Polyuria, cystitis, and even perforating ulcers of the bladder may occur.

Various clinical types of syringomyelia have been noted. Blocq describes two: in one the atrophy commences in the ulnar nerve distribution and is followed by spastic paraplegia, in the other the atrophy begins in the radial nerve and is followed by tabetic incoördination. The cases vary, however, so much that no description of types is of value. If the gliosis attacks especially the posterior portion of the cord, the prominent symptoms may resemble those of tabes. *Morvan's disease* appears to grade into syringomyelia through an unbroken series of cases, and must therefore be considered simply a variety of it. In it the tactile sense nearly always disappears with the other forms of sensibility; the trophic changes predominate, and almost exclusively consist of multiple whitlows, deep cracks and fissures in the skin, and arthropathies of the smaller joints. Moreover, in certain cases these affections are symmetrical on both hands and feet, and do not attack the remainder of the body. Finally, the muscular atrophy is slightly marked, and is not, as a rule, progressive.

Diagnosis.—Syringomyelia is distinguished from cervical pachymeningitis by being much less painful and not accompanied by rigidity of the neck, and by the peculiar disturbances of sensation. In sclerodactylitis simulating syringomyelia, sensation is preserved, whilst the inflammation of the skin is a dominant, not a secondary, feature of the case. In alcoholic paralysis thermo-anæsthetic disturbances resembling those of syringomyelia sometimes occur, but the symptoms usually appear in the lower extremities and are developed very rapidly, whilst tenderness of the muscles or of the nerve-trunks can be made out. The hysterical simulation of syringomyelia may be detected by noticing the presence of hysterical symptoms, the wide extent of the anæsthetic disturbance, and the absence of degenerative atrophy of the muscles. It must be remembered, however, that syringomyelia may develop in the hysterical subject.

Prognosis.—The course of syringomyelia is very slow, usually extending from ten to twenty years, and accompanied with marked remissions. The correctness of the assertion sometimes made that recovery may occur seems doubtful. Death is usually due to the spread of the disease to the medulla oblongata, or to exhaustion produced by cystitis, decubitus, septicæmia, etc.

Treatment.—No drugs are of any value except for giving relief of symptoms. The bodily health should be maintained, the analgesic skin carefully protected from injury, and muscular fatigue and cystic complications guarded against. Counter-irritation and suspension, as recommended by French authors, probably do no good. Electrical treatment of the spine is entirely without value, and even the attempt to maintain by electricity the nutrition of the affected muscles is hopeless.

LOCOMOTOR ATAXIA. TABES.

Definition.—A disease accompanied by sclerosis of the posterior root zones of the spinal cord, and characterized by loss of coördination and of the reflexes, with disturbances of sensation, but without paralysis or atrophy of the muscles.

Etiology.—Locomotor ataxia is not hereditary, is much more frequent in males than in females, and is a disease of middle life, although it may occur at any age. In the great majority of cases it develops in persons who have had syphilis, coming on from five to twenty years after the infection. It is not, however, specifically a syphilitic disease, and may occur in non-syphilitics. According to the observations of Tuczek, a disease resembling tabes symptomatically and anatomically may be produced by ergotin, and it is possible that there is a post-syphilitic poison which produces tabes. Sexual excess is not, as was at one time taught, an ordinary cause of locomotor ataxia, if indeed it ever produces it. Overwork, nervous strain, immoderate use of alcohol and tobacco, exposure to wet and cold, peripheral traumatism, lead and other poisons, have all been assigned as causes for the disorder, but their influence is very obscure.

MORBID ANATOMY.—The lesion of tabes may develop in any portion of the spinal cord, but usually is primarily lumbar; in the advanced degeneration the microscope shows a mass of connective tissue with atrophied axis-cylinders from which the myelin sheaths have wasted away. The connective tissue is fibrillated and nucleated, and excessively trabeculated; sclerotic changes are usually pronounced in the vessels. An early stage of the sclerosis is that in which there is increase of the nuclei and of the amount of the connective tissue, with wasting of the myelin sheaths. Westphal and some other pathologists state that preceding this stage can be demonstrated one of simple granular change.

There is still uncertainty as to whether the process begins in the posterior spinal roots or in the posterior root-zones (columns of Burdach) of the cord. Leyden, Marie, and other authorities believe that the degeneration ascends from the nerve-roots into the spinal cord, making the primary lesion of locomotor ataxia a degenerative neuritis, which is believed by some to commence in the nerve peripheries and extend upward,—a highly improbable view. It is certain that the columns of Burdach are very early attacked, and that the lesion following the ascending nerve-bundles passes up the cord, not only along the root-zones, but especially into the columns of Goll, which are usually diseased higher up than are the root-zones. There are cases in which the columns of Goll seem to be more thoroughly diseased than are the root-zones.

There are two beliefs among pathologists as to which tissue is primarily affected: some maintain that the disease begins in the neuroglia, and that the changes in the nerve-elements are secondary to the connective hyperplasia, whilst others affirm that the essential lesion is a parenchymatous degeneration of the nerve-elements, with hyperplasia of the connective tissue as a secondary result. Spitzka has put forward the very improbable view that there are two varieties of locomotor ataxia, one interstitial, one parenchymatous.

The disease process finally affects the gray substance, especially attacking Clarke's columns, and causing atrophy of the nerve-fibres and in some cases wasting of the cells. As was first demonstrated by Westphal, the peripheral nerves habitually suffer in tabes; the alteration is a parenchymatous neuritis, and appears to be confined to the sensory filaments. It is most marked in the extreme periphery of the nerve, so that the cutaneous filaments may be distinctly altered and the large nerve-trunks free. The optic, pneumogastric, trigeminus, and other cerebral nerves are sometimes affected. The peripheral situation of the neuritis, and the fact that in some cases the cord lesion apparently precedes, in others follows, the change in the nerves, indicate that the lesions of the nerves and of the spinal cord are not dependent one upon the other, but are due to some common cause.

SYMPTOMATOLOGY.—The symptoms of locomotor ataxia are usually very slow and insidious, and without fixed regularity or order in their development. They commonly appear first in the legs, but not rarely double vision, giddiness, or other head symptoms take precedence, and the upper extremities may be for years the exclusive site of the disease. Leaving the course and early stages of the disease for discussion in the paragraphs under diagnosis and prognosis, we shall study the general symptoms under the heads of Disturbances of Sensation, Disturbances of Motion, Disorder of the Organs of the Special Senses, Trophic Changes.

Disturbances of Sensation.—Pain is a prominent symptom in more than nine-tenths of the cases of tabes, and is variously described as though a red-hot wire or a sharp dagger were thrust through the limb. In some cases these so-called *fulgurant pains* occur continually; in other instances they come on in paroxysms; but almost invariably they temporarily disappear at intervals. They may follow the distribution of the nerves, but more commonly are most severe in the neighborhood of the joints, especially in the inside or the outside of the knee or of the ankle. Usually they are not associated with redness or any soreness, and often the patient seizes the affected part forcibly and obtains by the pressure some relief. A certain amount of redness and tenderness may, however, be present during the pain, and in very rare cases trophic eruptions occur. Sometimes the pain seems wide-spread and superficial, and is then usually spoken of as burning or more rarely as a sensation of intense cold.

Pain crises, which are almost pathognomonic of locomotor ataxia, consist of paroxysms of excessive pain with pronounced functional disorder of the viscus in which they are located, but without fever. The attacks usually begin and end abruptly, and may last from a few minutes to several days. The functional activity of the organ may be at once resumed when the pain is over. In the height of the agony syncope or a wild outburst of maniacal melancholy with attempts at suicide may occur. The most important of the pain crises are the muscular, the gastric, the rectal, the urinary, the genital, the cardiac, the laryngeal, and the lymphatic.

The *gastric crises*, the most frequent of any, are characterized by violent shooting and burning pains, having their focus in the epigastric region and radiating in all directions, laterally, upward, downward, until at times they seem to fill with agony the whole abdomen and chest. They are generally increased by epigastric pressure and by the ingestion of food, and are always accompanied by nausea and excessive vomiting. After the stomach has been once emptied the discharge is glairy or ropy, neutral or acid, and often streaked with blood; rarely there is abundant coffee-ground vomiting or even pronounced hæmatemesis. In some cases the focus of the pain is in the neighborhood of the umbilicus, when the crises might properly be spoken of as *intestinal.* Occasionally large quantities of gas form in the gastro-intestinal tract and produce a very obstinate meteorism, or there may be copious bilious, mucous, or serous stools. In such cases loss of the voice, suppression of urine, extreme coldness and cyanosis of the body, cramps, and collapse may closely simulate cholera, and death may result. In the *rectal crises* the pains radiate from the rectum, or this receptacle feels as though it were filled up by an enormous body heated to redness, burning and scorching every part near it. *Muscular crises* are extremely rare, and consist of an excessive muscular weariness and soreness which paralyze for a time the affected muscles. *Genital crises* may consist of violent paroxysms of pain centring in the testicle and shooting along the penis, or, in the female, darting from the ovaries, boring and burning through the whole genitalia. In some cases spontaneous venereal orgasms occur; at first each orgasm ends in a fury of voluptuous delight, but as time passes this delight takes on more and more the character of pain, until rapturous ecstasy is lost in agony. The *genito-urinary crises* may closely simulate renal colic; usually, however, they consist of intense burning or lancinating pains in the urethra, associated with unconquerable cystic tenesmus. When there is an anæsthesia of the bladder, with consequent urinary retention and ammoniacal fermentation, and deposits of phosphates, the symptoms may be strongly suggestive of calculi.

A *lymphatic crisis*, as first described by H. C. Wood, consists of a violent paroxysm of pain (accompanied with great swelling, redness, and tenderness) in the lymphatic glands, appearing with absolute suddenness and disappearing almost as abruptly, but leaving behind it an increase in the volume of the gland, which, if not perceptible at first, finally through successive crises becomes a distinct enlargement. In *cardiac crises* there are violent lancinating and constricting pains in the region of the heart, associated with great dyspnœa, intense distress, and irregularity of the pulse, with or without intermission of the heart-beats. *Laryngeal crises* consist of violent paroxysms resembling laryngismus stridulus, but associated with atrocious fulgurant pains in the larynx, back, and shoulders, and sometimes accompanied by the expectoration of little bloody pellets of mucus. Death from laryngeal asphyxia has resulted in these cases; and when laryngeal anæsthesia is present, food frequently enters the larynx and lungs.

Formications and other paræsthesiæ are common in tabes. The girdle sensation, a feeling as though a tight band were drawn around the head, the neck, the body, or the limbs, is usually very distinct. Numbness may develop early or late, and often when in the foot produces a sensation of walking or standing upon velvet or upon cushions of down. The mucous membranes of organs in which crises occur are prone to be completely anæsthetic; the numbness may exist without analgesia, or without paralysis of the tactile or of the temperature sense. All forms of sensibility are, however, in most cases sooner or later diminished. Delayed sensation is not uncommon, and five, ten, or even fifteen seconds may elapse between the time of the contact and its perception. It is not very rare for a distinct interval to exist between the perceiving of the contact of a sharp point and of the pain which it causes. Mendelssohn affirms that the normal reaction of the sensory nerve to electricity may be reversed, so that on closing the circuit the earliest sensation is at the positive instead of at the negative pole, as in health. The localizing power is sometimes curiously perverted: a single prick may be felt in many places (*polyæsthesia*), or a prick on one leg may be located on the other (*allocheiria*).

Motion, including Reflexes.—The cutaneous reflexes are usually at first unaffected or very rarely increased; when anæsthesia exists they are usually diminished, but they may be lessened or even abolished without marked disturbances of sensation. The deeper reflexes are profoundly affected; indeed, loss of the knee-jerk (*Westphal's symptom*) is one of the earliest and most constant phenomena, but may be absent when the lesion is confined to the upper cord. Loss of coördination is a most characteristic symptom; closely connected with it is the so-called *Romberg's* or *Brach's symptom*, which may precede all other evidences of the disease, and always belongs to the earliest stage. It consists of a peculiar vertigo, usually associated with a momentary sense of terror, produced by quickly stepping from a light into a dark place, or by quickly shutting the eyes.

In most cases the loss of coördination is shown by excessive swaying when the patient is made to stand with the feet close together or on one foot. If, however, this test fail, sudden turning or the attempt to walk a chalk line will usually succeed, and if the eyes be closed the lack of control becomes at once manifest. Sooner or later the so-called *ataxic gait* develops. The patient walks with his head a little bent forward and the eyes directed to the ground. The trunk inclines upon the thighs, whilst the feet are held in advance of the buttocks, with the legs widely separated from each other. At the same time, owing to the excessive contractions of all the muscles of the lower extremities, the leg proper is extended somewhat rigidly upon the thigh, and there is very little movement at the knee-joint. The advancing leg is therefore raised from the ground in some degree by an elevation of the pelvis, although at the same time some flexion does occur at the knee-joint. By these conjoint movements the foot is freed from the ground, and, having been flung forward and outward by a rapid muscular jerk, comes down with a thump like a solid mass. In some cases the heel is the last to leave the ground and the first to touch it. Not rarely the pelvis is so much inclined during walking as to carry the centre of gravity too far towards the side of the stationary leg. To counteract this and maintain the balance of the body the upper portion of the trunk is curved towards the advancing leg by a contraction of the erector spinæ muscles, or the arm corresponding to the advancing leg is thrust out laterally. The alternation of these movements at each step may give a pendulum-like swing to the body.

In the more advanced stages of locomotor ataxia the body is so far thrown forward that the patient can walk only by the help of two canes or crutches; all the movements executed with the legs are performed with great stiffness and by sudden jerks. Still later in the disorder the wild, irregular, choreiform movements of the legs make walking impossible. Finally, if the lesion travels up the spinal cord, all power of coördinating the muscles of the trunk may be lost, so that the patient is no longer able to sit unsupported.

Organs of Special Sense.—In the prodromic stage of locomotor ataxia transient ocular palsy with its resultant ocular diplopia not rarely occurs. In the later stages ptosis and internal or more rarely external squint are common symptoms; and even a general ophthalmoplegia may occur. The pupil is usually pronouncedly myotic, but may be mydriatic, and often irregular. *Argyll-Robertson pupil* or *reflex iridoplegia* is almost characteristic of the disease, and may be a late or an early symptom. In it the pupillary reflexes are abolished, but the pupil reacts with accommodation, so that although pinching of the skin of the neck or throwing light suddenly into or shutting light suddenly from the eye produces no pupillary movement, the pupil dilates when the gaze is suddenly directed from a near to a distant object. Contraction of the field of vision with disorder of the color sense is very frequent; the contraction is concentric, but usually somewhat irregular. Perception fails early for green and red, is long maintained for yellow and blue. These visual phenomena are caused by degeneration of the optic nerve. Deafness is infrequent; it may occur either as an early transient or as a late permanent symptom. According to Gowers, it is accompanied by a progressive limitation of the range of hearing analogous to the contraction of the visual field, the notes of the scale, beginning at the top, dropping out of the range of hearing one after the other, until all are alike inaudible.

Trophic Changes.—The most important trophic changes in locomotor ataxia are the perforating ulcer, alterations of the bones and joints, and perhaps cardiac disease. The perforating ulcer may attack the hands or the internal organs, but usually affects the foot. As it occurs in other diseases than locomotor ataxia, it will be found described under trophic diseases. (See page 621.)

As was first stated by Vulpian in 1879, valvular disease frequently occurs in locomotor ataxia, especially showing itself in insufficiency of the aortic valve. It is uncertain how far the various valvular lesions are directly secondary and trophic, but when a sharply defined perforating ulcer forms upon a valve in a case which suffers from a severe cardiac crisis, the existence of a trophic influence can scarcely be denied.

Tabetic arthropathy may first manifest itself by a peculiar articular crepitus; when fully developed the arthropathy consists of a serous effusion, free from blood, pus, or albuminous flocculi, in the articular cavity and extending it may be into the tissues outside of the joint, which is enormously swollen, hard, pale, and so resistant as not to yield to pressure. In the second stage of the affection the joint remains swollen, but is hard and bony, with an evident increase in the size of its bony surfaces. Destruction of the articulating surfaces marks the third stage; the epiphyses especially undergo atrophy, and even the head of the bone may be to some extent absorbed, the ligaments are elongated, and at last a condition of subluxation or perhaps of complete luxation results. The ataxic arthropathy is usually more or less symmetrical; it attacks especially the knees, but is frequent in the foot and in the hand. The atrophic shafts of the bones may undergo spontaneous fractures, and dropping out of the teeth, one by one or in mass, may result from wasting of the alveolar processes. The primary change in the bone is probably always an hypertrophy, the atrophy being secondary.

Diagnosis.—Two stages of locomotor ataxia are recognized by some systematic writers, the first or pre-ataxic being that which precedes the development of pronounced ataxia, the second that of ataxia. This division of the disease is altogether arbitrary, although it has a measure of usefulness in calling attention to the fact that the ataxia is commonly a late symptom; it may, however, develop early in the disease, or at least without the long presence of the ordinary symptoms of the first stage. In the majority of cases the symptom to which the patient's attention is earliest directed is pain, which is not rarely long treated under the supposition of its being rheumatism. Careful examination will at this time generally show that the knee-jerks are either wanting or greatly diminished, and usually some disorder of coördination can be discovered. The Argyll-Robertson pupil belongs in its first development to the early stage of locomotor ataxia, as do the transitory attacks of ocular motor paralysis, diplopia, etc. Permanent irregularities of the pupil, and contraction of the field of vision, usually come on towards the end of the first stage or later; they may, however, develop very early. Persistent vertigo occurs most frequently in the advanced stages of the disease.

The diagnosis of the advanced stage of locomotor ataxia is so easy as to require no further discussion, but the recognition of the disease in its incipiency may be very difficult. The bilateral character of the pains and the absence of soreness should always give rise to the suspicion that they are not of rheumatic origin. Nevertheless, gouty pains may have all the character of locomotor ataxia pains. If the pains be associated with Argyll-Robertson pupil, or with loss of the knee-jerk, or with any disturbance of coördination, there is sufficient ground for the diagnosis of a probable locomotor ataxia. The occurrence of three of these symptoms makes the diagnosis positive.

A pain crisis is of more diagnostic importance than lancinating pains. In rare cases, especially in cardiac crisis, there may be difficulty in distinguishing between the tabetic crisis and a functional pain attack such as that of angina pectoris. The seat and character of the pain, however, very rarely, if ever, exactly accord with the manifestations of the disease which is simulated. Thus, in a cardiac crisis the pain will centre in one shoulder or in an axilla, or will shoot into the abdomen, or into the right arm, etc.

A very important distinctive mark of the crisis is the sudden resumption of normal activity in the organ so soon as the pain ceases: moreover, there is no sign of disease in the organ between the paroxysms. Thus, after a gastric crisis the patient at once will begin to take and digest food, whilst a man who has had a cardiac crisis will be able to go up-hill, to run up-stairs, etc., without difficulty. When, however, as especially occurs in cardiac crises, there are trophic or other organic changes in the organ, functional power may be permanently altered; as in these cases the sclerosis may be so situated that the legs offer no ataxic symptoms, bulbar symptoms should be carefully looked for; giddiness, Argyll-Robertson pupil, diplopia, or strabismus would be almost decisive. The import of the ordinary visual symptoms of ataxia is so serious that whenever they occur in a middle-aged person and are neither hysterical nor gummous they should give rise to the suspicion of tabes, especially if there be any history of old syphilis.

PROGNOSIS.—The course of locomotor ataxia is in most cases extremely slow. The pre-ataxic stage may last ten or even twenty years, and the whole course of the disease stretch over a quarter of a century. Remissions of months or years without change for the worse are frequent; but we do not believe that a case of posterior sclerosis sufficiently developed to be positively recognized is ever cured. In women the disease does not prevent a successful pregnancy, which may for the time being hold the symptoms in abeyance, but has no permanent effect upon them. Death usually takes place from some intercurrent disease, but may be the result of chronic periencephalitis, or even of a sudden acute periencephalitis. Subacute myelitis is also said sometimes to occur as a complication.

TREATMENT.—In the management of locomotor ataxia rest, both bodily and mental, is vital. The life of the patient should be permanently arranged in such a way as to avoid all unnecessary expenditure of vital force. Physical labor is of course impossible, and mental work should be so reduced that it will merely be sufficient to divert the attention of the patient from himself. Absolute rest in bed for a series of weeks, combined with the use of massage to prevent the bad effects upon the general health which such confinement tends to produce, is often of the greatest service, and may produce a remission of the symptoms which will last for months or even years. Under no circumstances should the patient be allowed to take long walks; the effects of a single over-fatigue may last for many months.

Sexual intercourse should be as far as possible avoided. It is affirmed by good authority to be especially harmful in those cases in which there is a tendency to atrophy of the optic nerve, with increasing impairment of vision, rapid blindness having, under these circumstances, followed a newly-contracted marriage. Whilst open-air life is useful, the most scrupulous care should be exercised to avoid exposure to wet or cold, and, when possible, the winters should be passed in a warm, dry climate.

The diet in locomotor ataxia should always be nutritious, but non-stimulating; the very moderate use of wine or tobacco is not harmful, but the slightest excess is deleterious.

The effect of drugs upon posterior sclerosis is very slight; anti-syphilitic treatment is of no value, the contrary statements abundant in literature being based upon wrong diagnoses. We have not seen good produced by silver nitrate. Gold chloride and barium chloride are probably harmless remedies when not given in too large dose. Phosphorus has powers for evil rather than for good. Ergot has been largely used for the purpose of acting upon the blood-vessels, but relaxed blood-vessels have nothing to do with development of locomotor ataxia. The effect of ergot in producing a tabetic affection justifies its use by those who believe in the ancient Hippocratic fantasy of *similia similibus curantur;* we have used it repeatedly in large and in small doses without the slightest good effect. The continuous use of corrosive sublimate (one-fiftieth of a grain three times a day) sometimes does good.

The chief value of drugs in the disease is for the purpose of giving relief of symptoms as they arise, and for the moral support that comes to many individuals from the feeling that something is being done for them. Antipyrin, antifebrin, and phenacetin are very valuable analgesics, which have a distinct controlling influence over the nerve-storms of posterior sclerosis. They are much safer remedies than opium. Antipyrin may be given hypodermically, and in a severe crisis so administered in conjunction with morphine. In the use of morphine the practitioner must always remember the especial danger, in a chronic disease like locomotor ataxia, of the formation of the opium habit.

Counter-irritation is of no use, unless it be in the earliest formative stage of the disease. At such time the persistent use of the actual cautery along the spine may be justifiable. Later in the treatment it is to be absolutely condemned. In a crisis, sinapisms and other mild counter-irritation over the seat of the pain or over the root of the nerve supplying the affected part sometimes bring a measure of relief. It is essential to avoid using blisters or other counter-irritation which shall produce destructive inflammation of the skin, as such inflammation is apt to become uncontrollable. Any blisters or sores upon the feet in locomotor ataxia should receive the most careful attention, as a perforating ulcer is said to have followed so small an operation as the cutting of a corn. Very often in a crisis the application of moist heat in the form of the warm bath, hot fomentations, or even the hot-water bag, will give more relief than counter-irritation.

Various natural springs have been much used in locomotor ataxia. In France, Aix-la-Chapelle is highly recommended; in Germany, the carbonic acid thermal salt springs at Rehme and at Nauheim are most popular; in the United States the Arkansas Hot Springs are greatly resorted to. The baths at Töplitz, Wildbad, and Ragatz, formerly in high favor, have, according to Strümpell, at present lost their reputation. The whole question of which spring to select is probably decided according to the fashion of the hour rather than because one spring is really more valuable than the other. Whatever good is achieved is the result of the stimulating influence of hope, of travel, of freedom from care, of rest, aided by the hydrotherapeutic measures employed. Enormous amounts of mercury are habitually used at the Arkansas Hot Springs, and in cases in which gummous disease of the cord has been diagnosed as locomotor ataxia brilliant and unexpected results are sometimes obtained. The vapor bath and very hot baths used for the purpose of producing excessive sweating often do harm in locomotor ataxia; the continuous use of very cold baths or of cold packs also frequently acts unfavorably. The use of tepid baths with gentle friction, and of wet tepid compresses upon the abdomen or the legs, especially at night, is grateful and gives relief. The temperature of the water should be between 80° and 90° F. The bath may be employed from once to three times a day, according to the strength of the patient.

It is frequently asserted that electricity is a valuable remedy in the treatment of locomotor ataxia, but there seems no sufficient ground for believing that the assertion is correct. The galvanic current is usually selected and applied along the spinal cord; some authorities are urgent that the current should be passed down the cord, others are equally positive that the direction should be upward. There is no good reason for believing that the electrical current in these cases succeeds in getting through the thick, soft and bony tissues overlying the spinal cord. There is also no good reason for supposing that it is possible to stimulate by the ordinary medical galvanic current the deeply situated sympathetic ganglia in the neck: further, it is hardly conceivable that stimulation of these ganglia could do any good in locomotor ataxia. In some cases of locomotor ataxia relief of pain has been obtained by the passage of galvanic currents along the nerve-trunks; but it is probable that in these cases the central lesion was reinforced by a peripheral neuritis, and that it was the peripheral neuritis and not the centric lesion that was benefited. The local application of a dry brush with a moderately strong current sometimes relieves the numbness of tabes.

In all cases of tabes it is essential that the bladder be thoroughly emptied at regular intervals. Even in the earlier stages micturition may be so imperfect that there is a residual urine, which undergoes fermentation and sets up a cystitis, which, although slight, may yet be sufficient gradually to involve the ureters and the mucous membranes of the pelves of the kidneys, and finally the kidneys themselves. In this is probably found the cause of frequent death from kidney disease in locomotor ataxia. No hesitation should be felt, in any case, in using the soft catheter, and when the urine is ammoniacal the bladder should be washed out thoroughly every other day with a dilute solution of some acid antiseptic.

Langenbach some years ago proposed for the cure of tabes the stretching of the sciatic or other large nerve taking its origin in the affected region. The procedure is apparently devoid of any scientific basis, has been followed by death, and is not justifiable. The exploitation by Charcot of the method of Motschoutkowski—*i.e.*, that of suspension—led to its universal adoption in the treatment of tabes. The claims made by Charcot have, however, not been confirmed, although in a number of cases there has been an apparent advantage. It is stated that lost sexual function is often restored. Motschoutkowski believes that there is an absolute stretching of the vertebral interspaces and a direct influence upon the cord, but at present there does not seem to be any physiological explanation of any good result which may be obtained by the procedure. Charcot teaches that œdema, obesity, phthisis, valvular or other cardiac lesions, emphysema, and marked atheroma of the arteries are contraindications to the use of suspension, and certainly when either of these exists the practitioner is not justified in experimenting with the method. The best apparatus is probably that which is known in America as the Weir Mitchell apparatus. The suspension may be from five to fifteen minutes once a day, the spring balance always being used, that the amount of force applied to the head may be known. The physician himself should always be present at the first suspension, and at subsequent séances at least a thoroughly trained reliable nurse should superintend.

ANTERO-LATERAL SCLEROSIS. SPASTIC PARAPLEGIA.

Definition.—A disease due to sclerosis of the antero-lateral columns of the spinal cord, characterized by spastic contractions, with partial loss of power and exaggerated reflexes, without sensory disturbances or trophic changes.

Etiology.—The causes of antero-lateral sclerosis are those which produce posterior sclerosis. Our knowledge does not suffice to explain why in one case the sclerotic disease attacks one region, and in another case another region, of the cord.

Morbid Anatomy.—The sclerosis may affect the whole of the lateral columns, but is usually developed in the so-called crossed pyramidal tract. Its history and microscopic anatomy are those of other scleroses in various positions of the cord. (See Locomotor Ataxia.)

SYMPTOMATOLOGY.—Antero-lateral sclerosis may develop at any age, but is a disease especially frequent in adult middle life, and, as the lesion is usually located in the lower segments of the spinal cord, commonly first shows itself by clonic or tonic spasm in the legs after prolonged exertion. A little later the loss of endurance during walking is manifested, and by and by the characteristic gait develops. The rigidity of the various muscles prevents the free bending of the knee- and hip-joints, whilst the heel is drawn up by the great power of the contractures of the calf-muscles. Unable to lift the toes from the ground, the subject raises and rotates the pelvis, so that the body is inclined during the step towards the leg upon which its weight is rested, whilst the toes are pushed forward with the greatest difficulty along the ground through a step of from three to six inches. In bad cases violent tremors are apt to occur in the legs during effort, and at such times rhythmical movements may throw the heels of the patient up and down in regular vibrations. As the disease progresses, the heels are drawn up permanently, so that the subject must stand upon the toes, thereby throwing the trunk forward and necessitating the use of crutches or canes held well in advance of the body. A little later than this, walking becomes impossible ; the leg is now usually flexed upon the thigh, the heel drawn up and the toes turned inward,—this position being due to the superior power of the posterior muscles of the thigh and leg and of the abductors as compared with their antagonists. In some cases the legs are stiffly extended, very rigid, with the feet inverted and often crossed. Not rarely violent clonic spasms occur, from time to time causing rapid to-and-fro vibrations of the legs ; such spasms are especially frequent at night, are usually but not always painless, and constitute the condition which has been inappropriately named by Brown-Séquard "*spinal epilepsy.*"

There are no pronounced sensory or trophic symptoms, or at most there is slight numbness or paræsthesia. For reasons which are not apparent, lateral sclerosis is rarely followed by peripheral nerve changes, so that ocular and laryngeal and other peripheral symptoms are uncommon. The mind is rarely affected, unless the sclerosis exists as a secondary complication of a general paralysis. From the beginning the deep and superficial reflexes are exaggerated, the slightest touch producing a quick and violent response. Ankle-clonus and knee-clonus are common phenomena. Sometimes the touch of the bed or of the floor suffices to throw the muscles of the legs into violent clonic contractions. In advanced cases the contractures may be so great that the reflexes are entirely abolished.

DIAGNOSIS.—The diagnosis of lateral sclerosis rests upon the gradual development of loss of power which is accompanied by contractures and heightened reflexes and so situated as to be evidently of spinal origin, combined with the absence of girdle sensation, of pain, and of disturbance of sensation, of paralysis of bladder or rectum, of trophic changes, and of disorder of coördination. The diseases which produce groups of symptoms more or less closely simulating lateral sclerosis are spinal meningitis, chronic cerebral disease with secondary degeneration, and hysteria.

Spinal meningitis is accompanied by excessive pain, and any attempt at the extension of the affected limbs produces suffering which is much greater than that caused by similar procedures in lateral sclerosis.

The lesions of lateral sclerosis are practically the same as those of descending degeneration from cerebral diseases, and the symptoms are very similar, except in their distribution. Cerebral lesions are usually unilateral, spinal lesions bilateral. Almost invariably, therefore, a hemiplegic or a monoplegic character will betray the secondary degeneration. In rare cases a spinal sclerosis may in its early stages affect one side of the cord more than the other, but it is probably never purely hemiplegic. The two affections have also different histories: spinal spastic paralysis always develops slowly and insidiously, cerebral spastic paralysis dates back to birth or to an acute attack with cerebral symptoms.

The real difficulty of diagnosis is between hysterical and spinal spastic paralysis, a difficulty which is enhanced by the fact that hysterical contractures may in time be converted into antero-lateral sclerosis. In a case reported by Charcot contractures of all four extremities developed suddenly in a woman and continued for ten years, with temporary intermissions; after the last seizure the contractures continued until death, and at the autopsy symmetrical lateral sclerosis was found to extend almost the entire length of the cord. The rule laid down by Charcot is that whenever marked atrophy of the muscles and persistence of the contractures during anæsthesia are present organic degeneration of the spinal cord has set in. We have certainly seen the distinct hysterical contracture gradually take on the absolute clinical picture of an old lateral sclerosis. Ankle-clonus is not peculiar to either spinal or hysterical spastic paralysis; it occurs in each. It is probable that the peculiar lead-pipe rigidity seen at a certain early stage of spastic paralysis never occurs in hysteria. The great difficulty is the separation of the severe types of the two diseases. The distinguishing points in the hysterical disorder are the suddenness of its development, the history of marked hysterical symptoms in the past, the presence of anæsthesia or of other hysterical symptoms, and the occasional sudden intermission of contractures. Contrary to the statements of Charcot, our own experience shows that the hysterical contracture does not always relax during etherization.

PROGNOSIS.—The history of lateral sclerosis is that of other forms of sclerosis, except that secondary complications are much less apt to occur than in locomotor ataxia, and consequently life is more prolonged and is free from suffering.

TREATMENT.—Absolute rest in bed is sometimes of great temporary benefit in lateral sclerosis. Drugs have no effect upon the sclerotic tissue; nerve-stretching and suspension have probably about the same value as in posterior sclerosis.

COMBINED SCLEROSES.

Spinal scleroses of vertical tracts may coexist in various combinations, producing cases which clinically present various mixtures of symptoms according as the disease is more pronounced in one or other of the spinal tracts. These mixed cases have in general an etiology, pathology, prognosis, and treatment similar to those of the affections in which the lesion is not so wide-spread.

Although the mixed cases vary so much in rare instances as to baffle a simple description, two types of cases are so discernible that they have been described as distinct diseases.

The first of these types is the so-called *Ataxic Paraplegia*, in which there is a mixture of posterior and lateral sclerosis. Like other forms of sclerosis, this disease develops very insidiously. According as the lesion affects chiefly one column or the other the symptoms of the locomotor ataxia predominate or the lights and shadows of the clinical picture are chiefly those of the lateral sclerosis. Usually the tendency of the reflexes to be lost, so strongly pronounced in locomotor ataxia, is overcome by the irritation of the lateral columns, with the result of a loss of power of endurance, with loss of coördination and preservation or even excitation of the reflexes. The sensory symptoms are commonly not so severe as in tabes; fulgurant pains are uncommon; the girdle sensation is not present in a majority of cases. Ocular disturbance may or may not exist. The gait is a curious mixture of that of spastic paralysis with that of posterior sclerosis.

In some cases of ataxic paraplegia the disease begins in one tract alone, when may be seen the curious picture of a locomotor ataxia having the reflexes return and in other ways gradually conforming to a lateral sclerosis type; or the reverse of this may happen.

In *Amyotrophic Lateral Sclerosis*, so called, there is poliomyelitis accompanied by a pronounced sclerosis of the pyramidal tracts. It is very rare indeed in an old case of poliomyelitis not to find some sclerotic change in the motor tract, and there is also a regular series of cases grading between those in which poliomyelitis is supreme and those in which the lateral sclerosis is dominant. There does not seem to be at present sufficient reason for believing that either of these lesions is necessarily secondary to the other; they may each have their beginning in the original myelitic attack.

The symptoms of amyotrophic lateral sclerosis are wasting of the muscles, with loss of power, spastic contractions, and heightened reflexes. The upper extremities are usually first attacked; sometimes a hemiplegic arrangement of the symptoms is seen, and very frequently the trophic changes predominate in the arms, the spastic symptoms in the legs. The cranial nerves are usually affected very early, and there is an amyotrophic lateral sclerosis of the medulla which causes glosso-labial paralysis with spastic symptoms; when the sclerosis is not very pronounced the only evidence of its existence distinguishing the case from one of simple bulbar paralysis may be an increase of the jaw reflexes.

There can be no difficulty in recognizing the nature of a typical case of amyotrophic lateral sclerosis; but if the motor cells degenerate very rapidly the loss of muscle-tone may be sufficient to mask more or less completely the sclerosis of the white matter. Under these circumstances a slight stiffness of gait (the "frozen attitude") may alone reveal the true nature of the case.

The prognosis in amyotrophic lateral sclerosis is very unfavorable, death almost invariably resulting in from one to five years. No medicinal treatment is of any avail, but long-continued rest in bed, with massage, and careful nursing, may be serviceable.

FRIEDREICH'S ATAXIA.

DEFINITION.—A family disease, characterized by ataxic symptoms, nystagmus, contractures, and wide-spread paresis with subordinate disorders of sensation.

ETIOLOGY.—Friedreich's ataxia, although it almost invariably occurs in a number of individuals in the same family, is rarely directly inherited from parents, but is an outgrowth from an original neuropathic stock whose tendency to degeneration is further increased by intemperance, tuberculosis, or syphilis in the parent, and by consanguineous marriages. The attack frequently develops without immediate cause, but in some instances has been precipitated by an attack of typhoid fever, scarlatina, diphtheria, or other acute disorder.

MORBID ANATOMY.—In almost all the autopsies in cases of hereditary ataxia there has been found a sclerosis of the lateral or crossed cerebral tract and of the posterior columns of the spinal cord, and usually also one of the direct cerebral tract and of the cerebellar tract. The column of Clarke is also usually more or less degenerated. Nonne has reported one case in which there was no spinal sclerosis, but in which the cerebrum, cerebellum, pons, medulla, and cord were remarkably small, a condition which has also been noted in various cases in which sclerotic changes were pronounced. It is not probable that any of the scleroses is peculiarly primary. They are all probably the result of a common influence; it is possible that they are merely accidental complications of or developments from an original lesion. The nature of the fundamental lesion of the disease remains in doubt, the most probable theory being that of Kahler and Pick, in accordance with which the foundation of the disease is the imperfect development of the nerve-fibres. There is also an appearance of truth in the further generalization of Pick, that this failure of development is due to early vascular degeneration. Recent observers, however, deny that there is any alteration of the vessels, stating that in this fact Friedreich's ataxia absolutely differs from true locomotor ataxia.

Sclerosis of the posterior nerve-roots has been frequently noted, and Auscher states that the peripheral nerves are not found in a condition of degeneration, but have many of their nerve-filaments preserving their embryonal condition,—*i.e.*, being simply nerve-tubes without myelin.

SYMPTOMATOLOGY.—Sixty per cent. of the cases of Friedreich's ataxia have developed insidiously before the tenth year of age, but in individual cases various evidences of prolonged irregular nervous disturbances have been present. Usually the first symptom is a peculiar awkwardness, in most cases beginning in the legs, but sometimes attacking the speech, the lower extremities, and the upper extremities simultaneously, and very rarely taking a monoplegic and even a hemiplegic form.

The most characteristic symptom is the incoördination, which may produce a gait exactly resembling that of tabes, or may reveal itself in a peculiar step in which there is a strong tendency to the lateral projection of the foot, or in a rolling walk like that of alcoholic intoxication. In the arms the loss of coördination is evinced by irregular jerking movements and by the inability to perform delicate acts, and often becomes so extreme that in the impossibility of properly apposing the fingers the action of the hand resembles that of a paw. The *ataxia of quiet action* which Friedreich affirms to be characteristic of the disease and never to be present in locomotor ataxia is usually a rather late symptom, and is shown in the inability of the subject to hold the extremity in any quiet somewhat forced position. This *static ataxia* may be so severe as to produce peculiar athetoid symptoms in the fingers when lying in the lap, or a wavy or non-rhythmic oscillation in the arms and legs when at rest, or tremors, oscillations, or choreiform movements in the head. In rare cases these movements occur only under excitement, and simulate an intention tremor.

In all the cases recorded by Friedreich the knee-jerk was early abolished. It may, however, be normal or even exaggerated; an ankle-clonus has been noted. The attempt to make varieties of the disease based upon the condition of the knee-jerk is futile, since the condition of the knee-jerk must depend upon the position or development of the spinal sclerosis, which is probably only a secondary lesion.

Incontinence of urine is a very rare symptom, and sexual power is usually long preserved. Muscular weakness is sometimes spoken of as an initial symptom, and in the advanced disease may amount to an almost complete wide-spread paralysis. In a few cases the muscles undergo atrophy, which is rarely attended with the reaction of degeneration. Contractures are very frequent in the later stages of the disease, and not rarely give rise to deformities, such as curvature of the spine (which has been noted in about one-third of the cases reported), talipes equinus, and other forms of club-foot, besides various distortions of the limbs, toes, and fingers. Fulgurant pains may be an early symptom of the affection, but are usually absent throughout, the only disturbances of sensation consisting of aching pains, slight numbness, and various paræsthesiæ, which are rarely severe ; the girdle sensation has been noted in only a small proportion of the cases.

Disorder of speech is usually a late symptom. It varies in form and intensity : sometimes the subject speaks with hesitation and a drawl, sometimes the words are thrown out in a jerky, almost stuttering manner, whilst typical scanning has been reported. Irregularity of pitch, indistinctness of utterance, slurring of the syllables, in various cases have indicated that the laryngeal muscles are affected. Evident lack of control in the movements of the tongue and lips, tremors, choreic or oscillating movements of the tongue, fibrillary contractions of all the muscles about the mouth, loss of power of holding the saliva in the mouth, with a loss of tone in the muscles of expression,—any or all of these symptoms may be present as the outcome of a deep-seated bulbar involvement.

The eye-symptoms are peculiar. Strabismus with diplopia sometimes occurs ; blepharospasm with ptosis has been occasionally noted ; but the characteristic though usually late manifestation of the disease is *nystagmus*. This may take the form of what Friedreich calls *ataxic nystagmus*,—namely, oscillating movements appearing when the eyes are turned upon some object held near ; or that of *static nystagmus*,—that is, movements when the eyes are supposed to be at rest. The pupillary movements may be sluggish, but they are always present, and the Argyll-Robertson pupil has never been noticed. Atrophy of the optic nerve is rare. Vision is occasionally impaired, but contraction of the field has been observed only a few times. Color sense seems not to have been studied.

Although coldness and blueness of the extremities are ordinary vasomotor phenomena of the disease, no trophic changes of the joints and bones have been reported. The intellect is often dull, but there is never acute mental aberration.

DIAGNOSIS.—The symptoms of Friedreich's ataxia vary greatly according to the situation and the development of the spinal sclerotic changes. Usually the nature of the case can be recognized by the occurrence of several cases in one family, the subordination of the sensory to the motor symptoms, and the static incoördination, with the subsequent oscillating or choreic movements and the presence of disturbance of speech, and of nystagmus.

PROGNOSIS AND TREATMENT.—Friedreich's ataxia is incurable, but has little tendency to produce death, which almost invariably occurs from some intercurrent disease, it may be, as long as forty years after the first symptoms. Treatment is of no avail.

SPINAL SYPHILIS.

Syphilis may produce disease of the blood-vessels of the spinal cord and its membranes, with consequent hemorrhage, softening, etc., or it may give rise to a gummous infiltration which commences in the pia mater and spreads inward, causing thickening of the blood-vessel walls, with dilatation of the perivascular spaces and exudation of minute cells around the vessels. The so-called syphilitic callus, a condensation of the fibrous tissues around the cord, is probably not a primary syphilitic lesion, but the scar or remnants of a true gummous infiltration of the membranes.

SYMPTOMATOLOGY.—Spinal softening and spinal neoplasms due to syphilis produce symptoms similar to those caused by similar lesions not due to syphilis. The symptoms of gummous spinal meningitis are those of a localized subacute meningitis,—namely, pain and spasm, with paralysis, affecting some peripheral part corresponding to the seat of the lesion. The pains are sometimes exceedingly severe, furious agonies shooting along the affected nerves or fulgurant crises simulating those of true locomotor ataxia. Often there is aching in the back. When this aching is accompanied by marked soreness on pressure or on jarring, the vertebræ themselves may be considered to be affected. Various paræsthesiæ, marked hyperæsthesia or anæsthesia, girdle pains, tonic spasms, localized tremors, grossly exaggerated reflexes,—such are the symptoms of irritation, which may be followed by complete paralysis with trophic changes.

The symptoms of diffused syphilitic infiltration of the cord vary with the seat of the lesion, simulating now locomotor ataxia, now spastic paraplegia, now chronic myelitis.

DIAGNOSIS.—The diagnosis of spinal syphilis is usually to be reached by a study of the collocation of the symptoms rather than of the symptoms themselves.

The lesions of syphilis are prone to be multiple, and are rarely as strictly confined to individual functional tracts as in sclerosis: consequently, the symptoms of syphilis of the cord are very apt to be mixed. Thus, there will be loss of coördination associated with retention of the patellar reflex; or the patellar reflex may be lost at a time when there is marked loss of power in the muscles rather than loss of their coördinating function; or an apparent locomotor ataxia will be associated with loss of power over the rectum or the bladder; or a case which up to a certain point offers a typical outline of lateral sclerosis suffers from fulgurant pains or from paralysis of the sphincters.

Almost any conceivable mixture or interweaving of spinal symptoms may occur as the result of syphilis of the cord, so that the most pathognomonic evidence of the existence of the disease is an atypical aggregation of symptoms. Whenever the practitioner is confronted by a contradictory mass of phenomena evidently spinal in origin, suspicion should be strongly aroused.

PROGNOSIS AND TREATMENT.—Spinal syphilis is often very favorably affected by treatment, but in a majority of the cases it leaves behind it some permanent traces. Owing to the narrow boundaries of the cord, the secondary pressure and inflammatory effects of specific deposits are most serious : hence the treatment of a case of spinal syphilis should be very vigorous, mercury being used with the utmost freedom, unless there be pronounced cachexia, for the purpose of producing as rapid change as possible. After the mercurial course enormous doses of the iodide should be given, preferably in milk.

CHAPTER VI.

ORGANIC DISEASES OF THE NERVES.

As affording to the student a convenient method of study, and, to the medical practitioner, of reference, we shall begin the present chapter by pointing out the exact seat of the palsies which follow loss of function in the more important individual nerves. In the distribution of paralysis it makes no difference, of course, whether the lesion is centric or peripheral. Having, however, located the nerve territory of a paralysis, the practitioner should usually have no difficulty in deciding by an electrical study of the affected muscle whether the lesion is centric or peripheral,—it being understood that the word peripheral as here used includes the trophic centres in the spinal cord as well as the motor nerves. Localized spasms are, of course, referable to their proper nerve by reading spasm instead of paralysis in the text.

LOCAL PARALYSES OF MOTION.

Oculo-Motor Paralysis.—Dilatation of the pupil, ptosis or dropping of the upper lid, paralysis of accommodation, and squint with consequent double vision, are symptoms of loss of power of the oculo-motor nerve, whose superficial origin is from the inner border of the crus cerebri, the deep origin being in the locus niger of the peduncles and the gray nucleus in the floor of the aqueduct of Sylvius slightly below the tubercula quadrigemina. Partial paralysis of this nerve is frequent. In such cases the symptoms vary according to the portion of the nerve affected. The functions of the eye-muscles are as follows: to turn the eye—superior oblique, downward and outward; inferior oblique, upward and outward; superior rectus, upward and inward; inferior rectus, downward and inward; internal rectus, directly inward; external rectus, directly outward. All these muscles are supplied by the oculo-motor nerve except the superior oblique and the external rectus. When one of these muscles is paralyzed a squint results. In order to determine which muscle is affected, it is only necessary, at least in cases of fresh paralysis, to note the position of the head. The rule is, the head is so deflected that *the chin is carried in a direction corresponding to the action of the paralyzed muscle.* Megalopsia, or macropsia, in which objects look larger than normal, is said to indicate paralysis of the external rectus. Micropsia, in which objects look smaller than normal, is said to indicate paresis of the internal rectus. These two symptoms are very rare.

Paralysis of the oculo-motor nerve, also of the trochlear and of the abducens, is commonly due to pressure upon the nerve by basal exudations, which in adults are usually syphilitic, in children tubercular or rachitic. Rheumatic or gouty paralysis is not very rare. Centric palsy may occur.

Fourth or Trochlear Nerve.—Loss of power of the superior oblique muscle of the eye is diagnosed by the fixedness of the eye when the head is moved, or, in other words, by the moving of the eye with the head. Double vision occurs whenever the subject attempts to look straight downward or at objects situated towards the paralyzed side; but the second image disappears when the head is turned to look towards the sound side. The distortion of vision is especially manifested when any attempt is made to pick up a small object, as a coin, off a table. The nerve involved is the fourth, trochlear, or pathetic, whose apparent or superficial origin is in the superior peduncle of the cerebellum. Its fibres have been traced into the peduncle to the valve of Vieussens, near the tubercula quadrigemina, where they decussate with corresponding filaments of the opposite side.

Ophthalmoplegia.—Under the name of *ophthalmoplegia interna* Jonathan Hutchinson described an affection of the eye which he believed to be the result of paralysis of the ciliary ganglion. The symptoms are *iridoplegia*, or paralysis of the iris, both as to the circular and the radiating fibres, and *cycloplegia*, or paralysis of the ciliary muscle.

Ophthalmoplegia externa of Hutchinson, previously described by Von Graefe as *ophthalmoplegia progressiva*, is paresis or paralysis of all the external muscles of the two eyes. In most cases the internal muscles are affected. The cause may be an exudation beneath the brain involving both sets of nerves, or a poliomyelitis attacking the nuclei of the nerves.

Fifth or Trigeminus Nerve.—Loss of power in the muscles of mastication,—*i.e.*, the temporal, masseter, and pterygoids,—and in the mylohyoid, digastric, tensor palati, and tensor tympani, indicates paralysis of the motor root of the fifth or trigeminus nerve. This root has its apparent origin in the side of the pons; its deep origin is in a nucleus just below the lateral angle of the fourth ventricle, immediately in front of the nucleus of the facial nerve.

Paralysis of those fibres of the trigeminal or fifth nerve which come from the ascending or sensory root produce loss of sensation in the face, including the forehead, the eye, and the external ear, and also in the mucous membrane of the mouth, the hard and the soft palate, the nose, and the middle ear, and in the teeth. Disturbances of the trigeminal nerve are also apt to be accompanied by disorder of the secretory function of the lachrymal, nasal, and buccal glands, by herpetic eruption, and by a severe conjunctivitis which is believed by some to be of trophic origin, and by others to be caused by the loss of sensibility preventing the immediate recognition of foreign bodies in the eye.

Sixth or Abducens Nerve.—Paralysis of the abducens nerve causes loss of power in the external rectus, with consequent internal strabismus, or squint, double vision, and sometimes macropsia. Internal squint does not, however, always indicate paralysis of the sixth nerve, because the weakness of the external rectus muscle is a very frequent result of imperfection of vision. The apparent origin of the abducens nerve is from a groove between the anterior pyramid of the medulla and the posterior border of the pons. There are usually two roots, one from the medulla and the other from the pons. The fibres have been traced to a nucleus which lies underneath the fasciculus teres in the floor of the fourth ventricle. A few fibres are believed to pass from this nucleus upward and across to join the third nerve of the opposite side. In this way are explained certain rare cases of conjugate paralysis involving the internal rectus of one side and the external rectus of the other side and accompanied by atrophy of the nucleus of the abducens nerve.

Facial Nerve.—Of all the nerves of the body the facial or seventh nerve is most frequently paralyzed. The superficial origin of this nerve is in a groove between the olivary and restiform bodies of the medulla. Its deep origin is probably in the upper portion of the pons, although its fibres have not been distinctly traced farther than a nucleus in the upper half of the floor of the fourth ventricle near the postero-median fissure. It supplies all the muscles of the face, except those of mastication, also the levator palati and the tensor tympani.

Centric paralysis of the facial nerve is common. It is never complete, and almost invariably affects the muscles about the corner of the mouth. Complete paralysis of the nerve is always peripheral, and is usually due to neuritis or perineuritis. For an account of it, and also of paralysis of different portions of the nerve, see Neuritis, Facial, page 611.

Auditory Nerve.—The eighth, or auditory, nerve has the nucleus of its larger root in the floor of the fourth ventricle, that of its minor root near the restiform body. The roots pass obliquely outward and unite into a single trunk, which appears at the lower edge of the pons on the outer side of, and close to, the facial nerve. After leaving the medulla oblongata the nerve is directed outward, in company with the facial nerve, to the internal auditory meatus.

Deafness from disease of the auditory nuclei is very rare. Peripheral neurotic deafness is much more common. The auditory nerve is liable to be pressed upon by syphilitic, tubercular, or other deposits at the base of the brain, and is especially exposed to paralysis from disease of the mastoid processes of the temporal bone. It may therefore be laid down as a diagnostic rule, the exceptions to which are very rare, that a nervous deafness not associated with marked giddiness is dependent upon a lesion of the nerve-trunk. Hyperæsthesia of the auditory nerve produces a loss of hearing which is characterized by excessive susceptibility to sounds. The normal stimuli of the nerve produce pain rather than normal functional excitement, so that, although unable to perceive minute differences in sounds, the patient suffers acutely from loud noises.

PNEUMOGASTRIC NERVE.

The important branches of this nerve are the pharyngeal, the laryngeal, and the cardiac.

Pharyngeal Branches.—As these branches in connection with the glosso-pharyngeal nerve form the pharyngeal plexus, their paralysis is followed by difficulty of swallowing. The part played in the formation of the plexus seems, however, somewhat secondary, as loss of power in a single pneumogastric nerve does not seriously impair the power of swallowing.

Laryngeal Branches.—The superior laryngeal nerve supplies the laryngeal membrane above the cords and the crico-thyroid muscle; the inferior or recurrent laryngeal supplies the remainder of the mucous membranes and muscles of the larynx. Owing to its proximity to the arch of the aorta on the left and the subclavian artery on the right side, the inferior nerve is frequently involved in aneurisms.

The symptoms of paralysis of the laryngeal muscles vary according as the abductors or the adductors are affected. Adductor paralysis is the common form, producing the hoarseness or aphonia of hysteria, ordinary colds, etc., without disturbances of respiration. The muscles involved are the lateral crico-arytenoid and the arytenoid.

The failure of the abductors, the posterior crico-arytenoids, to draw apart the vocal cords during inspiration, results in the glottis being almost or entirely closed by the pressure of the air from without, so that inspiration is almost impossible and is accompanied with a loud stridor. This form of laryngeal paralysis is dangerous to life. It may result from pressure upon both vagi or both recurrent nerves, but may be due to a central lesion or to hysteria. When only one nerve is involved there is merely hoarseness with failure of the cord to move in inspiration, as seen by the laryngoscope. Spasm of the abductor muscles seems to be rare, and to produce only disturbances of voice; on the other hand, spasm of the adductors by closing the glottis causes violent dyspnœa, as in laryngismus stridulus, false croup, etc.

Anæsthesia of the larynx is especially important, because by preventing the automatic watchfulness of the glottis it is apt to lead to the entrance of particles of food into the larynx or the lungs. It is a common condition in advanced dementia paralytica.

Cardiac Branches.—So far as concerns the heart, the vagi have motor, sensory, and trophic functions; irritation of the vagi produces slowness, paralysis of the vagi great rapidity, of the heart's action. We do not know what symptoms are produced by disturbances of the sensory fibre of the pneumogastric; it may be that the pain of angina or other heart disease is connected with them.

Pulmonary Branches.—Of these we have not sufficient knowledge to speak with any confidence. It is possible, but not proved, that asthma is connected with them.

Gastric and Œsophageal Branches.—These branches play an important part in the act of vomiting. They probably carry both afferent and efferent impulses between the base of the brain and the stomach. *Gastralgia*, or neuralgia of the stomach, is believed by some authorities to be an irritation of the pneumogastric peripheral endings, and it is possible that these nerve terminations are connected with many dyspeptic disturbances.

Glosso-Pharyngeal Nerve.—Paralysis of the glosso-pharyngeal nerve is revealed by difficulty of swallowing, with great tendency to regurgitation of food through the nostrils, and loss of taste in the posterior third of the tongue. The superficial origin of the nerve is in the groove between the lateral tract and the restiform body of the medulla oblongata. Its fibres have been traced to a nucleus in the floor of the fourth ventricle.

Spinal Accessory Nerve.—The spinal accessory nerve is composed of fibres springing from the lateral columns of the medulla oblongata and of fibres which rise between the anterior and posterior roots of the first and fifth cervical nerves, the two parts being united in the cranium and escaping as one nerve through the jugular foramen. The spinal accessory nerve sends communicating fibres to the pneumogastric, which go to the laryngeal muscles and control phonation; the act of deglutition is also affected by the nerve, which further affords the chief but not the only supply of the sterno-mastoid and trapezius muscles. Paralysis of the sterno-mastoid muscle causes slight elevation of the chin, with rotation towards the paralyzed side, causing an oblique position of the head. There is difficulty in depressing the head towards the paralyzed muscle, whose normal outline in the neck is also softened. If both muscles are affected, the head is held straight, and is rotated with great difficulty; great difficulty is also experienced in depressing the chin. Paralysis of the trapezius muscle is shown by sinking of the point of the shoulder, by drooping downward of the scapula, the inferior angle of which is in the relation of adduction to the spine as compared with its fellow, and by prominence of the clavicle and supraclavicular space. If there is also difficulty in raising the scapula and clavicle and in elevating the arm, the upper fibres of the muscle are especially involved; while if the scapula is not easily approximated to the spinal column, the middle and lower fibres are chiefly affected. If after complete paralysis of the trapezius there is absolute inability to draw the scapula towards the spine, palsy of the rhomboideus major and rhomboideus minor muscles may be inferred. Under similar circumstances, loss of the power of elevating the scapula and of moving the neck after fixation of the scapula indicates paralysis of the levator anguli scapulæ.

Long Thoracic Nerve.—If the scapula is drawn upward with its lower angle approximated to the spine, and if during the act of elevating the arm the lower angle of the bone does not describe an arc outward, as it normally should, but approaches still nearer to the spine, while the vertebral border stands out in a wing-like manner, leaving a well-marked depression between it and the thorax, there is paralysis of the serratus magnus, which is supplied by the posterior thoracic or long thoracic or external respiratory nerve of Bell.

Subscapular Nerves.—Difficult adduction of the arm, with loss of the normal power of depressing it and drawing it backward, especially in the act of placing the hand in contact with the buttock, shows paralysis of the latissimus dorsi muscle, which is chiefly supplied by the subscapular nerves. Inability to perform properly inward rotation of the humerus, diminished power of pronation, excessive outward rotation of the upper arm, and consequent faulty position of the hand, denote paralysis of the subscapularis and teres major muscles, which receive their nerve-supply from the subscapular nerves.

Suprascapular and Circumflex Nerves.—Impaired power of outward rotation of the humerus, and consequent difficulty in performing such acts as writing, drawing, and especially sewing, in which this movement is essential, together with excessive inward rotation, even to the point of turning the ulnar border of the hand uppermost, indicate paralysis of the important external rotator of the humerus, the infraspinatus muscle, as well as of its assistant, the teres minor muscle. The former is supplied by the suprascapular nerve, and the latter by the circumflex.

When the arm cannot be directly elevated,—*i.e.*, brought at right angles with the trunk,—but hangs helpless close to the thorax, and, later, when a definite space appears between the head of the humerus and the acromion, there is paralysis of the deltoid muscle, which is supplied by the circumflex nerve. Anæsthesia of the skin is not always present.

Anterior Thoracic Nerves.—Inability to adduct actively the arm so as to draw it across the chest or to place the hand on the opposite shoulder, abnormal prominence of the ribs and intercostal spaces, and loss of tension of the anterior border of the axillary space, are the symptoms which show paralysis of the pectoralis major and pectoralis minor muscles, supplied by the anterior thoracic nerves.

Musculo-Cutaneous Nerves.—Absence of the greater part of the power to flex the forearm, with loss of some of the power of supination, and partial lack of ability to draw the humerus forward, inward, and towards the scapula, points to paralysis of the group of muscles supplied by the musculo-cutaneous nerve,—viz., the biceps cubitis, the coracobrachialis, and part of the brachialis anticus.

Musculo-Spiral Nerve.—If the hand hangs at right angles to the forearm (wrist-drop) and the power of extension at the wrist-joint and elbow-joint is absent, with the hand in pronation, the fingers bent, and the thumb flexed and adducted, the deformity is characteristic of the group of muscles supplied by the musculo-spiral nerve and its posterior interosseous branch,—viz., the triceps and anconeus, the supinator longus, the extensor carpi radialis longior, the extensor carpi radialis brevior, and all the extensor muscles of the superficial and deep posterior brachial regions. Other prominent symptoms are that the effort at extension of the fingers is possible only in the second and end phalanges, while the first phalanges are more flexed (the interossei flexing the first phalanges and extending the others). The hand-grip is weakened unless the wrist-joint be put into extension, and when the hand and the forearm are put prone upon the table there is diminished power of abduction and adduction. The forearm cannot be brought midway between pronation and supination, and when it is in this position the ability to perform elbow-joint flexion is impaired. Finally, the forearm cannot be extended upon the arm. Numbness and tingling may exist, but complete anæsthesia is very rare.

Median Nerve.—Loss of the power to flex all the second phalanges and the end phalanges of the index and middle fingers, preservation of this motion in the first phalanges (interossei), and its partial preservation in the two outer fingers, inability to flex the thumb or to bring it in apposition with the little finger, diminished power in flexing the wrist, which, when this is attempted, throws the hand into a marked adduction, and impaired pronation with lessened sensibility of the first two fingers and the radial side of the ring finger, indicate paralysis of the median nerve. This nerve supplies all the flexor and pronator muscles of the deep superficial and anterior brachial region, with the exception of the flexor carpi ulnaris and the ulnar half of the flexor profundus digitorum, which are supplied by the ulnar nerve, and also all the muscles of the thumb except the adductor and one head of the flexor brevis pollicis, and finally the two outer lumbricales. Sensory fibres supply the radial side of the palm, the front of the thumb, the first two fingers, half of the third finger, and the back of these three fingers.

Ulnar Nerve.—Imperfect flexion of the hand, which is drawn towards the radial side; impaired power of adduction of the hand; lessened ability to separate the fingers (abduction) or to bring them together (adduction); absence of the power to flex the first row of the phalanges and extend the other two rows; almost entire immobility of the little finger; difficulty in opposing the thumb to the metacarpal bone of the index finger, with disturbed sensation of the entire little finger and the ulnar side of the ring finger, constitute the symptoms of paralysis of the muscles supplied by the ulnar nerve. These muscles are the flexor carpi ulnaris, part of the flexor profundus digitorum, the interossei, and the two inner lumbricales, all the muscles of the little finger, and the adductor of the thumb and one head of the flexor brevis pollicis.

When the interossei and lumbricales are no longer able to flex the first row of the phalanges and extend the other two rows, but the extensor communis digitorum excessively extends the first row of the phalanges, while the flexor muscles bend the second and third rows, the condition of "claw-hand" is produced, which may mean paralysis of the ulnar nerve just above the wrist, so that the innervation of the interossei and lumbricales alone is affected. Affections of the ulnar nerve produce sensory disturbance of the ulnar side of the hand, two and a half fingers on the back, and one and a half fingers on the front.

Spinal Nerves.—If the head hangs forward and cannot be extended, or at least can be extended only by the aid of a swinging motion, there is paralysis of the extensors of the cervical vertebræ,—*i.e.*, the rectus capitis posticus major, the rectus capitis posticus minor, the upper portion of the trapezius, and the splenii.

When the spine tends to assume a posterior curvature, most marked in the dorsal region, and the patient presents the appearance of "old man's back," in which he cannot voluntarily straighten the curvature, although this may be done by passive action, there is paralysis of the extensor muscles of the back, chiefly the longissimus dorsi and sacrolumbalis, with the condition of *paralytic kyphosis.* The production of *lateral curvature*, or *paralytic scoliosis*, means that the paralysis is limited to one side.

When a patient carries the body with the upper portion bent backward, so as to throw it behind the centre of gravity, and when the body if inclined too far anteriorly falls forward and cannot again assume the erect posture until the hands, being placed upon the legs, help the arms by a sort of climbing process to bring the body again to its backward posture, the condition of paralysis of the extensor muscles of the lumbar region obtains,—*i.e.*, the erectores spinæ. In this condition the patient further stands with the head bent forward, walks with a swaying motion of the trunk, and when he sits down the upper part of the body apparently sinks, so that the dorsal spine is bent (*kyphosis*), while there is a deep concavity of the lumbar spine (*lordosis*). The nerves concerned in these palsies of the back are the posterior branches of the spinal nerves, cervical, dorsal, or lumbar, according to the region involved.

Ilio-Hypogastric, Ilio-Inguinal, and Intercostal Nerves.—Inability to compress properly the contents of the abdominal cavity, so that such acts as urination, defecation, and vomiting are performed with difficulty, and diminished power in the effort of respiration, together with a tendency to fall backward when the upper part of the trunk is inclined posteriorly, show paralysis of the abdominal muscles, which are supplied by the ilio-inguinal, ilio-hypogastric, and lower intercostal nerves.

Anterior Crural Nerve.—Loss of the power to flex the thigh upon the abdomen and extend the leg at the knee, and impaired ability to raise the body from the recumbent posture and to perform the acts of walking, running, going up-stairs, and the like, are the symptoms which indicate paralysis of the group of muscles supplied by the anterior crural nerve,

—viz., the iliacus, the pectineus, and all the muscles on the anterior surface of the thigh except the tensor vaginæ femoris.

Obturator Nerve.—When the act of pressing the knees firmly together, or of crossing one leg over the other, cannot be properly performed, and when there is impaired power of external rotation of the thigh while in the sitting posture, the indications are that there is paralysis of the gracilis and adductor muscles of the internal femoral region and of the external obturator muscle, which group is supplied by the obturator nerve.

Superior and Inferior Gluteal Nerves.—Uncertainty in the act of walking or standing, together with loss of power of internal rotation of the thigh and impaired power of external rotation, difficulty in abducting the thigh, with disturbed relation of the thigh to the pelvis, and inclination of the latter to the opposite side during attempted action on the part of the affected limb, are the symptoms which point to paralysis of the muscles supplied by the superior and inferior gluteal nerves. The inferior gluteal nerve is distributed to the gluteus maximus, which muscle can forcibly extend the thigh on the pelvis and perform outward rotation of the thigh. The superior gluteal nerve passes to the tensor vaginæ femoris and to the gluteus medius and gluteus minimus. The anterior fibres of these latter muscles rotate the thigh inward, whilst their posterior fibres rotate it outward. The muscles of the gluteal group when they take their fixed point from the pelvis are abductors of the thigh; when they take their fixed point from the femur they support the pelvis on the femur. The tension of the fascia lata, which may be slackened in palsy, is usually maintained by the gluteus maximus and the tensor vaginæ femoris.

Sciatic Nerve.—Inability to flex or bend the knee, to oppose resistance to passive extension of the knee, and to raise the heel towards the buttock, would show loss of power in the semimembranosus, semitendinosus, and biceps femoris muscles, a group supplied by the great sciatic nerve. This is a possible form of paralysis; but more usual are the palsies which occur from affections of the principal branches of its distribution, and consist in loss of the extension and flexion of the foot and toes and abduction and adduction of the foot.

External Popliteal Nerve.—If the foot cannot be flexed or abducted, nor completely adducted, and hangs downward, so that the patient in the act of walking raises the foot by flexing the hip-joint and then places it again upon the floor in such a manner that the point of the toes and the outer border of the foot touch the ground first, the symptoms are characteristic of paralysis of the muscles supplied by the external popliteal or peroneal nerve. This nerve, through its two branches, the anterior tibial and the peroneal cutaneous, supplies the muscles of the anterior portion of the leg and the extensor brevis digitorum on the dorsum of the foot. Its sensory fibres go to the outer half of the leg, to the dorsum of the foot, and to the toes, except the outer side of the little toe and the adjoining sides of the great and the second toe.

Internal Popliteal Nerve.—If the foot cannot be extended, nor the toes flexed or moved laterally, and if the patient cannot stand upon his toes or properly adduct the foot and raise its inner border, paralysis of the group of muscles supplied by the internal popliteal nerve and its continuation, the posterior tibial nerve, may be inferred. This group consists of the muscles of the calf and of the deeper posterior leg-region, and, through the external and internal plantar nerves, of those of the sole of the foot. In this palsy the great toe can neither be flexed nor moved from side to side. The foot may assume an appearance similar to the "claw-hand" described under palsy of the ulnar nerve, and for the same reasons. The external or short saphenous branch of the internal popliteal sends sensory fibres to the outer side of the foot and the little toe, whilst the posterior tibial supplies the heel and the sole of the foot.

NEURITIS.

Neuritis is divisible into simple neuritis or perineuritis, multiple neuritis, and mesoneuritis. Of these forms simple neuritis and multiple neuritis are especially related, because in their clinical manifestations the cases grade into one another, and because the two disease processes may apparently coexist in one nerve-trunk and give a mixed lesion.

Pressure Palsy.—Although the lesion is probably not a true neuritis, at this point may be considered the paralysis which follows continuous pressure upon the nerve. Any nerve of the body may be affected, but in the ordinary form of the disorder the musculo-spiral of the arm suffers. The common cause is pressure by the head upon the arm during the sleep of drunkenness, or occasionally in the newly married. The chief symptoms are marked numbness and tingling, without great pain, and loss of power in the tributary muscles.

The treatment consists of massage, faradization of the muscles, and in extreme cases hypodermic injections of strychnine into the affected muscles. The prognosis is absolutely favorable.

The functions of a nerve may also be suspended by violent blows, and it is probable that from such cause the peripheral nerve-endings in the muscles especially suffer. The circumflex nerve with the deltoid is the most commonly affected, because the points of the shoulders are so often hurt in falling. The treatment should be leeching, fomentations, lead-water and laudanum, until the immediate effects of the bruise have subsided, followed by the use of electricity and massage.

NEURITIS. PERINEURITIS.

DEFINITION.—An inflammation which primarily attacks the sheaths and connective tissue of nerves, and usually is confined to a single nerve or a very few nerves.

ETIOLOGY.—Simple neuritis may be the result of injuries to the nerve, of extension of inflammation from diseased neighboring parts, of exposure to cold (this form probably rheumatic), and of the action of the rheumatic and certain other poisons. Toxic neuritis (except the rheumatic) is, however, usually of the multiple type.

Pelvic neuritis is not rare after childbirth. It may be septic, but more often is a direct result of an injury to the pelvic nerves by the forceps or by the head of the child.

MORBID ANATOMY.—Simple neuritis is a neuritis affecting primarily the sheath and extending to the connective tissue between the fibres. The nerve is red, swollen, and infiltrated throughout with leukocytes. The nerve-fibres are finally invaded, the nuclei of the sheaths increasing greatly in number, and the axis-cylinders becoming varicose and at last disintegrating into a granular débris. The process continuing may end in the complete destruction of the nerve.

SYMPTOMATOLOGY.—The symptoms of neuritis are pain, tenderness, and disturbances of motion, sensibility, and nutrition. The pain is shooting or burning, and follows closely the distribution of the nerve affected. It is persistent, with paroxysmal exacerbations, and is greatly increased by pressure and by active or passive movements. Early in the attack there is hyperæsthesia, usually over the whole nerve distribution ; later there are losses of general sensibility, and in rare instances analgesia. In most cases atrophy of the muscles develops very slowly, but it may finally be complete. It is accompanied by changes in the electro-contractility of the muscles, reaction of degeneration, and even complete loss of galvanic contractility. There is a true loss of power in the muscles, which in the early stages may be entirely merged in the complete immobility produced by excessive pain. Pronounced trophic disturbances may occur in any or all of the tissues supplied by the nerve. Herpetic, bullous, or eczematous irritations appear, but more characteristic is the so-called glossy skin, in which the surface of the skin has a very fine, almost silky appearance, with pronounced glossiness and loss (especially apparent about the hands) of the normal lines and creases. These changes are associated with local rise of temperature, excessive sweating, alteration of the hairs, which are sometimes enormously developed, irregularity in the growth of the nails, and deformations involving changes in the bony structure, especially about the small joints.

Simple neuritis may exist for years without destruction of the nerve, but in rare cases it finally gives rise to paralysis with atrophy and contractions of the muscle, anæsthesia, and other disturbances of sensation ; complete relief from pain seems almost never to come.

DIAGNOSIS.—The only disorder with which a simple neuritis could be confused is neuralgia, but in the latter affection there are no marked tenderness of the nerve-trunks, no excessive pain on passive movements, no loss of motor power, and no trophic changes in the muscles, skin, nails, etc.

PROGNOSIS.—The prognosis of simple neuritis is always serious, unless the lesion be due to rheumatism or other cause which can be readily overcome. The more severe the symptoms, especially the more pronounced the trophic lesions, the less the probabilities of rapid cure.

TREATMENT.—When a cause can be assigned for a neuritis and is amenable to treatment, such treatment should be vigorously carried out. Thus, the salicylates should be given in large doses in a rheumatic neuritis, the iodides in a syphilitic neuritis, etc. So far as the neuritis itself is concerned, there is no reason for believing that drugs are directly effective. The general health must, of course, be sustained, and anodynes as necessary given for the relief of pain. Absolute rest of the part must be enjoined ; with it should be associated free counter-irritation by means of blisters, which are to be repeated from time to time almost indefinitely. In the very onset local bleeding by means of leeches may be of value. In some cases the continued application of cold by means of ice or of cold water is of service, but, as this measure is very capable of doing harm, it must be tried with caution. In other cases warm or even hot water gives the greatest relief. In our experience the immediate effect upon the patient of these applications affords a true index of their usefulness.

In the beginning of the attack no form of electrical treatment should be employed ; later, great relief may sometimes be obtained by passing towards the periphery of the nerve a continued electrical current, of such strength as not to cause distinct pain. It is essential that it be passed uninterruptedly from ten to thirty minutes once or twice a day. When the electrical current does good in these cases it almost invariably relieves pain during its application ; if it increases the pain it is almost certain to do harm. The faradic current usually increases the pain and is distinctly injurious, but in a few cases in which the symptoms are very lethargic it is of service.

Early in the attacks massage is very painful and of exceedingly doubtful utility, though temporary relief and perhaps permanent good may be obtained by the most gentle effleurage ; later, when there is no activity of inflammatory processes, and when the nerve-sheaths are full of exudation, cautious, careful massage may do great good, although it produces pain at the time of the application.

PARENCHYMATOUS NEURITIS. MULTIPLE NEURITIS.

DEFINITION.—A peculiar degeneration or inflammation which attacks especially the nerve-fibres, and usually involves numerous nerves at one time.

ETIOLOGY.—Multiple neuritis may be produced by a poison, or may occur during certain dyscrasiæ and diseases of the nerve-centres. In rare cases it arises without obvious cause and is spoken of as spontaneous. The most common cause is alcohol, but the disease may be due to lead, arsenic, mercury, antimony, and other metallic poisons, or to various non-metallic poisons, among which may be mentioned carbonic oxide, sulphuret of carbon, sulphuretted hydrogen, sewer gas, and phosphorus: it is probable that the cases which follow infectious diseases, such as diphtheria, small-pox, typhus or typhoid fever, spotted fever, sepsis, beri-beri, etc., are caused by autochthonous poisons. Even when due to dyscrasiæ such as occur in diabetes, phthisis, and cancer, it is probably of toxic origin. Sometimes in old people it is the first symptom of dry gangrene, when it is probably due to disturbance of the blood-supply of the nerves by atheromatous obstruction.

In organic diseases of the nerve-centres, notably locomotor ataxia, parenchymatous neuritis is often a late complication; on the other hand, it sometimes precedes the coming on of the central disease; so that there is plausibility in the theory which assigns the diseases of the centre and of the nerves to the action of a common irritant poison.

MORBID ANATOMY.—The lesions of parenchymatous neuritis resemble those which follow section of the nerve. The nuclei of the sheath of Schwann proliferate freely, the myelin becomes swollen and finely granular, and the axis-cylinders themselves grow granular, deformed, swollen, varicose. The sharp line between the two parts of the fibre disappears, and the nerve-fibre finally consists of débris in the sheath of Schwann. Rarely is the lesion uniform along the nerve, and it may exist in segments separated by an almost unaltered nerve, constituting the *segmentary neuritis* of Gombault. In some cases along with the changes which have been noted there is free proliferation of the connective tissue of the nerve and of its sheath, forming what must be looked upon as a mixed type, uniting parenchymatous with simple neuritis.

The muscles supplied by the affected nerve undergo a degeneration in which the fibrillæ shrink, become fatty, and finally disappear.

SYMPTOMATOLOGY.—In its most malignant form acute multiple neuritis commences abruptly with a chill, great prostration, and high fever, accompanied by violent fulgurant or burning pains, formication, numbness, paræsthesia, and hyperæsthesia. The loss of power which is manifested from the first usually develops simultaneously in two or sometimes in all four extremities, especially affecting the muscles of the hands and arms and those of the legs proper. Rapidly the palsy creeps towards the trunk, and at the same time hyperæsthesia passes into the loss of all forms of sensibility, constituting an anæsthesia dolorosa, which may affect the skin before it does the deeper tissues, so that there arises a pathognomonic association of complete loss of sensibility in the skin with excessive sensitiveness of the muscles and other deep structures. Involvement of the nerves of the trunk may give rise to a pronounced girdle sensation. The trigemini usually escape, but may be involved.

The nerves of special senses may also be implicated, with resultant partial blindness and deafness and loss of taste, whilst double vision and irregular pupils show that the oculo-motor or other eye-nerves are affected. As the paralysis spreads, speech and swallowing become involved, and finally death may occur in forty-eight hours from respiratory paralysis. If the patient live, trophic changes show themselves in pigmentation and thickening of the skin, in eczematous eruptions, in alterations of the nails, and even in œdema and bed-sores. The muscles waste early, and the reaction of degeneration soon appears. The deep reflexes are early abolished, but the sphincters are rarely if ever affected.

In some very acute cases of polyneuritis the disturbances of sensation are very slight, the symptoms being those of an ascending paralysis. There are probably, however, always more numbness, tingling, and tenderness, especially over the nerve-trunks, than in true Landry's paralysis.

In chronic multiple neuritis there is a symmetrical loss of power in the extremities, slowly increasing through many weeks without much pain, and little by little putting on the trophic symptoms of the fully formed disease. In the full cycle of its development such a case may last for years, passing through an initial stage, a progressive stage, a stage of standing still, and a stage of regeneration, and terminating in an after-condition of more or less impaired function. In such cases motion usually suffers much more markedly than does sensation. Often the first evidence of recovery is the ability of the patient by a strong effort of the will to make movements in the muscles, which are, however, so weak that they do not affect the parts supplied. The anal sphincter and the bladder are very rarely attacked. Girdle pains are extremely infrequent. When present, they probably depend upon degeneration of a nerve of the trunk. In some cases coördination may be lost long before muscular power, and a pronounced retardation of sensation has been noted. Very frequently in severe cases there are marked psychical disturbances, with a strong depression of intelligence and of memory.

Any of the nerves may be attacked, but the hypoglossal, the spinal accessory, and the glosso-pharyngeal rarely suffer, unless it be in the diphtheritic form of neuritis. It is extremely unusual for the nerves of special sense to be implicated. The usual course of the disease from the centre to the periphery is said to be sometimes reversed.

Between the two extremes of the very acute and the very chronic form of polyneuritis every grade occurs. As the symptoms correspond in some degree with the cause, the following etiological varieties may be recognized :

Alcoholic Neuritis.—Although alcoholism is much more common in men than in women, alcoholic neuritis appears to be more frequent in women. It is usually of slow development, the loss of power being preceded by sensory symptoms, such as numbness and tingling, often accompanied by excessive pain and painful cramps. The hands and feet, which are the first parts affected, are usually cold, often discolored; urticaria or other irritation, or glossy skin, may be present. The paralysis is mostly localized in the extensors. The course of the disease is usually essentially chronic, but there is an acute form in which death from respiratory paralysis may result in a few days. If the disease persists, contractures and deformities occur. Alcoholic neuritis is often associated with cerebral disturbances; in the acute cases convulsions, delirium with hallucinations, mania, melancholia, and delusions occur, but they should be looked upon as due to alcoholism rather than as belonging to the neuritis, the brain and other nerve-centres in these cases suffering along with the peripheral nerves from the effects of the poison.

Post-febrile Neuritis.—The neuritis which follows small-pox or other exanthematous disease most frequently attacks the legs, and is not rarely limited to the nerves of one leg. It is not commonly accompanied by much pain. (For diphtheritic neuritis, see Diphtheria.) The *diabetic neuritis* is apt to be accompanied by excessive pain, and in some cases is followed by rapid trophic changes, and even sloughing. *Senile neuritis* is usually accompanied by much greater sensory than motor disturbances. Its ordinary seat is the feet and the calves of the legs.

DIAGNOSIS.—Acute multiple neuritis is distinguished from Landry's paralysis and from acute poliomyelitis by the presence of sensory disturbances, and especially of tenderness of the nerve-trunks. Pain is said to be wanting in some cases of multiple neuritis, but if in any case there is no tenderness of the nerve trunks the diagnosis of multiple neuritis is not justified. We have seen cases of lead poisoning without fever, with wasting of the muscles, without disturbances of sensibility and without tenderness of the nerve-trunks, but with implication of the sphincters. The probability is that these cases are of spinal origin, a conclusion which is confirmed by the extraordinary benefit derived in them from the use of strychnine. Locomotor ataxia and peripheral neuritis frequently coexist. In such case neuritis is to be recognized by the nerve-trunk tenderness. Cases have been reported in which an apparently true ataxia has been proved by post-mortem to have been due to neuritis without spinal disease. The gait of neuritis usually differs from that of locomotor ataxia in that the loss of power in the extensors of the feet causes the legs to be lifted very high in order to avoid catching the toes in obstacles (*steppage gait* of Charcot).

PROGNOSIS.—In malignant cases the danger of death is in direct proportion to the severity and rate of progress of the symptoms. Most cases of multiple neuritis recover more or less completely, but months and sometimes years are necessary. Cases produced by arsenic and other metallic poisonings often get well after the muscles have so far disappeared as to yield no contractures to any stimuli.

Beri-beri.—This is an endemic disease of China, Japan, and various tropical countries, which has as its basal lesion a wide-spread peripheral neuritis. As there are associated with this neuritis rapidly developing anæmia, œdema, general anasarca, serous effusions, and in severe cases gastro-intestinal symptoms, suppression of urine, and a rapidly progressive exhaustion, it is plain that the disorder is not in its essence a peripheral neuritis, but a general disease whose nature remains very obscure. The discovery of a specific micrococcus has been asserted. It is said to have been almost driven out of the Japanese navy, where it was formerly particularly abundant, by an improvement of the rations. An epidemic of multiple neuritis which has been described as occurring on fishing-vessels upon the Grand Banks of Newfoundland has associated with it œdema, shortness of breath, and great general failure, which suggest kinship with beri-beri.

MESONEURITIS.

Definition.—An inflammation of the nerve lymphatics.

In 1881 Renaut described a lymphatic apparatus within the sheath of the nerve-trunks, which, rudimentary in man, is completely developed in the horse. In 1885 Varaglia first spoke of the occurrence of disease in this apparatus. Schultze subsequently gave detailed descriptions. The lesion has been noted as occurring in numerous diseases, as tabes, acromegalia, syringomyelia, acute multiple neuritis, myopathies, alcoholism, etc., but we have no clinical knowledge of symptoms produced by it. Mesoneuritis occurs in two forms: the nodular type consists of minute spheroidal masses in the centre of which is an amorphous substance, while in the outer portion are swollen, granular, endothelial cells with nucleated corpuscles; the lamellated type consists of plaques composed of thickened lamellæ analogous to those constituting the sheath. We have no distinct knowledge of the nature or etiology of these changes, which are supposed to be the result of local irritation.

SCIATICA.

Definition.—A perineuritis of the sciatic nerve.

Any nerve-trunk may be attacked with inflammation, but neuritis of the trigeminal, the trifacial, and the sciatic nerves is so frequent and so important as to require special notice.

Etiology.—Sciatic neuritis may be produced by any of the causes of nerve inflammation heretofore enumerated. In its ordinary form it is a gouty or a rheumatic affection. It is frequently produced by exposure, and is more common in men than in women.

Symptomatology.—The one prominent symptom of sciatica is pain, increased by movement, both passive and active, and also by pressure, especially upon the nerve-trunk. Hyperæsthesia, and later anæsthesia, are almost constant phenomena in bad cases. The pain is more or less constant, but often occurs in paroxysms of great intensity, and, whilst it may affect the whole or any part of the distribution of the nerve, is especially referred to certain points. These are the lumbar (origin of the nerve); the posterior sacro-iliac, roots of the sacral plexus; the trochanterian, issue of the nerve between the trochanter and the ischium; the popliteal; the patellar, terminations of the external popliteal branches; the peroneal, situated at the head of the peroneal group; the internal malleolar, internal popliteal and posterior tibial nerves; and the external malleolar, saphenous nerve. Of these points the most important are the trochanterian, the popliteal, and the malleolar. In some cases there are also pain centres in the ankle, especially the inner side, and in the dorsum of the foot. At the painful points there is usually marked tenderness. Spasms of the muscles may be severe; they often attend violent paroxysms of pain, or may be produced by attempts at movement: in rare cases they are clonic and so frequent as to constitute a tic. Herpetic and other acute trophic disturbances are unusual, but in protracted cases the muscles undergo atrophy with contractures. Debove has called attention to polyuria as a symptom of sciatica: how far this excess of secretion is due, as he believes, to a general increase of the blood-pressure, and how far it is neurotic, is not certain. Every degree of sciatica in regard to severity and chronicity occurs.

In 1888 Charcot called attention to *scoliosis* as a secondary result of a long-continued sciatica; usually the concavity of the bend is opposite the sciatica, and has evidently been produced by the natural tendency of the body to bend towards the supporting leg in standing upon one leg. In rare cases the bend is towards the sciatica; it is possible that spasm from involvement of the sacral nerves may in some cases be the cause of this, but such a scoliosis would be produced by habitually standing with the painful leg half crossed, its foot resting upon the other.

DIAGNOSIS.—Usually its unilateral character separates the pain of sciatica from spinal pains; but even in double sciatica the tenderness and pain on movement render the diagnosis easy.

PROGNOSIS.—The prognosis in rheumatic sciatica is usually favorable; but in old cases it should be guarded, especially if there be trophic changes.

TREATMENT.—In the treatment of sciatica the first attempt should be to remove the cause. In rheumatic cases the ordinary hygienic treatment of the rheumatic diathesis should be practised: the salicylates should be used in very large doses, and may be followed by potassium iodide, which is, however, chiefly valuable when there is a syphilitic basis. In the immediate treatment of the sciatica, if the symptoms are severe, absolute rest is of the greatest importance: not only should the patient be put to bed, but the limbs should be placed between salt-bags or in an anterior splint in such a way as to secure immobility and yet allow local treatment. Even in old chronic cases entire rest for six weeks often does great good. For the combating of the pain hypodermic injections of morphine with atropine may be practised in the immediate neighborhood of the nerve, and when brought in contact with the nerve-trunk often seem to exert more than a simple analgesic influence. In some cases the hypodermic injection of cocaine or of antipyrin upon the nerve brings relief. Even hypodermics of simple water are sometimes efficient.

In the beginning of the treatment the patient should be thoroughly purged, and the bowels throughout should be kept loose, as a loaded rectum and congestion of the pelvic circulation may aggravate a sciatica. In the acute disorder leeching is often of service, and the free, persistent use of blisters is in all forms of sciatica of the greatest importance. When there is febrile reaction the combination of aconite with morphine is often useful. When the symptoms are very acute even the lightest galvanic current often increases the pain; but when they have in a measure been subdued the continued current should be tried. (See Peripheral Neuritis.)

Great good may sometimes be achieved by passing a stream of water as hot as can be borne by the patient for a length of time down the back of the limb. In the more obstinate cases of the disease counter-irritation by means of the hot iron should be practised; and massage and electricity may be of the greatest service. Whilst the nerve is very tender massage may readily do harm. Acupuncture has been practised with asserted great relief. Usually a chronic sciatica is a form of chronic rheumatism, and as such is to be combated by sulphur baths, pine-needle baths, steam baths, etc. Oil of turpentine has a very old reputation in the treatment of the chronic disease.

Congelation of the skin over the painful points by methyl chloride has been strongly recommended by Debove. Stretching of the nerve is justifiable only in cases of the greatest obstinacy; in the majority of instances it fails, but brilliant results are occasional. Even the mild stretching obtained by forcible flexion of the thigh upon the body sometimes seems to do good.

NEURITIS OF THE FACIAL NERVE. BELL'S PALSY.

For anatomical reasons, the facial nerve is extremely liable to sudden inflammation from exposure to cold, or from involvement in otitis or other contiguous disease, and frequently suffers complete loss of function by the pressure of exudation within the long bony canal.

SYMPTOMATOLOGY.—As the facial is a purely motor nerve, there is no pain accompanying the inflammation. The onset is usually sudden.

In the course of a few hours the paralysis is complete, or nearly so. The face is strongly drawn towards the opposite side. The power of completely closing the eye is lost, because the orbicular muscle is not able to raise the lower lid. The wrinkles in the forehead and the various folds of the skin, to which the face owes so much of its expression, entirely disappear or are greatly flattened out. The saliva is with difficulty retained. Articulation is distinctly impaired. Mastication is not interfered with, except by the difficulty in retaining the food between the teeth caused by the loss of power in the buccinator muscle.

Bilateral facial paralysis, *facial diplegia*, or simultaneous palsy of both facial nerves, is exceedingly rare, though it is sometimes produced by a long transverse lesion crossing the anterior half of the pons, or by a transverse lesion encroaching upon the facial nerves after their emergence. It is characterized by a fixed, immovable, expressionless countenance, a peculiar dropping of the angles of the mouth and collapsed appearance of the nostrils during inspiration, and a marked flapping in and out of the cheeks during respiration. The voice is usually nasal, and the articulation is very bad, owing to an impossibility of pronouncing labial consonants. There is excessive difficulty in retaining the food between the teeth, and the saliva in the mouth.

There are three distinct positions at which lesions of the facial nerve-trunk may occur and produce characteristic symptoms. The first and most frequent is that in which the point of pressure is near the point of escape from the temporal bone. Under these circumstances the paralysis is limited to the muscles of expression.

The second form of facial palsy is that in which the lesion is situated above the origin of the chorda tympani nerve, but on the distal side of the petrosal nerve. Under these circumstances, to the paralysis of expression is added great diminution of the sense of taste in the anterior two-thirds of the tongue.

In the third variety a lesion behind the ganglionic enlargement which gives origin to the third petrosal nerve causes loss of power in the muscles of expression, loss of taste. paralysis of the soft palate. as revealed by a depression of the arch of the palate upon the affected side, and a loss of power in the tensor palati muscle, so that the soft palate is drawn towards the normal side. At the same time the sense of hearing is generally abnormally acute, and the secretions of the parotid and submaxillary gland are deficient.

Diagnosis.—The only difficulty in a case of peripheral facial palsy is to decide as to its cause. Paralysis from exposure usually involves only that portion of the nerve which is external to the bony canal, though the inflammation may extend backward into the canal. Complete peripheral palsy of the whole nerve is in the great majority of cases due to disease of the bone, or to tubercular or syphilitic basal meningitis.

Prognosis.—A facial neuritis can usually be cured if seen early, unless it be due to disease of the bone or other incurable affection. When the muscles fail entirely to respond to any form of electricity the prognosis should be guarded, and if the condition has lasted one month without marked improvement recovery is improbable.

TREATMENT.—Leeching may be practised within the first twelve hours of the onset of the disease. Afterwards small, repeated blisters should be applied to the back of the ear. Salicylates may also be given at first, and afterwards small doses of the iodides. Electrical treatment of the affected muscles should be begun very early, although at first it should be mild, for fear of irritating the nerve-trunk. That current should be selected which produces the greatest contraction of the muscle with the least pain to the patient.

INFLAMMATION OF THE TRIGEMINAL NERVE.

ETIOLOGY.—Acute trigeminal neuritis is commonly the result of exposure to cold. Chronic or persistent neuritis may be due to propagation from an inflamed tooth-pulp ; to pressure upon peripheral nerve-filaments by alveolar thickenings, especially in old people ; to syphilitic, cancerous, or other tumors ; to carious bones ; and to various obscure causes.

SYMPTOMATOLOGY.—The chief symptom in trigeminal neuritis is pain, which varies in seat and intensity according to the degree and amount of nerve involvement. In severe cases the suffering is intolerable ; pains of the most furious character follow one another along the course of the nerves in incessant flashes for a few seconds or minutes and then abruptly cease ; the pains may be accompanied by clonic and tonic contractions of the muscles of the side of the face (*tic-douloureux*). In some cases paroxysms occur only a few times a day, but more frequently they repeat themselves at short intervals. The lower jaw and cheek are probably the most frequent seats of the pain ; somewhat more rarely are the branches of the upper maxillary and even of the ophthalmic nerves affected. Only in exceptional cases do the mucous membranes suffer ; but frightful burning, shooting, stinging, darting pains may be felt in the mouth, and become excessively severe as they run through the tongue. Often, impelled by an irresistible impulse to do something, or perhaps led by a slight feeling of relief, the patient, during the paroxysms, incessantly rubs the affected part with the hand, either alone or with a handkerchief. Tenderness is usually pronounced. It may be exquisite, a touch or a draught of cold air serving to bring on violent pain. The hyperæsthesia may affect the mucous membrane or the skin, but may be chiefly manifested at certain points to which attention was first called by Valleix.

In the ophthalmic branch, the most important point is the supra-orbital foramen. Less commonly to be recognized are the palpebral points of the upper eyelid, the nasal on the nose, where the ethmoidal nerve emerges from the nasal cartilage, the inner angle of the eye, corresponding to the supra-trochlear nerve, and the parietal prominence. In the superior maxillary branch, the most important point is over the infra-orbital foramen ; next in order is that in the upper lip, then the points in the gums or in the alveolar processes of the upper jaw. In the inferior maxillary branch, the point on the chin is the most frequent, next is one in front of the ear, while very inconstant and rarer are points on the lower lip, on the side of the tongue, and on the alveolar processes of the lower jaw.

DIAGNOSIS.—The pain of trigeminal neuritis cannot be distinguished from that due to other causes than inflammation. Reflex trigeminal pains of a severe type, though usually less severe than those of neuritis, have been recorded as being produced by injury to distant nerves, by various forms of intestinal parasites, by hemorrhoids and other rectal diseases, and by irritation of the reproductive organs, especially in women. The diagnosis of such a case rests upon the recognition and removal of the irritation.

Trigeminal pains of a severe type are sometimes the result of a neuralgic temperament, or at least exist without its being possible to discover any sufficient cause for their existence. The nature of such pains is to be recognized by their inconstancy, their shifting from one nerve to another, their presence in other nerves than the trigeminal, and the existence of a general neurotic temperament. In a large proportion of cases these so-called neuralgias are gouty. The most violent attacks of trigeminal pain may be of centric origin. In a case of H. C. Wood's they were the result of a cerebral hemorrhage. They frequently occur in elderly people, apparently as the result of changes in the nerve-centres due to interference with their nutrition by atheromatous vessels. In these cases the pain may be followed after a time by difficulty of swallowing or other symptoms which show that the motor as well as the sensory portions of the medulla are suffering.

It is especially in these centric cases that the condition known as *anæsthesia dolorosa* exists, in which affection there is loss of sensibility with violent prosopalgic pains. It is true that an anæsthesia dolorosa may arise from a tumor which presses upon the nerve and simultaneously produces an ascending neuritis and a loss of function in the part immediately involved in the pressure. Such cases are very rare, and their true character can be recognized only by discovering the tumor. It is very doubtful whether a complete anæsthesia dolorosa ever exists in simple trigeminal neuritis.

It may be laid down as a working rule that neuritis exists whenever there is distinct tenderness of the peripheral filaments of nerve or of Valleix points; whilst the absence of such tenderness is equally conclusive that the disease if organic must be centric, or else due to some morbid growth.

PROGNOSIS.—Acute trigeminal neuritis due to cold usually yields rapidly to treatment. Deep-seated chronic forms of the disorder are, however, very obstinate, and their prognosis is always serious unless some persistent cause can be found and removed. A large proportion of the cases cannot be relieved except by a surgical operation, the effects of which are also uncertain.

TREATMENT.—The treatment of trigeminal neuritis is in great part similar to that of inflammation of other nerves. (See page 605.) Certain specific remedies have, however, been recommended, especially croton chloral, to which, with doubtful correctness, has been attributed a special anæsthetic action over the trigemini. It may be given in ascending doses, commencing with five grains, three times a day. In our hands it has not given satisfaction. Amyl nitrite has been very highly recommended by Manzi and others, but the reports of the cases read like those of hysterical neuralgia: it may be given every two hours by inhalation in such doses as are necessary to produce flushing of the face and other physiological effects. Aconite and its alkaloid, aconitine, have been highly recommended by various authorities: half a milligramme of the crystallized aconitine of Duquesnel, or of the pure amorphous aconitine, may be given three times a day, increased as high as six milligrammes if the patient can bear it. Great care, of course, must be taken, and the dose must be reduced so soon as lowering of the pulse or other physiological effect is apparent. In neuralgic cases large doses of quinine are often of great service. In gouty or syphilitic cases proper constitutional treatment usually brings relief. Freezing the face with methyl chloride is alleged to have cured some patients.

Surgical interference is very frequent in trigeminal neuritis. As a very large proportion of cases begin in diseases of the teeth or the alveolar processes, it is extremely important that the local disease of these parts be surgically attended to with care in the earliest stages of the disorder. Whenever the fixity of the pain and of the tenderness indicates that one branch of the nerve is chiefly or solely affected, surgical interference should be prompt to prevent extension of the inflammation. In no case should simple section of the nerve be relied upon; as long a piece as is practicable of the nerve should be destroyed by resection, by thermo-cautery, or by other means. In obscure cases, with wide-spread distribution of the pain, removal of the Gasserian ganglion, although accompanied with some risk to life, is certainly a proper operation.

Removal of the nerve almost invariably gives relief; in a few cases it fails altogether; frequently the pain returns after an interval of weeks or months of comfort, probably because there is a reunion of the nerve or the development of a neuritis above the point of section. When a trigeminal pain is due to disease of the nerve-centres, exsection is of no value, and, if the diagnosis has been clearly made out, is an unjustifiable operation.

NEURITIC MUSCULAR ATROPHY.

DEFINITION.—A disease characterized by a peculiar degeneration of the nerve-trunks, in many cases spreading to the spinal cord and producing a very slow wasting of the muscles, accompanied by the appearance of the reaction of degeneration.

SYNONYMES.—Peroneal type of muscular atrophy; Charcot-Marie type of muscular atrophy.

ETIOLOGY.—Neuritic muscular atrophy is a distinctly family disease, occurring very frequently in groups of cases in successive generations. In typical cases no cause can be assigned for the attack except heredity. But it has been alleged that the disease may be due to long-continued muscular strain, or to the effects of habitual pressure upon the affected part incurred in the daily occupation of the patient. Thus, in a man accustomed to carry heavy loads on his shoulders and neck the affection began in the shoulder and neck muscles. In a mason the muscles of the intra-metacarpal region and of the ball of the thumb of the left hand, with which he was accustomed to pick up stones, were the first affected. It is probable that these irregular cases are distinct from the hereditary typical disease, and represent really an accidental neuritis.

MORBID ANATOMY.—The characteristic lesion is a peculiar degeneration of the intra-muscular nerves, accompanied by the disappearance of their myelin and the hypertrophy of their sheaths. The individual muscle-fibres undergo atrophy, which is apparent before the loss of their striation; finally they completely disappear. The intra-muscular vessels have their coats extremely thickened. The peripheral nerve-trunks, both sensory and motor, are usually much altered. No unmistakable lesion of the gray matter of the spinal cord seems to have been detected, whilst Dejerine, Dubreuilh, and others found the gray substance intact. The posterior columns have been so uniformly degenerated that Marinesco asserts that the spinal lesion is primary and that the nerve-lesions are secondary.

SYMPTOMATOLOGY.—The muscular atrophy commences in the feet, usually in the extensors of the great toe, and afterwards involves the common extensors and the peronei muscles. The small muscles of the feet may be early affected, and, as the disease begins usually in early childhood, a form of club-foot is frequently produced. The evolution of the disease is very slow, and it may be years before changes occur in the upper extremities: these usually begin in the hand. In the thenar, hypothenar, and interossei muscles they are often symmetrical, and result in the production of the claw-hand. The muscles of the trunk and of the face are very rarely, if ever, attacked. Fibrillary contractions are pronounced in the atrophied muscles, as is also the reaction of degeneration. The knee-jerk is diminished or lost when the thighs are involved. Disorders of sensibility are frequently pronounced, but do not appear early. They consist of pains, especially of painful cramps, anæsthesias, and paræsthesias. In only one out of twenty-five cases studied by Bernhardt did pain appear before the fifteenth year of age and the sixth of the disease. In about one-third of the cases it came on between the ages of twenty and twenty-five. Distinct incöordination has been noted in various cases, and vaso-motor disturbances in the affected part, as shown by discoloration and coldness, are very common.

In rare cases neuritic atrophy begins in the hands, and cases have been described in which other portions of the body were primarily affected; but it is very doubtful whether such cases really represented the present disorder.

DIAGNOSIS.—Neuritic atrophy is distinguished from the true myopathies and from progressive muscular atrophy by the presence of the reaction of degeneration, of pain, and of other sensory disturbances, by the age of attack, and by its being a family disease. From muscular atrophies following parenchymatous neuritis it is distinguished by its being a family disease, by the age of attack, and by the peculiar method of its development. (See Etiology.) From syringomyelia it is at once distinguished by the absence of the peculiar disturbances of sensation and nutrition. The nature of those cases in which it has been alleged that the disease was produced by local cause (see Etiology) must, in the present state of our knowledge, be considered doubtful.

PROGNOSIS AND TREATMENT.—Recovery never occurs, and there is no known effective treatment.

NEUROMA.

DEFINITION.—Tumor involving a peripheral nerve.

ETIOLOGY.—In many cases neuromata are traceable to injuries: thus, the bulbous nerves of amputation are the neuromata that produce painful stumps. The fact that neuromata are sometimes multiple indicates that they may be due to a wide-spread vice of constitution. They may occur at any age and in either sex, although multiple neuromata are said to be very rare in women. Any nerves, either within or without the bony cavities, may be attacked.

MORBID ANATOMY.—Neuromata may be divided into true neuromata, which consist of an abnormal growth of nerve-fibres, constituting an irregular mass, and pseudo-neuromata, various heterologous growths, such as fibroma, sarcoma, etc. True neuromata were subdivided by Virchow into the myelinic, consisting of medullated nerve-fibres, and amyelinic, consisting of non-medullated nerve-fibres. Neuromata may vary in size from that of a small bird-shot to an inch in diameter. Plexiform neuromata consist of interlacing neuromatous nerve-cords. They may be of great extent, but occur almost exclusively as a congenital malformation.

SYMPTOMATOLOGY.—Neuromata may be latent; usually, however, they are accompanied by pain, which is referred to the distribution of the nerve, often very severe and not rarely paroxysmal. In most cases there are no motor symptoms, but the functions of the nerves may be so interfered with as to produce partial or complete paralysis of the tributary muscles. Reflex spasms in adjacent or distinct muscles are not rare, and violent epileptiform convulsions may mark the peripheral irritation. Marked tenderness upon pressure is a frequent but not an invariable symptom. Pressure above the tumor on the nerve-trunk sometimes alleviates pain. A peculiar variety of neuroma is that known as *tubercula dolorosa*, in which various parts of the skin are covered with small, subcutaneous, painful tumors, which may be made up in great part of nerve-fibres or be largely composed of adenomatous or other tissue. Neuromata may be latent and produce no pain and give rise to no tenderness, so that if deep-seated their presence may not be suspected.

DIAGNOSIS.—A superficial neuroma cannot be overlooked if it causes symptoms. A deep-seated neuroma has often been unrecognized for a long time. Even if there be no evident local swelling, a nerve should be exposed and examined whenever there is a fixed pain of indefinite continuance, not accompanied by the characteristic symptoms of a neuritis, not readily explained as the outcome of a local non-nervous disease, and not relieved by treatment.

TREATMENT.—The only treatment for a neuroma is excision, which is permanent in its influence, as the tumors have no tendency to return.

SYPHILIS OF THE NERVE.

Gummous infiltration of the nerve-trunk may be the result of contact with a gummous tumor outside of the nerve, or in very rare instances may primarily develop in the nerve. The blood-vessel walls are first infiltrated with minute cells, which soon force their way through the trabeculæ of the nerve, cause atrophy of the nerve-fibres, and finally destroy the arteries themselves. In its full development the syphiloma contains round cells, spindle-shaped cells, fibrous structure, and the débris of nerve-elements, all enclosed in the greatly distended nerve-sheath. The point at which the nerve is attacked is almost always inside of the vertebra or of the cranium, and almost uniformly there are other large neighboring gumma. As gummous disease of the nerve without implication of the nerve-centres is very rare, the disease is usually accompanied with complex symptoms. Complete abolition of the function of the nerve is very uncommon, so that trophic changes are extremely rare, and the loss of mobility or of sensibility is never complete. Spasm is sometimes present; pain is commonly the chief manifestation. The trigeminus nerve is, according to our experience, more frequently attacked than any other, the result being a specific tic-douloureux.

The prognosis is favorable if the case be seen early. The treatment should be actively antisyphilitic.

CHAPTER VII.

VASO-MOTOR AND TROPHIC DISEASES.

RAYNAUD'S DISEASE.

DEFINITION.—A disease which in its acute form is composed of three stages, local syncope, local asphyxia, and local or symmetrical gangrene, and which in its chronic form consists of repeated paroxysms of one or more of these stages.

ETIOLOGY.—Acute Raynaud's disease, or *symmetrical gangrene*, occurs chiefly in young neuropathic subjects, and has been noted as a complication of organic spinal diseases. It appears sometimes to be produced by violent emotional strain, especially by fright, but usually has no apparent exciting cause.

Chronic Raynaud's disease occurs also in neuropathic subjects, and has been especially noted in alcoholics and in opium-eaters. The individual paroxysms are often provoked by an exposure, either locally or generally, to cold,—in some individuals being confined to the winter months. Emotional excitement may produce an attack.

MORBID ANATOMY.—The basal pathology of Raynaud's disease is entirely obscure. The peripheral neuritis found by Pitres, and the endarteritis and endophlebitis noted by Von Dehio, were probably secondary lesions. The disorder has occurred so frequently in locomotor ataxia and syringomyelia as to indicate a spinal origin of the attacks. It is probable that the local syncope depends upon an obliterating spasm of the arterioles, and that the local asphyxia is the result of a complete vaso-motor paralysis. Violent spasms have been noted in the retinal artery during an attack.

The clinical differences between the chronic and the acute form of Raynaud's disease are in individual cases so great as to cause hesitation in considering them as one disease; nevertheless, separation of the two affections would seem impossible in the face of the facts that every variety of paroxysm between the slightest "dead finger" or local syncope and the most acute gangrene exists, and that at any time, even after a lapse of years, the chronic form of the disease may end in acute gangrene.

ACUTE RAYNAUD'S DISEASE.

SYMPTOMATOLOGY.—The first stage of acute Raynaud's disease (*local syncope*) is sudden and usually painless in its onset. The skin of the affected part becomes of a dead-white color, sometimes even a little yellowish, and appears entirely devoid of blood. Cutaneous sensibility is lessened or altogether destroyed. Even when the sensation of contact is entirely lost the power of distinguishing heat and cold may be retained. The temperature of the parts is very notably diminished; the power of movement is lost. This condition may last for only a few hours, or as long as a month. When the second stage (*local asphyxia*) is reached the white color gives way to a cyanotic tint, which deepens to violet, and in some cases to black. Pressure on the parts now produces whiteness, followed by instant return of color on removal of the pressure, showing that the discoloration is owing to blood still inside of the capillaries. The parts are at this time swollen and much below the normal temperature. There is excessive burning pain, which may begin even before the congestion. The pain is more or less paroxysmal, occurring, it may be, in agonizing crises, which usually pass off with an abundant emission of urine. After some hours or days gangrene (*local gangrene*), sometimes but not always preceded by blebs containing bloody serum, rapidly develops. Sloughing may be complete in ten days from the beginning of the attack, but usually a longer time is required.

During the whole process the bodily temperature is not altered, but even in the beginning failure of appetite, vomiting, and other abdominal disturbances are very common, whilst a peculiar apathy with irritability is almost characteristic. Transient loss of consciousness and epileptic seizures have been recorded. A wide-spread acute synovitis, with great swelling of the affected joints and the parts surrounding them, bleeding from the nose, hæmoglobinuria, amblyopia, tinnitus aurium, partial deafness, anomalies of taste, irregularities of the pupil, aphasia, have all been noted, apparently results of disturbances of the circulation similar to but less pronounced than those occurring in the diseased parts. The gangrene is almost universally more or less symmetrical. Its especial seat is the ends of the fingers and of the toes, but the tip of the nose, the ears, the buttocks, or the muscles of the back may be affected.

DIAGNOSIS.—The only disease with which acute Raynaud's disease could be confounded is senile gangrene, from which it is distinguished by its occurring in young subjects, by its being symmetrical, and by the distinctness of the three stages of local syncope, local asphyxia, and gangrene. Difficulty may sometimes arise in deciding whether a case should be looked upon as the chronic or the acute form of the disorder. Usually the acute disorder can be recognized by the intensity and persistency of the symptoms, but sometimes it may be necessary to wait for the appearance of gangrene before giving a positive opinion.

PROGNOSIS.—Loss of tissue by gangrene seems to be invariable, but the amount of destruction is generally much less than would appear unavoidable in the beginning. In most cases there is only a single attack of local gangrene; but there may be repeated recurrences.

TREATMENT.—There is no known specific medication in acute Raynaud's disease. Tonics, analgesics, laxatives, bismuth subnitrate, and other appropriate remedies may be used to combat symptoms as they arise. The affected part should be protected by wrapping, preferably in carded wool, and prolonged immersion in hot water may relieve pain. Galvanism has been recommended by Barlow, who applies it by immersing the part in a basin of warm salt water in which the negative electrode has been put, whilst the positive electrode is applied over the spine. It sometimes relieves pain. It is especially important to avoid local irritation; but, when the slough has formed, carbolated cosmoline or other mild antiseptic dressing may be used.

CHRONIC RAYNAUD'S DISEASE.

Chronic Raynaud's disease consists of a series of paroxysms simulating those of the acute disease, but not ending in gangrene. There is usually a stage of local syncope, followed by one of local asphyxia, but the local asphyxia may occur without being preceded by local syncope. It was to cases of this character that Weir Mitchell especially gave the name of *erythromelalgia.* On the other hand, cases are not rare in which the local syncope may recur a great number of times without local asphyxia.

During an attack of simple local syncope the parts are very white, shrunken, stiff, but usually free from pain and tenderness, except as the syncope is passing off, when the pain may be severe, even when there is no pronounced asphyxia. We have seen attacks of local syncope of the fingers accompanied by or sometimes replaced by violent hemicrania. Apoplectiform attacks, epilepsy, acute mania, amblyopia, and aphasia, with temporary hemiplegia, have been similarly noted.

The disorder may persist for many years, causing much suffering, but having no effect upon life, and producing no loss of tissue; or local symmetrical gangrene may occur at any time, either affecting the parts deeply or else simply giving rise to blebs, ulceration, loss of nails, etc. Under appropriate treatment the disease may entirely disappear. The treatment must be primarily directed to building up the strength and health of the patient. Complete change of the habits of life, out-door exercise, nutritious, non-stimulating food, and other measures such as are used to combat neurasthenia, are essential. Antipyrin and allied drugs may be tried. When relations between cold and the attacks can be traced, absolute avoidance of exposure is essential, and change of climate may be very beneficial. If the disease occur in a narcotic devotee, the habit must be abandoned.

PERFORATING ULCER.

Definition.—A peculiar rapid ulceration, usually developed in the foot without obvious cause.

Etiology.—Perforating ulcer occurs with comparative frequency in locomotor ataxia, has been noted not infrequently in neuritis or after section of the sciatic nerve (Morat), and is probably always of trophic origin. In locomotor ataxia it may be one of the first indications of the disease.

Symptomatology.—Usually, but not always, after a severe local pain, a small hemorrhagic or ecchymotic spot appears under the epidermis of the foot; in the course of a few hours the skin detaches itself, or more frequently it becomes excessively thickened into a large, dry, corn-like mass; a small slough soon separates, leaving the ulceration round, with sharp, acute edges, piercing, it may be, only through the skin, but usually to the deeper tissues, and even to the joint or bone. In most cases the bone becomes seriously diseased, when the lesion appears as a small aperture leading by a narrow sinus to diseased bone and surrounded by thickened superimposed layers of epidermis. The surface of the spot is usually cold and anæsthetic, the characteristic feature of the ulcer being its insensibility to irritants and its freedom from pain during rest. Walking may cause suffering; and the fulgurant pains of locomotor ataxia are very frequently present, but do not have their origin or focus in the ulcer. Erysipelatous inflammation or erythematous exudations are apt to occur, and may terminate in death. Except in the rare cases in which the ulcer heals early, the bones of the foot, and indeed all the tissues of the foot, are finally affected. The small joints frequently become inflamed and eventually anchylosed, or undergo ulceration and destruction, resulting in luxations and deformities. The nails of the foot usually become brownish, dry, greatly thickened, curved, and furrowed. In some cases there is a marked increase in the growth of the hair and in the pigmentation of the leg, and the whole foot may be bathed in a peculiarly fetid sweat.

Prognosis.—The prognosis is very unfavorable in proportion to the severity and immutability of the organic nervous disease producing the perforating ulcer. Rarely the patient may escape with exfoliation of the bone.

Treatment.—The treatment consists especially in the use of absolute rest, high feeding, massage, alcohol, etc., to build up the strength of the patient. (See surgical text-books.)

ANGIONEUROTIC ŒDEMA. GIANT URTICARIA.

Definition.—A circumscribed œdematous swelling of the skin, apparently of purely neurotic origin.

Etiology.—Angioneurotic œdema occurs in neurotic individuals, the individual attacks being provoked by severe exposure, indigestion, great mental excitement, and other transient causes. In some cases it appears to be an hereditary family disease.

Morbid Anatomy.—This affection seems to be a purely functional neurosis. As we have seen this disease alternate with attacks of urticaria, and even grade into urticaria, the two affections are very closely allied, if indeed they are not one disease.

Symptomatology.—The characteristic symptom is circumscribed œdematous swelling, which may involve both the skin and the mucous membranes, and gives rise to rounded elevations having a diameter of from two to eight inches, pale or sometimes deep red in color, coming and going, often, in the course of a few hours. In typical cases there is no itching or pain other than a slight burning; but there may be intense itching, as in urticaria; again, the wheals may at first be very small, as in true urticaria, but soon spread over a wide area. Not rarely they are symmetrical upon the two sides of the body. The affection is usually not serious, but at least one case has been noted with paroxysmal hæmoglobinuria, and death is said to have been produced by œdematous swelling of the glottis. In some cases there is marked gastro-intestinal irritation with the attack.

PROGNOSIS.—An obstinate affection, usually recurring for many years, sometimes reappearing month after month at the menstrual period.

TREATMENT.—The most important part of the treatment is that of the underlying bodily condition. The digestion should be especially attended to. Large doses of extract of ergot at times seem to do good; antipyrin, phenacetin, and similar compounds we have seen act happily; quinine in full doses has been especially recommended by Oppenheim.

Intermittent Dropsy of the Joints is a rare disease, in which at intervals of from two days to several weeks a joint, usually the knee, suddenly swells enormously, remaining pale, cool, and free from pain, and recovering the normal state in from three to eight days. The treatment is that of the neurasthenia or hysteria which always underlies the disease.

Acro-Paræsthesia, or *Night Palsy*, is a disorder especially met with in women about the climacteric, in which there are persistent, often very disagreeable, paræsthesiæ in the hands, and especially in the points of the fingers; most pronounced at night, and in the morning when the subject wakes; sometimes, but not usually, attended with pallor of the fingers. The affection continues indefinitely, but is chiefly important from the fact that its subjects often look upon it with great alarm as the precursor of paralysis. It has, however, no such significance, nor is it connected with weakness of the heart.

SCLERODERMA.

DEFINITION.—A disease characterized by a cirrhotic hardening of the skin and subcutaneous tissues, followed by atrophy.

ETIOLOGY.—Scleroderma is not hereditary. It occurs in every race and at all ages, about seventy per cent. of the cases being in women. It has been frequently attributed to exposure to wet and cold, and the local affection is said to have followed a prolonged immersion of the hands in cold water. Injuries of the surface, the irritation of mustard plasters or blisters, and infectious fevers are also alleged causes.

MORBID ANATOMY.—The lesion of scleroderma consists of a marked increase of the elastic and cellular tissues of the skin and other parts, with an infiltration with leukocytes and embryonal elements along the lines of the blood-vessels. According to Unna, the lymph-vessels are narrowed and the lymph-spaces outside of the vessels are increased. When the bones are involved, leukocytes appear in the periosteum and even in the osseous tissue. Pigmentation of the skin is frequent, and broad, homogeneous bands in the corium are said to be a characteristic alteration. The blood-vessels everywhere have their walls strongly thickened and finally undergo obliteration. The infiltration and hardening are followed by atrophy. No constant or peculiar changes have been found in the nerves.

SYMPTOMATOLOGY.—Scleroderma may be ushered in by distinct prodromes, such as fever, pains, paræsthesiæ, malaise, and a special feeling of weakness, accompanied by local cyanosis or diffused redness, or redness in spots, with or without swelling of the skin. The discolored skin soon becomes œdematous and often erysipelatoid. The œdema frequently disappears during the night, to reappear in the day, or it may persist steadily. The first or œdematous stage of the disorder may remain without change for two or even more years, and it is affirmed may be recovered from; but in most cases the skin, sooner or later, gradually hardens so that the finger can no longer leave an impress.

During the second or indurated stage of the disease the surface of the body is hard, light-colored, furrowed, and preserving the coarse and fine markings of the skin, or it may be entirely smooth. It may be dry or sweating, and often suffers from vaso-motor disturbances. It usually finally becomes excessively pigmented. Paræsthesiæ and neuralgic pains are very common. Sometimes there is great tenderness, but anæsthesia more or less complete has been noted in a large proportion of the cases.

The hardening may persist for many years, gradually involving the deeper tissues, and even the bones. The general health usually fails. Dyspepsia, albuminuria, and various nervous disturbances, such as loss of memory, hallucinations, insomnia, giddiness, headache, and epileptoid attacks, have been noticed. Finally, if the patient survive, the third stage, or that of atrophy of the parts, appears, ending in death from ulceration and exhaustion, or more commonly from some complicating disease (especially pneumonia). Owing to the hardening and subsequent atrophy, the face in scleroderma is expressionless, and chewing and other motions are often interfered with. The mucous membrane of the mouth, pharynx, and vagina may be attacked, and an extraordinary narrowing of the laryngeal entrance is occasionally a troublesome complication.

Scleroderma varies indefinitely in the duration and the comparative development of the different stages, and also in its distribution. In a collection of nine hundred cases, Lewin and Heller found that in sixteen per cent. the whole body was involved, in twenty-three per cent. the trunk, in twenty per cent. the head, in seventeen per cent. the lower extremities, and in thirty-two per cent. the upper extremities.

TREATMENT.—All forms of salves, local applications, baths, etc., have been used in scleroderma, apparently without definite result. The surgical extirpation of the affected portion of the skin has also been tried, without distinct advantage. No internal medication is of value. The general health of the patient should be sustained, and, as there is usually great susceptibility to cold, the effect of a warm climate may be beneficial.

Morphœa (*keloid* of Addison) is a local form of scleroderma in which the changes are not diffused, but occur in patches or bands of irregular shape, and of a dead or old ivory-white tint, often bordered with a narrow violet, lilac, or pink zone of dilated vessels. When the band is narrow it may make a distinct sulcus. Cases occur in which diffused scleroderma occupies one portion of the body with morphœa patches on another. The diffuse disease in some instances has appeared before, in others after, the development of the patches. The most common local form of scleroderma is that in which the finger is attacked (*sclerodactylia*), with consequent great thickening and subsequent atrophy, even to the entire disappearance of the phalanges.

Facial Hemiatrophy most frequently develops in childhood, but may occur at any age. It usually begins with pain, which is often severe, followed by progressive atrophy affecting the skin and the deep tissues of the face. The skin may be white or pigmented, evidently thinned, but still preserving its sensitiveness; the hairs fall out, the bones waste, even the teeth drop out, from destruction of the alveolar processes. The prognosis is unfavorable, no known treatment having any effect upon the disease. As the disease is strictly limited to one side of the face, the contrast between the two sides is often very marked. Facial hemiatrophy is thought by some to be a trophic neuritis, but is more probably a local form of scleroderma.

Allied to scleroderma are some of the skin atrophies, especially *xeroderma pigmentosum*, which begins usually in the second year of life with freckles, followed by telangiectases or nævi, these by atrophy of the skin, with superficial ulceration, and after some years warty tumors with free discharge and marked pain, and in the end death from exhaustion.

Sclerema neonatorum, a disease allied to scleroderma, but believed by most authorities to be distinct from it, occurs as a congenital affection. It is characterized by stiffness of the joints and jaws and by large areas of induration of the skin, which is tense and glossy, but does not pit on pressure. In most cases the disease spreads and a fatal result is soon reached. Recovery is said to occur in very rare instances.

Œdema neonatorum, a subcutaneous œdema with induration, occurring in the new-born, commences in the legs and rapidly spreads until almost the whole body is affected. In rare cases it begins in the hands. There are marked drowsiness, great failure of vital power, and usually collapse ending in a short time in death. It is to be diagnosed by the lividity of the skin, the pitting of the parts on pressure, and the freedom of the joints from stiffness.

ACROMEGALY.

Definition.—A chronic disease characterized by enlargement of the hands, feet, and face.

Etiology.—Acromegaly has been recorded as congenital, but usually it develops from the eighteenth to the twenty-fifth year, though it may come on at any time. No known causes can be assigned for it; any influence of heredity is very obscure.

Morbid Anatomy.—The nature of acromegaly is unknown. The enlargement of the bones is due to a true hypertrophy, which affects also the skin, the connective tissue, and the blood-vessels. In a number of cases the thyroid and the thymus gland have been found enlarged. The pituitary body also has been found hypertrophied; and Marie, who first described the disease, believes that the affection is a dystrophy, bearing the same relation to the pituitary body that myxœdema bears to the thyroid gland. So many cases, however, of acromegaly without disease of the pituitary body, and so many cases of disease of the pituitary body without acromegaly, have been recorded, that the connection between the two is extremely problematic.

Symptomatology.—The first change in acromegaly begins in the ends of the fingers and toes, the tip of the nose, the lips, and the chin. The swelling primarily affects the bony tissues, but soon invades all the tissues of the part, and finally spreads until it involves not only the whole face and the ankles and wrists, but also the clavicles, the sternum, the patellæ, and even the vertebræ. The resulting appearance is very characteristic: the large feet and hands, the magnified face with its bones enormously thickened and lengthened, give an aspect that can scarcely be mistaken. The great toe and the ends of the fingers are apt to be especially out of proportion to the rest of the body; the nails become broad and vertically grooved; the lower jaw-bone may be so much affected as to project beyond the face, whilst the teeth are widely separated by the hypertrophy of the alveolar processes. Very late in the disease kyphosis, due to enlargement of the vertebræ, is often present.

Early in the disease some muscular feebleness, especially loss of endurance, is apparent, although fine movements may be executed; later awkwardness is a common symptom. A peculiar apathy, with sleepiness and dulness, is almost characteristic; the headache may be so severe and persistent as to cause the patient to wish for death. Disturbances of vision, diabetes, and various other symptoms have been noticed, evidently the outcome of disturbances of the basal brain-centres.

Diagnosis.—Acromegaly is distinguished from *osteitis deformans* of Paget by the facial rather than the cranial bones being attacked, and by the shafts of the long bones escaping. According to Marie, in Paget's disease the face is triangular, with the base upward, whilst in acromegaly it is ovoid or egg-shaped, with the large end downward. Under the name of pulmonary osteo-arthropathy Marie has reported a group of cases which resemble acromegaly, but which he considers to be separated from it by the facts that the disease is a complication of long-standing affections of the lungs, that the bones of the extremity, and especially their shafts, are early attacked, and that the phalanges are bulbous and enlarged, with curved nails, instead of being flattened as in acromegaly.

PROGNOSIS AND TREATMENT.—Acromegaly may persist for many years without change, but seems never to undergo cure. No known treatment is of avail.

SECTION III.

DISEASES OF THE CIRCULATORY APPARATUS.

CHAPTER I.

DISEASES OF THE PERICARDIUM.

PNEUMOPERICARDIUM.

THE presence of air or gas in the pericardial cavity is a rare condition. Air enters either through the perforation of a foreign body from the cutaneous surface, or from the œsophagus, or through the rupture of a pulmonary cavity. It may also enter in consequence of the advance of a cancer of the œsophagus, stomach, or intestine, or in the progress of a corrosive ulcer of the stomach. Gas may be formed in the pericardium from the admission of putrefactive bacteria. The presence of a considerable quantity of air or gas interferes with the action of the heart, resulting in dyspnœa, cyanosis, collapse, and death; lesser quantities rapidly cause inflammation. The area of cardiac dulness is replaced by a metallic tympany or cracked-pot sound, and a change in position causes tympany to be replaced by dulness. If fluid is likewise present, splashing may be heard. The valvular sounds have a metallic character, and are faint and often associated with a friction-sound. The physical signs thus resemble those of a circumscribed pneumothorax or large pulmonary cavity in the left chest near the heart. The dyspnœa, however, is not of such rapid onset in these affections, while the audible metallic sounds persist when the breath is held, and percussion makes apparent the loss of cardiac dulness. The cardiac sounds may present a metallic character when the stomach is distended with air or gas, which also may cause palpitation and dyspnœa. The recognition of the increased area of gastric tympany and the temporary nature of the disturbance are sufficient in the diagnosis. Although recovery from pneumopericardium sometimes occurs, the prognosis is generally grave, from the usual severity of the immediate causes. The treatment is that of a severe pericarditis, but is usually of no avail.

HÆMOPERICARDIUM.

Blood may be poured into the pericardial sac from injured vessels in the wall or from a traumatic perforation of the heart or the great vessels.

The same result follows rupture of the heart, or of an aneurism of the adjacent portion of the aorta or of the coronary artery. The blood may enter the pericardium slowly, as in wounds or rupture of the heart, and the patient live for some days although a considerable quantity of blood accumulates, or it may enter rapidly from an aneurism. A small hemorrhage produces temporary faintness, while slowly progressing hemorrhage causes pain in the region of the heart. Sudden profuse hemorrhage stops the action of the heart and occasions anæmia of the brain, associated with an increase in the area of cardiac dulness and a disappearance of the heart-sounds and the apex-beat. Hæmopericardium, except in the case of wounds which are not severe, is almost of necessity fatal.

The chief indication is for removal of the blood by the aspirator, a procedure which is said to have relieved the symptoms in some traumatic cases.

HYDROPERICARDIUM.

The normal pericardium contains from one to several drachms of serous fluid. When several ounces, perhaps two or three pints, are present, the condition is known as hydropericardium, or pericardial dropsy. Although the fluid is usually serous in character, in rare instances it is milky from the presence of chyle. Such an excess of fluid is usually considered to be the result of general or local mechanical causes, of venous or lymphatic stagnation, or of disturbances of the nutrition of the walls of the blood-vessels. The general disturbances of circulation are to be found in disease of the valves of the heart or of the myocardium, and in obstruction to the passage of blood through the lung. The local obstructing causes are various thoracic tumors situated near the base of the heart. Disease of the kidney, cancer, tuberculosis, and profound anæmias are the possible sources of cachectic hydropericardium.

A moderate quantity of fluid produces no symptoms; large quantities interfere with the action of the heart and cause a sense of constriction, dyspnœa, and cyanosis. The physical signs indicative of the presence of fluid are an increase of dulness in the cardiac area, extending beyond the audible or palpable position of the apex, and enfeebled although otherwise unaltered heart-sounds. Hydropericardium is distinguished from serous pericarditis by the absence of the fever and friction-sounds indicative of an inflammatory process, and by the existence of general or local causes of dropsy, in which the pericardial effusion is usually of late occurrence. The increased area of dulness may suggest enlargement of the heart, but in cardiac hypertrophy the apex-beat is to be appreciated at the outer border of dulness, and is not intensified when the patient leans forward. The diagnosis may be especially difficult in dilatation of the right ventricle, which also increases the area of pericardial dulness, and aspiration may be necessary to determine the presence of free fluid. Since hydropericardium usually represents a late stage in the progress of incurable diseases, the prognosis is that of the basal disease, and is unfavorable unless the cause can be removed. When the effusion is large, paracentesis may be practised.

PERICARDITIS.

Etiology.—Inflammation of the pericardium is usually regarded as due to an infection either from unknown sources or from recognized infectious processes. The former, the rarer variety, is called primary or idiopathic, while the latter is designated secondary pericarditis. It occurs in the course of a variety of diseases, in which the infectious agent is supposed to be transmitted to the pericardium by means of the blood-vessels. Among these are acute rheumatism, scarlet fever, variola, pneumonia, erysipelas, pyæmia and puerperal fever, and tuberculosis. These are all affections of which bacteria are regarded as the probable, if not the demonstrated, cause. Bacteria are also brought by means of the circulation from local lesions in more or less remote parts of the body, as from abscess or gangrene of the lungs, from visceral or peripheral abscesses, from cutaneous erysipelas, or from infectious periostitis or osteomyelitis. The pericarditis occurring in scarlatina probably often arises from the transfer of bacteria from a complicating tonsillitis, while the virus of acute rheumatic pericarditis enters through unknown channels of invasion even before any articular inflammation is apparent. Pericarditis also arises from the extension of a bacterial inflammatory process from neighboring parts, whether following the course of a wound of the pericardium or proceeding from an abscess of the heart or from a pneumonic lung. Bacteria are also admitted from the peritoneal cavity through the diaphragm or from perforating abscesses of the liver. The inflammation of the pericardium may be an extension from a neighboring cancer, as of the œsophagus or the stomach, from an aneurism of the aorta, from tubercular affections of the pleuræ or peribronchial lymph-glands, and from carious ribs, sternum, or vertebræ. The occurrence of pericarditis as a complication of chronic nephritis and of scurvy is attributable to a toxæmia occurring in these affections, or to the favorable opportunities which these diseases furnish for the growth of bacteria in the pericardium.

Pericarditis is more often found among males than among females, and is present at all periods of life, even in the fœtus and in extreme old age.

Morbid Anatomy.—According to the anatomical changes a distinction is drawn between acute fibrinous, sero-fibrinous, hemorrhagic, and purulent pericarditis, an acute or chronic tubercular pericarditis, and a chronic fibrous pericarditis. The anatomical appearances consist in the alterations of the pericardium, and in the variations in the characteristics of the exudation. Usually both parietal and visceral layers of the pericardium are diseased, although inflammatory changes may be limited to the one or the other layer. In acute pericarditis the pericardium is swollen, injected, and spots of punctate hemorrhage are to be seen. In the earliest stage the surface is smooth, without lustre, but soon becomes roughened and opaque, and eventually covered to a greater or less extent with fibrinous false membrane, and the subjacent superficial layers of the myocardium may be of an opaque gray color. In acute tubercular pericarditis miliary tubercles, at times in enormous numbers, lie within the layers of the pericardium or slightly project from its surface. The exudation is composed of fibrin, serum, and cells, the last being both leukocytes and red blood-corpuscles. Bacteria are also frequently found, and comprise streptococci, staphylococci, and pneumococci, as well as the bacilli of tuberculosis, the colon bacillus, and, according to H. C. Ernst, a variety of the bacillus pyocyaneus.

According as one or the other of these constituents predominates is the anatomical variety of pericarditis designated. In fibrinous pericarditis there may be so little serum present that the term *pericarditis sicca*, or dry pericarditis, is applied. The fibrin may form a delicate, easily detached layer either adherent near the base of the heart or diffused over the greater part of the pericardial surface. Although at the outset the fibrinous exudation is confined to a limited surface, the incessant motion of the heart rapidly causes it to appear over constantly increasing areas. The fibrin may be present as a thick layer, the surface of which is irregularly ribbed and elevated, forming a tripe-like membrane from which papilliform masses project, producing the appearance known as villous heart, *cor villosum*. The fibrinous adhesions also may extend from one pericardial surface to the other, perhaps enclosing spaces in which more or less serum is confined. In serous, more properly sero-fibrinous, pericarditis the serum is constantly associated with fibrin, and the liquid exudation varies in quantity from several ounces to a quart or more. The serum is opaque from the presence of fibrinous flocculi, cells, and granular material, and gravitates towards the lowermost portions of the pericardial cavity unless confined by adhesions. In purulent pericarditis the quantity of fibrin is less, the number of leukocytes greater, and considerable differences exist in individual cases in the proportion of pus-corpuscles to pus-serum. Usually a thin pus is present, and the quantity of fibrin is small. In hemorrhagic pericarditis the exudation, largely serous, contains red blood-corpuscles in greater or less number, but sufficient to produce a reddish discoloration of the liquid exudation, although usually not present so abundantly as to form blood-clots. A hemorrhagic exudation is commonly present in tubercular or cancerous pericarditis. In tubercular pericarditis there is a combination of miliary tubercles within the pericardium and a sero-fibrinous, purulent, or hemorrhagic exudation upon the surface. The tubercles may first become apparent after the fibrinous membrane is detached. In rare instances the bacilli of tuberculosis have been found in the purulent exudation of pericarditis although tubercles were absent, and have been regarded as the cause of the inflammation. In chronic tubercular pericarditis softened or calcified cheesy tubercles, inspissated exudation, and fibrous adhesions are combined.

In chronic fibrous pericarditis the pericardium is opaque, thickened, and indurated. The resulting changes may be localized or diffused, are present as a so-called milk-spot or tendinous patch, or produce an obliteration of the pericardial cavity from adhesions between the apposed surfaces. Chronic fibrous pericarditis is often the result of an acute pericarditis, the fibrous adhesions being replaced by fibrous bands. The fibrinous and cellular exudations are then disintegrated and encapsulated, and pockets filled with softened or calcified necrotic caseous material are seen between the parietal and the visceral pericardium. Mortar-like material or bone-like plates are to be found in the same region, and in extreme cases the heart may lie within a calcareous shell. The myocardium is usually in a condition of fatty degeneration or of brown atrophy, although the heart often is hypertrophied and dilated. In the severer varieties of acute pericarditis the inflammatory process may extend to the pleuræ or to the mediastinal tissues, and an acute pleurisy or mediastinitis, perhaps suppurative in purulent pericarditis and indurative in chronic fibrous pericarditis, occurs. A suppurative pericarditis may result in perforation through the skin or into the pleural cavity or lung, more rarely into the œsophagus.

SYMPTOMS.—In the consideration of the symptoms of pericarditis a distinction is to be drawn between acute pericarditis and chronic pericarditis, although at times the transition between the two is so gradual as not to be recognized. In acute pericarditis the symptoms may be so slight as to attract little or no attention, or there may be pain, perhaps aggravated on pressure, in the region of the heart, especially near the ensiform cartilage. It is either continuous or occasional, and may radiate from the region of the heart into the neck or towards the left arm. With increasing exudation the pain becomes diminished and there is a sense of constriction often associated with palpitation. The pulse is usually feeble, rapid, and irregular, although it may be unexpectedly strong and not accelerated. The breathing becomes quickened, is perhaps difficult, and headache, dizziness, sensations of faintness, and a feeling of anxiety, sometimes ending in despondency or delirium, may occur. During the further progress of the disease there is usually moderate fever, the temperature generally not exceeding 102° F. In certain cases of pericarditis the onset is violent, being announced by a severe chill, and there is hyperpyrexia, the temperature rising to 107° or 108° F., especially towards the end of life.

On physical examination at the beginning a sense of friction may be felt, but the sound of friction is to be heard either with the systole or with the diastole, sometimes with both, and in the latter case has a to-and-fro character not connected with the heart-sounds, and perhaps a gallop-rhythm. The friction-sound is furthermore characterized by inconstancy and variability, now disappearing to return in the course of a few hours and perhaps showing an altered relation to the heart-beat. It is best heard in the immediate vicinity of the sternum, but may be confined to the base of the heart or to the apex, or be audible throughout the cardiac area. The sound is often superficial, being louder in the upright position of the patient, and may be increased on pressure of the stethoscope. Although this sound is usually compared to the creaking of leather, it may have a blowing or musical character not differing from that of valvular murmurs, but is not connected with the valvular sounds, and generally presents the same intensity during systole and diastole. With the increase of the liquid exudation the intercostal spaces may be widened, and the region of the heart become abnormally prominent; the sound of friction disappears, the cardiac sounds become faint, and the apex-beat is indistinct and is perhaps not to be felt. Epigastric pulsation is often observed, attributable to transmission of the aortic beat through the liver enlarged by passive congestion. The area of cardiac dulness is increased at first near the base of the heart, but later forms a triangle, the base of which may lie even below the sixth rib and extend between the nipples, while the apex is rounded and may lie in the vicinity of the second costal cartilage. The apex-beat is sometimes to be recognized when the body is bent forward, and then is found between the sternum and the limit of dulness on the left. As the exudation increases, be it slowly or rapidly, the patient assumes a semiprone position, the expression is anxious, the breathing rapid, there is frequent cough, the skin is dusky, and the cervical veins are distended and pulsating. The radial pulse is feeble and sometimes paradoxical,—that is, more frequent during inspiration than during expiration. The voice may be husky from pressure on the left recurrent laryngeal nerve, and swallowing be difficult from pressure on the œsophagus. The lower lobe of the left lung is retracted to make room for the pericardial effusion, and a dull tympanitic note is to be heard at the left of the area of cardiac dulness, where there are increased vocal resonance and bronchial breathing. In some cases these signs may be heard also below the angle of the left scapula, in consequence of compression of the lung. They diminish or disappear when the patient bends forward, especially if he can assume the knee-elbow position.

The course of an acute pericarditis varies in accordance with the severity of the inflammatory process. In the milder cases, in which there is but little exudation, the discomfort ceases in the course of a few days and the temperature returns to the normal. In the more prolonged cases, in which there is abundant exudation, a favorable progress is indicated by a diminution of the area of dulness, which may occur rapidly, and a return of the friction-sound which had disappeared with the formation of the exudation. The presence of pus is to be suspected from repeated chills and wide variations in the course of the temperature.

DIAGNOSIS.—The increased area of pericardial dulness may be discriminated with difficulty from that due to dilatation. The beat of the dilated heart is more readily recognized, and lies at the outermost border of dulness, not changing its situation with an alteration in the position of the patient. The heart-sounds are louder, and the evidence of retraction of the left lower lobe of the lung is lacking. Rotch attaches especial importance in the differential diagnosis to the absence of resonance in the fifth right intercostal space in pericardial exudation. The dulness from an encapsulated pleuritic exudation is to be excluded by the respiratory sounds and the vocal fremitus being enfeebled and not exaggerated as in pericardial effusion. The signs of pleuritic friction may be mistaken for those of a pericardial rub, and may be caused by the transmitted beating of the heart although the breath is held. The inconstancy, variability, and wider diffusion of the pericardial sounds in the course of time enable the pleuritic friction to be excluded. A double friction-sound at the base of the heart may be mistaken for a double aortic murmur, but the latter is constant, is transmitted into the cervical arteries, and is associated with the characteristic water-hammer pulse and the systolic flushing of the capillaries. There are no means except aspiration by which the variations in the quality of the pericardial fluid can be absolutely determined.

PROGNOSIS.—The prognosis of acute pericarditis is usually favorable, although this affection may end in obliteration of the pericardium. When there is abundant exudation death may occur in the course of a few days or within a fortnight, either from the pressure upon the heart of the exudation or from an associated degeneration of the muscular fibre. Usually when the exudation is fibrinous or serofibrinous, as in the pericarditis of acute rheumatism or of pneumonia, recovery takes place; but when the exudation is purulent or hemorrhagic the prognosis is grave, since purulent pericarditis is often the result of pyæmia and may precede death but a few days, while hemorrhagic pericarditis is commonly caused by tuberculosis or cancer. Pericarditis occurring in chronic nephritis or in an alcoholic patient is of grave prognosis.

TREATMENT.—In the treatment of pericarditis it is essential that the patient be kept absolutely quiet in bed, with a total avoidance of any emotional or physical excitement which should increase the activity of the heart. In rare cases, when the patient is robust, from two to eight ounces of blood may be taken by leeches or cups, to be followed by a local application of cold by means of ice-bags or Leiter's tubes. If the heart's action be excessive and the pulse strong, aconite may be given in drop doses at varying intervals according to effect. It is essential to watch carefully the action of this drug, lest its influence be overexerted; and when in the advancing periods of the disease cardiac embarrassment occurs, aconite is capable of doing great harm; at such stages digitalis may be employed. What is said of the action of digitalis in myocarditis (page 647) is true also in pericarditis.

The appearance of a pericarditis in a rheumatic attack should usually be the signal for at least the temporary increase of the anti-rheumatic remedies, and in robust persons, when the effusion is serous and excessive, the old pill of calomel, digitalis, and squill, one grain each, may be tried.

When there is much pain, anxiety, restlessness, or insomnia, opium should be given in sufficient doses to control the symptoms. Sulphonal is probably a harmless hypnotic, though uncertain; trional may be given cautiously; chloral must be avoided. In the advanced stages of pericarditis, especially when there is much exudation, blisters should be used.

Paracentesis of the pericardium is indicated when the heart is embarrassed by a large serous exudation. Uncertainty as to the nature of the exudation often interferes greatly with the selection of suitable cases for the operation; but, as the operation is not a serious one, it may be used in all doubtful cases. Our own experience with it in rheumatic pericarditis has been that it has done neither good nor harm. In a collection of sixty cases of pericarditis of various character made by Roberts there were twenty-four recoveries after paracentesis. A small needle should be thrust directly backward in the fifth intercostal space, two inches to the left of the left edge of the sternum, so as to avoid the internal mammary artery. When the effusion is very large, some authorities prefer introducing the needle close to the costal margin in the left costo-xiphoid angle and pushing it upward and backward. Rotch asserts that aspiration should be practised at the fifth interspace to the right of the sternum. The fluid should be drawn slowly; if it prove to be purulent, an incision may be made, or even a resection of the rib; cautious irrigation of the pericardial sac sometimes works well, but is not free from danger, and is usually unnecessary.

Cardiac weakness in the advanced stages of pericarditis must be met by the use of digitalis and strophanthus, and occasionally by more rapidly acting diffusive stimulants, such as alcohol, camphor, or Hoffmann's anodyne.

Chronic Pericarditis.—Chronic pericarditis is either chronic from the outset or more frequently represents the persistence of an acute pericarditis, during the occurrence of which it is impossible to determine whether or not a chronic pericarditis is to result. Although an acute fibrinous or sero-fibrinous pericarditis may pursue a chronic course, tubercular pericarditis is the variety almost sure to become chronic in case the patient lives. In the chronic pericarditis which represents the outcome of an acute attack, the fever disappears, perhaps to return temporarily for short intervals, but the area of cardiac dulness remains enlarged, although gradually diminishing, and the result is an obliteration of the pericardium. The symptoms and signs of an *obliterative pericarditis* are therefore those of a chronic fibrous pericarditis. The anatomical changes have already been mentioned, and the nature of the degeneration of the myocardium stated. The symptoms are attributable to this degeneration, and consist in the evidences of a weakened myocardium,—namely, palpitation, dyspnœa, perhaps a sense of constriction in the region of the heart, and eventually cyanosis and dropsy. These general symptoms are slowly progressive and are aggravated by motion. The weakness of the heart is also indicated by a feeble apex-beat and pulse. The symptoms associated with chronic fibrous pericarditis are therefore not characteristic, and this lesion is generally discovered after death, having been unsuspected during life. The increased area of cardiac dulness is due to the enlargement of the heart, which is usually both hypertrophied and dilated. The sign to which especial importance is attached is a systolic retraction of the apex, frequently associated with which is a collapse of the cervical veins during diastole. According to Riess, the heart-sounds have a metallic character in obliteration of the pericardial sac when the stomach is dilated and tympanitic.

The treatment in chronic pericarditis consists of rest, avoidance of excitement, and the use of cardiac stimulants or sedatives *pro re nata*. In some cases repeated blistering is of service. When the pericardium is adherent, all that can be done is to meet symptoms as they arise.

CHAPTER II.

DISEASES OF THE HEART AND MYOCARDIUM.

MALFORMATION.

MALFORMATIONS of the heart are due either to arrest of development or to fœtal endocarditis or to both. When not inconsistent with life they are productive of a series of disturbances sometimes continued into adult age. The most important of these malformations are certain defects in the formation of the septa between the auricles and ventricles. An open foramen ovale is of frequent occurrence, but is of such slight clinical importance as to attract but little attention even when large, since, owing to the presence of the Eustachian valve, it seldom gives rise to symptoms. In rare instances there is little or no interauricular septum, and the heart practically contains but one auricle and two ventricles, and is then called a trilocular biventricular heart. More common is a defective formation of the interventricular septum, which when slight is manifested by an opening at the upper part of the septum beneath an aortic crescent. In some cases there is no septum, and the heart containing three cavities is called a trilocular biauricular heart. In the most extreme instances both the auricular and the ventricular septa are lacking and a bilocular heart is present.

The most common malformation is a stenosis of the pulmonary orifice, either from a muscular thickening of the conus arteriosus or from a fœtal endocarditis of the pulmonary valves. The presence of the latter causation is to be admitted only when sclerosis or vegetations are present and the rest of the heart is normal. Frequently the pulmonary stenosis is associated with a patent foramen ovale, a perforate ventricular septum, a persistent ductus arteriosus, and hypertrophy of the right ventricle.

Stenosis of the aortic orifice is rare. More common is an excess in the number of crescents, which may be four or five. Thickening and deformity of the tricuspid valve are occasionally to be found in the absence of other lesions of the heart, and are suggestive of a fœtal endocarditis. A fœtal endocarditis of the mitral valve is also possible, though generally considered as of rare occurrence. The frequent presence of abnormal tendinous threads, especially stretched across the left ventricle, although a congenital malformation, is of no symptomatic importance.

SYMPTOMS.—Malformation of the heart may produce no symptoms, the lesion being discovered after death. On the other hand, disturbance of circulation may become apparent soon after birth, usually increasing with the growth of the individual, and may be continued into adult life, even into old age. The symptoms often lessen with the growth of the individual, either from compensatory hypertrophy or from the more frequent avoidance in the adult of the exciting causes of undue activity of the heart. The most common symptom of congenital heart disease is cyanosis, a bluish discoloration of the skin, especially of the face and extremities, which is increased on exertion. This symptom may be associated with dyspnœa and cough, perhaps with dizziness and faintness. The skin is frequently cool, and the fingers and toes are clubbed. The cyanosis is regarded by some as a passive congestion of the cutaneous vessels, and by others as the result of an admixture of arterial and venous blood through an open foramen ovale, or of a defective aeration of the blood in consequence of narrowing of the pulmonary orifice.

DIAGNOSIS.—The diagnosis of congenital heart disease is based upon the occurrence of the above-mentioned symptoms, especially of cyanosis and the presence of a murmur. The nature of the malformation is rarely to be recognized by characteristic signs. An open foramen ovale causes no abnormal heart-sounds. A perforate ventricular septum is likely to cause hypertrophy of the right ventricle from overwork, and may produce a systolic murmur at the base of the heart, accompanied by a thrill, which is not continued into the aorta. Stenosis of the pulmonary orifice is indicated by a single or double murmur in the region of the pulmonary valve. A persistent ductus arteriosus also causes hypertrophy of the right ventricle, a systolic murmur, and a palpable thrill in the region of the pulmonic orifice; but the frequent association of these lesions makes it impossible to explain the physical signs by one or another malformation, since any one or all may produce the same result. The rare stenosis of the descending portion of the arch of the aorta. which is associated with an open ductus arteriosus, is manifested by hypertrophy of the left ventricle, dilatation of the arch of the aorta, and the visible enlargement of the anastomoses between the branches of the epigastric and internal mammary arteries. According to Hochsinger, loud cardiac murmurs heard in children are of probable congenital origin, whether with or without increase of dulness of the right side of the heart. They are also suggestive of a congenital origin if heard in the pulmonary area without accentuation of the second pulmonic sound. The presence of a palpable thrill suggests an open ductus arteriosus or a perforate septum.

ATROPHY.

The heart may be abnormally small as the result of a symmetrical arrest of development (*aplasia* or *hypoplasia*). Such a heart is usually associated with a thin, narrow, and elastic aorta, with small arteries, and sometimes with irregularities in the development of the genitals. The significance of this condition in chlorosis has already been mentioned. (See page 5.)

Atrophy of the heart, the result of acquired conditions, is to be found in elderly persons and in emaciated patients suffering from chronic wasting diseases, as cancer and tuberculosis: hence senile and marantic atrophies are to be recognized. The heart may also become atrophied in consequence of the long-continued pressure of abundant exudation or from chronic adhesive pericarditis. The heart is diminished in size, the pericardium wrinkled, the subpericardial fat greatly diminished in quantity, gelatinous and saffron-colored, while the muscular substance is of increased consistency and of a reddish-brown color from the presence of numerous pigment-granules in the vicinity of the nuclei. The atrophied heart is weak, but produces no characteristic disturbances.

TREATMENT.—There is no special treatment of cardiac malformations or of cardiac atrophy. The general therapeutic principles of chronic heart disease are to be applied to the individual case. (See page 668.)

HYPERTROPHY.

Hypertrophy of the heart is divided into simple hypertrophy, in which there is no dilatation of the cavities, and eccentric hypertrophy, in which dilatation of the cavities and hypertrophy of the wall are combined. A distinction is further drawn between primary or idiopathic hypertrophy, in which anatomical causes are not readily appreciated, and secondary hypertrophy, in which mechanical obstructions to the circulation are easily recognized.

ETIOLOGY.—The immediate cause of cardiac hypertrophy is an increased demand for the work of the heart, but that hypertrophy shall result it is essential that the patient be well nourished. A demand for increased work is produced by obstruction to the circulation either in the general arterial system, in the lungs, or in the heart itself. Obstruction in the general arterial system may be caused by aortic hypoplasia, aneurism, or arterio-sclerosis. The pulmonary circulation is obstructed in emphysema, in fibrous pneumonia, and, by compression of the lung, in chronic pleurisy or curvature of the spine. Valvular disease and obliterative pericarditis are cardiac causes of obstruction. Hypertrophy may also be produced by excessive action of the heart in the absence of any obstruction to the circulation. Such excessive action may be due to the occupation of the individual, and be either continuous or the result of temporary but extreme strain. It may likewise be occasioned by disturbance of the nervous system in consequence of mental excitement, or of the conditions occurring in Graves's disease. The hypertrophy is furthermore attributable to the effect of certain poisons upon the nervous system, as tea, coffee, tobacco, and alcohol. The hypertrophied heart in chronic interstitial nephritis is usually regarded as the result of increased tension of the arterioles dependent upon defective elimination of waste through the kidneys, and lesser degrees of hypertrophy are to be found in acute nephritis. The frequency of cardiac hypertrophy in consequence of excessive beer-drinking is a well-recognized fact.

MORBID ANATOMY.—The hypertrophied heart is increased both in size and in weight, the latter being of more value than the former as evidence of hypertrophy, since enlargement of the heart also results from dilatation of the cavities, in which case the thickness of the wall may be even less than normal. The weight of the normal heart is from eight to ten ounces, while the hypertrophied heart may weigh in the vicinity of three pounds, and the wall, which is red and resistant, may be three times as thick as normal. The papillæ and trabeculæ usually are enlarged, and the papillæ of the mitral valve may be nearly of the size of the thumb. The appearances of the hypertrophied heart vary somewhat according to the cause. If the obstruction is at the aortic orifice or in the general arterial system, the left ventricle becomes conspicuously enlarged and the heart elongated. If the obstruction is in the pulmonary circulation, the right ventricle is particularly hypertrophied and the heart is widened. When both ventricles are hypertrophied, dilatation is usually associated, and the heart, both elongated and widened, attains an enormous size, and is compared to that of cattle, *cor bovinum.*

SYMPTOMS.—Hypertrophy of the heart, if just sufficient to overcome the obstructing cause, produces but little disturbance. The patient may have a sense of fulness in the head, of ringing in the ears, or of weight in the epigastrium, and may complain of powerful beating of the heart. A tendency to nasal hemorrhage is not infrequent, and it is in such cases that the possibility of cerebral hemorrhage should be held in mind. The pulse is full, strong, and resistant, the apex-beat is visible and palpable over an abnormally large area, and pulsation of the carotids is unusually vigorous. The first sound of the heart is dull and prolonged, and the second sound at the base is accentuated. The accentuation is of the aortic second sound in predominant hypertrophy of the left ventricle, and of the pulmonic sound when the right ventricle is conspicuously hypertrophied. In the latter case the patient is more likely to complain of short breath, and the impulse, perhaps exaggerated, of the heart is to be recognized near the ensiform cartilage, but the radial pulse lacks the characteristics above mentioned. The area of cardiac dulness is elongated downward and outward from hypertrophy of the left ventricle, and in extreme cases of hypertrophy with dilatation may reach as far to the left as the nipple-line and below the sixth interspace. If the hypertrophy affects chiefly the right ventricle, the area of cardiac dulness is extended to the right beyond the edge of the sternum, and the increased area of cardiac dulness to the left of the sternum is chiefly due to the displacement caused by the enlarged right ventricle. In extreme cases of hypertrophy and dilatation the præcordial region is distended, and the beat of the heart is to be recognized over a wide area.

DIAGNOSIS.—The diagnosis of hypertrophy of the heart is easily made from the persistent powerful beat of the heart, the character of the pulse, and the accentuation of the second sounds at the base.

PROGNOSIS.—Since hypertrophy of the heart is to be regarded as a means of compensating for pathological conditions, its presence is to be regarded as fortunate. Its occurrence, therefore, is favorable while the nutrition of the body can be maintained and the amount of work done is in proportion to the strength of the individual. Insufficient nourishment or overwork and the persistence or aggravation of the exciting causes rapidly or gradually induce a failing compensation, the prognosis of which is uncertain.

TREATMENT.—See Treatment of Chronic Heart Disease, page 668.

DILATATION.

Dilatation of the heart is the result of weakness of the cardiac muscle. This weakness may be caused by a sudden, prolonged, or extreme strain, or it may be due to a degeneration of the myocardium occasioned by infectious diseases, by such poisons as arsenic and phosphorus, or by disease of the myocardium resulting from affections of the coronary vessels or from the extension of a pericarditis. The most frequent cause of dilatation of the heart is the failure of the hypertrophied myocardium to overcome the resistance which has been offered, this failure being the result of the persistence, perhaps of the progressive nature, of the causes, and the lack of sufficient rest and nutrition.

MORBID ANATOMY.—Simple dilatation of the heart is characterized by an increase in the size of the cavities and by a thinning of the walls. The heart is therefore enlarged, although the weight is not increased, unless, as is often the case, hypertrophy is associated, in which event the wall of the heart may be of normal or increased thickness. Since dilatation is often the result of degenerative changes in the myocardium, the consistency of the muscle is either diminished, the color being of a reddish gray or yellow when granular or fatty degeneration is present, or the density is increased and the muscle in considerable part replaced by fibrous tissue in the case of fibrous myocarditis. When hypertrophy is combined with dilatation, the latter being conspicuous, the papillæ are flattened and the endocardium is thickened and opaque, especially in the ventricle exposed to the greatest strain.

SYMPTOMS.—The symptoms of a dilated heart are essentially those of cardiac insufficiency, either gradually progressing from bad to worse or preceded by evidence of hypertrophy in case the dilatation is secondary to hypertrophy. Palpitation, pain referred to the region of the heart, dyspnœa, bronchial catarrh, perhaps pulmonary hemorrhage, vertigo, faintness, and wakefulness, partly in consequence of the tumultuous beating of the heart, are the immediate results of dilatation of the left ventricle. The pulse is soft, frequent, and irregular. The heart-sounds are sharper, though fainter than in the earlier stages of hypertrophy, and reduplication of the first sound is frequently to be heard. Eventually symptoms of insufficiency of the right ventricle predominate, and there is obstruction to the passage of blood through the lungs, which is shown by increased dyspnœa and frequent cough. Obstruction to the peripheral venous system follows, and is made evident by dropsy, enlargement of the liver, gastro-intestinal catarrh, and a concentrated albuminous urine.

Diagnosis.—The diagnosis of dilatation of the heart depends upon the association of increased cardiac dulness, feeble impulse, and gallop-rhythm. No considerable alteration of the area of cardiac dulness takes place unless dilatation is associated with hypertrophy. In case of dilatation and hypertrophy of the left ventricle, if the dilatation predominates the apex-beat is less vigorous and the accentuation of the aortic second sound is less pronounced than when hypertrophy is the more conspicuous. If dilatation of the right ventricle predominates over hypertrophy the physical signs are less in evidence than are the symptoms, although accentuation of the second pulmonic sound is less conspicuous than when hypertrophy exists. The increased area of dulness may be simulated by an encapsulated pleurisy in the immediate vicinity of the heart, but in this affection, while the area of dulness may change, the heart-sounds are not modified on alteration of the position, and the apex-beat is independent of the outer line of dulness. An increased area of dulness may also result from tumor or excessive fat in the mediastinum. In such cases there is likely to be no modification of the pulse, the heart-sounds, or the position of the apex.

Prognosis.—If the causes of dilatation of the heart are temporary, recovery may take place; if permanent, relief is afforded only by the occurrence of hypertrophy, which demands diminished work or increased nutrition, or both. The prognosis in the individual case, therefore, is determined largely by the results of treatment, and is in general the more favorable the younger the patient.

Treatment.—See Treatment of Chronic Heart Disease, page 668.

FATTY INFILTRATION.

Fatty infiltration of the heart is indicated by an increased accumulation of fat in the subpericardial, interstitial, and subendocardial fibrous tissue. These structures represent one of the physiological reservoirs of fat, and store this material under conditions which favor its accumulation in the other fat-reservoirs of the body: hence fatty infiltration of the heart is found in obese persons in whom, from an inherited tendency, sedentary habits, or an excessive fat-forming diet, or from a combination of these factors, fat is more rapidly absorbed than consumed. The degree of fatty infiltration varies extremely: the more abundant the subpericardial infiltration the more does the fat spread over the heart, following the distribution of the coronary vessels, and the more likely are the intermuscular tissue and the subendocardial tissue to become infiltrated. The muscular bundles may be separated by the fat, and in extreme cases atrophy of the muscular fibres occurs, although considerable degrees of fatty infiltration may exist without degenerative changes in the muscle. It is even possible that hypertrophy and dilatation of the heart may be combined with fatty infiltration.

SYMPTOMS.—Fatty infiltration of the heart to a considerable degree may exist without the production of symptoms; but, especially when, as happens in the majority of cases, it is combined with excessive general obesity, it may cause marked shortness of breath upon exertion, though the muscle-fibre has not undergone degeneration. Any extra work, mental or physical, thrown upon such a heart, or depression from intercurrent acute disease, may bring sharp manifestations of a weak heart, such as palpitation, præcordial distress, rapid breathing, vertigo, faintness, or cyanosis. With associated degeneration of the muscular fibre of the heart these attacks become more frequent, and are combined with those of fatty degeneration.

There are no characteristic signs of fatty infiltration, and the increased area of cardiac dulness is so slight as to be recognized with difficulty. The apex-beat is feeble, the valvular sounds are faint, and the pulse has no constant character, although it is usually regular and of moderate tension. The diagnosis is therefore based upon the occurrence of the symptoms of a weak heart in a fat person. Since excessive fat may be removed from other fat-reservoirs under appropriate treatment, the same result may occur in the heart, and the distressing symptoms of fatty infiltration may disappear with the loss of weight, not to return, provided a reaccumulation of fat does not take place. On the contrary, persistent fatty infiltration of the heart may prove a source of danger, as well as of discomfort, by favoring fatty degeneration of the muscular fibre, with its possibilities of sudden death from paralysis of the heart under even comparatively slight provocation.

TREATMENT.—The treatment of fatty infiltration of the heart is that of obesity. (See page 59.) With the reduction of the general corpulence improvement of the heart almost invariably takes place. When the cardiac symptoms are very severe, care is required, especially in the beginning of the treatment, to graduate the exercise. In these cases the plan of hill-climbing, as instituted by Oertl, is especially useful. A mountain-path is marked at regular distances, and the patient is required each day to walk so many yards farther than the day before, the amount of work done being steadily increased until it becomes very large. There is no reason for supposing that this form of exercise is superior to other forms, except in the certainty with which the amount of work can be regulated. Graduated exercise is of the greatest service in all cases of fatty infiltration, and should be carefully tried even when there is reason to fear that there have been some changes in the heart-fibres.

FATTY DEGENERATION.

Fatty degeneration of the heart is considered to be due mainly to impairment of nutrition or defective oxidation. Local causes may exist, as sclerosis of the coronary arteries, fibrous myocarditis, chronic pericarditis, and the conditions producing hypertrophy. General causes are also important, as poisoning by phosphorus, arsenic, and alcohol, and by the toxins of certain acute infectious diseases, especially diphtheria. Fatty degeneration of the heart occurs also from the impoverishment of the blood in profound or pernicious anæmia, and in cachexia, particularly that arising from cancer or pulmonary tuberculosis.

The entire heart may present a homogeneous opaque grayish-yellow color, with or without opaque yellow spots seated in the papillary muscles or in the wall of the heart, which between them is of a red color. The more extensive the distribution of the fatty changes the more diminished is the resistance of the flabby heart. If local causes of fatty degeneration exist, the appearances of fatty degeneration are to be found in the regions conspicuously affected, whether in one side of the heart, in a cavity, or in a portion of the wall.

The symptoms of fatty degeneration are those of cardiac insufficiency or weakness, and are usually indicative of a predominance of the fatty degeneration in the left half of the heart. At times the function of the right half of the heart is conspicuously affected, or, when the causes are general, the signs of weakness of both ventricles are present. Palpitation, rapid breathing, cardiac pain, perhaps attacks of angina, and a soft, irregular pulse of small volume, feeble heart-sounds, often reduplication of the first sound at the apex, occur in fatty degeneration of the left ventricle. There are also dizziness, faintness, ringing in the ears, and Cheyne-Stokes breathing. Fatty degeneration of the right side of the heart causes catarrhal conditions, dropsy, and cyanosis, although these effects are not often observed except when the fatty degeneration occurs in the sequence of hypertrophy and dilatation.

The diagnosis of fatty degeneration of the heart is based upon the occurrence of symptoms of cardiac weakness not relieved by treatment in connection with the above-mentioned causes. Arcus senilis and resistant or calcified radial arteries are not evidences of fatty degeneration of the heart.

The prognosis is always grave, since the lesion represents a terminal stage in the processes or conditions producing it. When fatty degeneration is circumscribed the fat may be absorbed and a scar remain. Extensive fatty degeneration results in death either suddenly from cardiac paralysis or more gradually from œdema of the lungs.

TREATMENT.—The treatment of fatty degeneration of the heart is in great part that of cardiac dilatation. (See page 670.) The method of Oertl (see page 643), which yields such good results in fatty infiltration, is very dangerous in fatty degeneration, and, if practised at all, should be restricted to the milder forms of the disease. For evident reasons, when profound structural change has occurred in the heart-muscle, digitalis and other drugs which act upon the muscle-fibres are almost or altogether powerless. Under such circumstances absolute rest affords the only relief for the patient.

MYOCARDITIS.

The occurrence of anatomical changes in the myocardium under various conditions has long been known, but the associated symptoms do not differ materially from those attributable to a weak heart from fatty infiltration or fatty degeneration. Of late years a special place in nosology is often assigned to myocarditis, and a primary or idiopathic variety is distinguished from the secondary variety occurring in the course of endocarditis or pericarditis. A further distinction of importance is that between acute and chronic myocarditis.

ETIOLOGY.—The most important cause of acute myocarditis is to be found in infectious diseases, and its symptoms may be expected in the course of diphtheria, scarlet fever, typhoid fever, small-pox, septicæmia, pyæmia, erysipelas, puerperal fever, influenza, and gonorrhœa. Chronic myocarditis occurs at any age, though usually after middle life, and is of especial frequency from excess in food and alcohol, from physical strain, and from arterio-sclerosis however caused. A chronic infectious myocarditis results from the invasion of the myocardium by the bacilli of tuberculosis, by the virus of syphilis, and by actinomyces. The lesions occurring in actinomycosis are so rare as not to require particular notice, while those present in tuberculosis and syphilis are especially mentioned in the general consideration of those subjects.

MORBID ANATOMY.—The anatomical changes are found both in the muscle-cells and in the fibrous tissue: hence a parenchymatous myocarditis is distinguished from an interstitial myocarditis, it being always acute, while interstitial myocarditis may be either acute or chronic. The latter distinction is one of convenience, since parenchymatous changes probably precede the development of fibrous tissue.

In acute parenchymatous myocarditis the muscle is opaque gray and brittle, the muscle-cells are in a state of granular, fatty, or hyaline degeneration, and the muscular fibres are frequently separated. This last condition is known as disassociation or fragmentation, and has been regarded as an important pathological state. It occurs under a variety of circumstances, not only in acute but also in chronic myocarditis, in sudden death from violence, in death from shock after operations, and in diseases of the brain. It is now generally considered that the fragmentation of the fibres takes place from irregular and powerful contractions of the heart during the death-agony. The characteristic appearances of acute interstitial myocarditis are to be found as minute opaque gray or yellow spots with a red margin, the miliary abscesses, which sometimes become confluent, and are readily seen beneath the pericardium and the endocardium. A dark red or reddish-yellow, soft, and friable portion of the heart-wall, generally in the vicinity of acutely diseased valves, is also indicative of an acute interstitial myocarditis, and represents the result of the extension of a septic inflammation from the valve to the heart-wall, being at times due to the friction of a valvular vegetation or thrombus.

Chronic myocarditis is manifested in the earlier stages by the presence of opaque white or yellow spots and patches, best seen on section of the wall of the left ventricle and of the interventricular septum. At a later period they are soft and depressed below the surface, the condition to which Ziegler has applied the term *myomalacia*. After the softened material has been absorbed, fibrous scars remain, which weaken the ventricular wall and favor a dilatation of the cavity. A portion of the ventricular wall may thus be transformed into a fibrous plate which serves as a source of aneurism of the heart, but sometimes becomes calcified and forms an unyielding plate.

SYMPTOMS.—The symptoms of myocarditis are those indicative of a weak heart,—namely, palpitation, præcordial discomfort, sometimes angina pectoris, vertigo, faintness, rapid breathing on slight exertion, disturbance of digestion, and cyanosis, perhaps eventually dropsy. The grouping and severity of these symptoms vary. In acute myocarditis, for instance, the rapid, often irregular and weak pulse and palpitation by some are regarded as due to the alterations of the myocardium, by others are considered as evidences of a disturbance of cardiac innervation. Such symptoms may occur and no lesions be found after death, or the typical lesions of acute myocarditis may be found in the absence of such symptoms. On the other hand, the severer symptoms so often present in chronic myocarditis are to be found in other lesions of the heart, and the anatomical changes of chronic myocarditis may occur even in persons dying suddenly yet without antecedent symptoms calling attention to disease of the heart. In general, the symptoms of chronic myocarditis are those of progressive weakening of the heart, of slow development, but suddenly becoming serious, if not fatal, under physical or mental excitement. The auscultatory signs attributable to an acute myocarditis are merely weakness and rapidity of the heart-sounds. In chronic endocarditis there is likely to be an increased area of cardiac dulness, especially to the left, and the apex-beat is feeble, but visible over a somewhat widened area. The heart-sounds are also feeble, especially the second aortic sound, while there is a tendency to reduplication of the first sound at the apex. Murmurs are absent unless the valves eventually become incompetent. The pulse is irregular, soft, and of small volume, usually quickened under slight nervous or physical excitement, though sometimes slow, and does not materially change in character under treatment.

DIAGNOSIS.—The diagnosis is based upon the association of symptoms of a weak heart with a rapid, irregular, feeble pulse independent of valvular disease and unaffected by treatment, and upon the progressive nature of the symptoms. The distinction between an acute and a chronic myocarditis depends upon the occurrence of the symptoms of a weak heart as the result of an infectious disease in a person previously well.

The discrimination between an acute interstitial and an acute parenchymatous myocarditis is not to be absolutely made, since a parenchymatous affection is always associated with the interstitial process. In favor of an acute interstitial myocarditis is the occurrence of chills in malignant endocarditis or in a septic thrombophlebitis, especially when evidences of minute arterial embolism are to be found in the skin or the retina. The diagnosis of acute parenchymatous myocarditis is safely to be made when the symptoms of a weakened heart are present in acute infectious diseases in which evidences of a localized suppurative or necrotic process likely to cause bacterial embolism are lacking. The symptoms of chronic myocarditis may be confounded with those of a cardiac neurosis, but their persistence, their independence of emotional excitement and of the abuse of tea, coffee, and alcohol, and the absence of digestive and pelvic disturbances, are important in differentiation. Although the symptoms resemble those of a fatty heart, fatty infiltration of the heart is excluded by the absence of excessive obesity. The distinction between fatty degeneration of the heart and chronic myocarditis is based upon the difference in etiology and upon the more protracted course of chronic myocarditis. A dilated and hypertrophied heart also presents similar symptoms, but the dilated hypertrophied heart is essentially in a condition of chronic myocarditis. The distinction, therefore, is based upon the etiology, the antecedent symptoms of hypertrophy, the presence of an increased area of dulness, and especially, for a time at least, the beneficial results of treatment.

PROGNOSIS.—The prognosis of acute myocarditis is essentially that of the disease in which the myocarditis occurs. It is therefore favorable in some cases, although sudden death from cardiac paralysis or rupture may occur. It is to be remembered, however, that the eventual progressive occurrence of symptoms of chronic myocarditis may be the result of a previous acute infectious disease. It is obviously impossible to determine the extent and degree of the acute myocarditis in acute infectious diseases terminating in recovery. Chronic myocarditis may exist for years, but is a progressive affection. Compensatory hypertrophy may last for a while, but eventually, as in valvular disease, cardiac insufficiency arises, either rapidly or slowly, with its final fatal result.

TREATMENT.—In the treatment of acute myocarditis it is essential to prescribe absolute rest, physical and mental, with a careful use, if there be evidences of heart-failure, of digitalis and strophanthus. We have no definite knowledge, however, of the influence of these remedies upon an acutely degenerating heart-tissue ; but we do know that when the cardiac process is severe the heart will not respond functionally either to digitalis or to strophanthus. It is evident, therefore, that the exhibition of large doses of cardiac stimulants in acute myocarditis is of very doubtful expediency, and that the attempt to force the heart to respond to these remedies is hazardous.

In chronic myocarditis we have no control over the process of degeneration, and the treatment must be limited to the application of the principles of chronic cardiac therapeusis. (See page 668; also Fatty Degeneration, page 644.)

THROMBOSIS.

Thrombi are of frequent occurrence in the heart as a result either of alteration of the surface or of obstruction to the circulation, hence are to be found in acute and chronic endocarditis, and in hearts enfeebled either by wasting diseases or by chronic myocarditis. Thrombi are present on the valves and in the recesses of the heart, especially in the auricular appendages and between the ventricular trabeculæ. They are often confounded with post-mortem clots, but are to be discriminated by their adherence (not entanglement among the trabeculæ), their brittleness, their lack of lustre, and their color, which is more gray or reddish gray than red. In shape they are more globular or flattened than elongated. Since they occur in an enfeebled heart, the symptoms of cardiac weakness are associated. There is no reason to suppose that the thrombi are a cause of the weakness, although formerly the cardiac polypus was regarded as a frequent cause of digestive as well as circulatory disturbances, and the condition was often erroneously diagnosticated. Cardiac thrombi are to be regarded as complications of the general and local diseases in which they occur, and not infrequently prove fatal by becoming the source of emboli, which may produce sudden death by obstructing the pulmonary or the coronary artery or a valvular orifice in the heart. In rare instances the effect of their presence is beneficial, as when they obliterate the cavity of a cardiac aneurism.

TREATMENT.—There is no known means of affecting cardiac emboli or thrombi; the treatment of cardiac thrombosis is therefore that of acute myocarditis or myocardial degeneration which follows the arrest of circulation: in coronary embolism the treatment is that of angina pectoris. When a large area of the heart is involved, no remedies are of any avail, save for the relief of pain. (See Angina Pectoris, page 684.)

ANEURISM OF THE HEART.

Localized dilatation of the ventricular wall weakened in chronic fibrous myocarditis is a condition which has been designated aneurism, or partial aneurism, of the heart. A distinction is to be drawn between this, the usual variety, and acute cardiac aneurism, which is the result of an acute interstitial myocarditis extended from an acute valvular endocarditis, and, according to Ponfick, caused by the friction of a diseased valve upon the heart-wall. As the destruction of the wall advances from within, a cavity is formed in the myocardium, often sinuous, and eventually, perhaps, leading to perforation. The chronic dilatation is most frequently found at the apex of the left ventricle, although sometimes observed in the interventricular septum, in which case the aneurism protrudes into the right ventricle in consequence of the greater pressure of the left ventricle against its contents. The aneurism varies in size, and either is a localized bulging of the outer surface of the ventricle or appears as a sac nearly as large as the fist. It is usually single, although sometimes two are present. In the latter case there may be one or two openings between the heart and the aneurismal sac. The wall of the aneurism is composed of pericardium and endocardium with the remains of the myocardium, and the opening between the ventricular cavity and the interior of the aneurism is either round or elongated, large or small. The larger aneurisms contain lamellated thrombi, which may almost entirely fill the cavity. The pericardial cavity is usually obliterated, and the wall of the aneurism may be calcified. At first sight the larger cardiac aneurisms suggest simply an hypertrophied heart, and the symptoms present during life are merely those of a weakened heart the beat of which is to be seen over an abnormally wide area, although the radial pulse is strong in comparison with the weak impulse of the apparently enlarged heart. Death usually results from progressive cardiac insufficiency, although sometimes from rupture of the aneurism into the pericardium or the lungs. The diagnosis is generally made after death. No treatment is of service.

RUPTURE.

Rupture of the heart results both from violence and from disease of the myocardium. Traumatic rupture is the result of penetrating wounds of the heart, or of violence applied to the surface of the body without producing evidence of any considerable external injury. In the latter case a sharply defined tear through the wall of the heart, especially of the auricle, takes place, there being but little hemorrhagic infiltration and no evidence of degeneration in the vicinity.

Spontaneous rupture occurs as the result of a previously diseased heart, oftenest in myomalacia from obstruction of the coronary arteries or from fatty degeneration, more rarely from the friction of a diseased valve during acute endocarditis or from the extension of an abscess to the surface of the heart. Rupture also occurs from the bursting of an aneurism of the heart, and in rare instances from tumors in the wall or from a thinning of the wall, or in consequence of the advance of a gastric ulcer through the intervening diaphragm and pericardium. The usual seat of spontaneous rupture is near the apex of the left ventricle, but the ventricular septum is sometimes perforated. The tear, often closed by a clot, is generally zigzag, the inner and outer openings not being on the same level. The walls of the rent are infiltrated with blood, and the limiting muscular fibre is either necrotic or in a condition of fatty degeneration. The pericardium contains more or less clotted blood. The symptoms due to the rupture generally follow those attributable to stenosis of the coronary arteries, chronic myocarditis, or fatty degeneration of the heart. The perforation through the pericardium is frequently preceded, perhaps for several days, by attacks of præcordial pain, a sense of oppression, and faintness or dyspnœa. Finally, perhaps after slight exertion, there are sudden intense pain referred to the heart, great anxiety, rapid respiration, absent pulse, perhaps cyanosis, followed by convulsions and death. The immediately fatal issue depends upon the compression of the heart by the abundant hemorrhage into the pericardial sac, which produces an increase in the area of cardiac dulness. Treatment of this condition is obviously of no avail.

TUMORS.

Primary tumors of the heart, as fibroma, myxoma, lipoma, and sarcoma, are rare. Of especial pathological interest is the congenital myosarcoma, with its striated muscle-cells. Secondary tumors, as cancer or sarcoma, may grow into the heart from adjacent structures, or may be therein localized in consequence of disease in remote parts. Single or multiple nodules may be present and result in extensive deformity of the heart. Since they produce no characteristic symptoms and demand no specific treatment, the interest in them is more pathological than clinical.

CHAPTER III.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

INFLAMMATION of the endocardium is to be regarded rather as a condition occurring in a variety of diseases and from numerous causes than as a sharply defined disease. All endocarditis is, therefore, to be considered as secondary, although clinically a primary endocarditis is of necessity admitted when satisfactory evidence of an antecedent disease is lacking. Usually the valvular endocardium is especially involved, but the inflammatory process may be limited to various parts of the parietal endocardium, especially to the trabeculæ and apices of the papillæ, while in many instances both the valvular and the parietal endocardium may be simultaneously diseased. In general, when the term endocarditis is used valvular endocarditis is the variety meant. Since, however, the clinical characteristics and perhaps the etiology of chronic endocarditis differ widely from those of acute endocarditis, the first distinction of practical importance to be drawn is that between acute and chronic endocarditis.

ACUTE ENDOCARDITIS.

ETIOLOGY.—It has long been assumed that inflammation of the endocardium is the result of an irritant brought by the blood, this irritant being supposed to exist in certain diseases from the frequent occurrence of endocarditis as a complication of such diseases. Importance in the etiology of endocarditis, therefore, has been assigned to acute articular rheumatism, chorea, influenza, diphtheria, pneumonia, scarlet fever, measles, variola, typhoid fever, relapsing fever, the infectious wound diseases, erysipelas, osteomyelitis and periostitis, pyæmia, septicæmia, puerperal infections, dysentery, malaria, tuberculosis, cancer, diabetes, and nephritis. Of late years attention has been directed to the occurrence of endocarditis as well as myocarditis in gonorrhœa. The discovery of the frequent presence of bacteria within and upon the diseased endocardium in infectious diseases has led to the prevailing view of the bacterial origin of endocarditis; hence the occurrence of an apparently primary inflammation of the endocardium is usually explained by the assumption of an infection from a suppurative process anywhere in the body, perhaps concealed or manifested by an apparently insignificant superficial abscess. The occurrence of endocarditis as a complication of ulceration of the alimentary canal or of an ulcerating cancer is also thus explained. Bacteria are found with great frequency in certain cases of endocarditis, although they have not been found in all. Among those found are the streptococcus, staphylococcus, diplococcus pneumoniæ, gonococcus, the bacillus of tuberculosis, the bacillus of diphtheria, and the typhoid bacillus. They may be present in abundance or so few in number that they are discovered only by the results of cultivation. When found they are to be considered as important in the etiology of the process, Orth having demonstrated that when bacteria alone were introduced into the circulation endocarditis did not follow, but if at the same time the valves were injured an infectious endocarditis arose, although injury alone was not equally efficient. All acute endocarditis, however, has not been proved to be bacterial, and since several varieties of bacteria are to be found in bacterial endocarditis, and this affection occurs in a variety of diseases, a ready explanation is offered for the otherwise perplexing variation in the symptoms, course, duration, and results of endocarditis.

A mixed bacterial infection is important in the etiology of endocarditis, since well-known micro-organisms are found in the affected valves in diseases whose specific bacterial origin is inferred but as yet not demonstrated. Local causes are also important, since bacteria may be present in the blood and no endocarditis result, and endocarditis is only an occasional complication of the various diseases in which it may occur. For example, endocarditis is more often seated in that side of the heart which works the harder. It is, therefore, more commonly found in the right side of the heart in the fœtus, and in the left side of the heart after birth. The lesions also are first to be found upon those portions of the valve which are exposed to the greatest friction. Its frequent occurrence in early life, especially in children, is attributable to a feeble resistance to bacterial action in youth. It is stated that a bacterial endocarditis may also result, although rarely, from embolism of the nutrient arteries of the valves, especially of the cuspid valves. Non-bacterial forms of endocarditis are designated as simple endocarditis. in contradistinction to bacterial or mycotic endocarditis. A clinical distinction is drawn between simple and septic or malignant endocarditis from the variations in the character of the symptoms, progress, and results. Abundant bacteria are present in septic endocarditis, whereas they are often to be found with difficulty, if at all, in simple endocarditis. The distinction, however, is one of degree in the severity of the endocarditis, and is not based upon absolute etiological differences.

MORBID ANATOMY.—At the outset of acute endocarditis there are slight swelling and opacity of the endocardium, which soon becomes further modified by the formation of granulation-tissue within the valve. These early changes are to be found along the line of apposition. As the inflammation progresses, granular or warty excrescences project from the surface, when the term warty or verrucous endocarditis is applied. The vegetations or outgrowths are covered with granular and fibrillated material composed of leukocytes, blood-plates, and fibrin, essentially a thrombus, or the thrombus may first be formed, being subsequently invaded by the granulations. The thrombi may attain a considerable size, and bacteria are sometimes present, but with far less frequency than in the ulcerative variety of endocarditis. These alterations, limited at the outset, involve in the course of time more and more of the valve, and may extend to the tendons and adjacent portions of the parietal endocardium.

Acute endocarditis may also progress with extensive destruction of the valve, and is then designated diphtheritic, acute ulcerative, septic, or malignant endocarditis, according as the anatomical changes or the clinical characteristics are made prominent. The alterations at the outset resemble those occurring in verrucous endocarditis, but the valve rapidly becomes thickened and opaque and of diminished consistency. Superficial loss of substance early occurs, and thrombi of considerable size are formed upon the valve. Both the thickened valve and the thrombi usually contain bacteria in enormous numbers, and the inflammatory process rapidly extends to the valve-tendons and to the parietal endocardium, especially where this is exposed to the friction of a diseased valve. In consequence of the diminished resistance of the valve, the weakest portion of the crescents or cusps often yields to the pressure of the blood, and a localized diverticulum, perhaps of the size of a pea, the *acute valvular aneurism*, arises, which not infrequently becomes perforated. Some of the softened tendons of the mitral valve may be torn apart. As previously stated, the so-called acute aneurism of the heart is the result of the beating of the diseased valve against the myocardium, which is invaded by bacteria and undergoes an acute necrosis. Acute ulcerative endocarditis is therefore regarded as a secondary condition occurring in the course of septic infection, and the localization in the valvular endocardium of the infectious bacteria is favored by previous disease of the valves.

Embolism may occur both in simple or verrucous endocarditis and in the septic or acute ulcerative variety. In the former the emboli are carried to the spleen, kidneys, brain, and extremities, and are usually productive of mechanical disturbances alone. In malignant endocarditis the emboli, being small but septic, are less productive of mechanical than of inflammatory disturbance. The numerous minute emboli are often transferred to various parts of the body, and give rise to miliary abscesses either in the heart or in the spleen, kidneys, liver, stomach, and intestine, brain, or eye. Miliary embolism also occurs in the joints and skin.

SYMPTOMS.—The symptoms of acute endocarditis vary within wide limits. In the simple form, which is usually of the warty variety, there may be no symptoms indicative of this lesion. On the other hand, the evidence of an endocarditis may first be furnished by the symptoms of embolism, as sudden hemiplegia, renal pain, and hæmaturia, or pain in the region of the spleen in case of arterial embolism, or dyspnœa, hæmoptysis, cough, and pleuritic pain in pulmonary embolism. Since simple endocarditis is of most frequent occurrence as a complication of acute rheumatism, in this affection repeated examinations of the heart should be made with reference to the discovery of abnormal physical conditions in this organ. Attention may first be directed to the heart by the occurrence of palpitation, a sense of præcordial oppression, or perhaps dyspnœa, or there may be a sudden increase in the fever without any modification in the course of the articular inflammation. On the other hand, there may be no especial alteration in the range of the temperature or pulse.

The appearance of a murmur over one of the valves is no necessary indication of an endocarditis, since the murmur may be functional or hæmic, and endocarditis often occurs without any murmur being recognized. The distinction between a murmur caused by organic disease of the valves or of the orifice and a murmur independent of such lesions is not always to be easily made. The cardio-respiratory murmurs, represented by a jerking inspiration synchronous with the beat of the heart, are readily eliminated, since they are limited to inspiration and cease when the breath is held. An endocardial murmur is to be differentiated from an exocardial murmur by its usual association with the systole or diastole, its greatest intensity near a valve, its deep seat, and its failure to become increased by pressure of the stethoscope or by a change to the upright position. The functional murmurs occur in relative insufficiency of the valves, in which dilatation of the orifice prevents closure by the cusps or crescents; and also in functional insufficiency, in which in consequence of fatty degeneration of the papillary muscles the cusps are not held in place. They are present also in anæmia and in convalescence from febrile diseases. A systolic murmur at the base is likely to be functional if limited to the left of the sternum and unaccompanied by a thrill; if at the right of the sternum, it is likely to be functional provided it occurs in a healthy person or in one not suffering from signs and symptoms of aortic stenosis. A systolic murmur at the apex in persons free from symptoms of cardiac incompetency may be functional or organic, and its actual nature is to be determined only in the course of time by the disappearance of the functional and the persistence of the organic murmur. Organic murmurs vary in intensity, pitch, and character, and when systolic are frequently associated with disappearance of the first or second sound, according to the seat of the murmur. Functional murmurs are generally soft and occur during the systole, although an organic murmur may be soft and systolic; but with functional murmurs the heart-sounds are unaffected.

Endocarditis is designated septic or malignant when symptoms of septicæmia are present and are associated with conspicuous evidence of a diseased endocardium. In infectious diseases the occurrence of malignant endocarditis is often manifested merely by an aggravation of the septic symptoms. Malignant endocarditis usually follows one of two types, the typhoid or the pyæmic. In the typhoid type there is a continued atypical fever with considerable variations in the morning and evening temperature, a rapid pulse of diminished tension and volume, marked prostration, headache, perhaps delirium, and muscular pains. The skin is often dusky, sometimes jaundiced, petechiæ or rose-spots are not infrequently present, and there may be profuse sweating. The abdomen is often moderately distended and tympanitic, and enlargement of the spleen is frequent. The tongue is dry, and diarrhœa is often present. The urine is high-colored and faintly albuminous. Although the physical examination of the heart may disclose a mitral or an aortic murmur, this is not necessarily present. The area of cardiac dulness is usually slightly enlarged. If the physical examination of the heart is negative, the symptoms are essentially those of a septicæmia of obscure origin. In the pyæmic type of malignant endocarditis there is less mental and physical prostration at the outset than in the typhoid variety, and the temperature is intermittent, with extreme variations in the daily range. The exacerbations are often associated with chills, and the fall of the temperature is accompanied by profuse sweating. The chills are usually irregular, though sometimes of such periodicity as to suggest intermittent fever, and in the intervals between them the patient may be comparatively comfortable. There is progressive though gradual loss of flesh and strength. This variety may be prolonged over a period of months, Osler having reported a case continued over a period of ten months with daily intermittent pyrexia, the height of the temperature varying from 102.5° to 104° F., occasionally preceded by chills, oftener by chilliness. In this variety the symptoms of embolism are of frequent occurrence, manifested in the skin by a petechial or a papular eruption, in the intestine by diarrhœa especially of a hemorrhagic character; by symptoms of acute meningitis, perhaps by hemiplegia, when the brain is affected, and by pain in the region of the spleen, hæmaturia, or signs of acute hemorrhagic nephritis, in the case of involvement of the spleen and kidneys. The physical examination of the heart is negative, or murmurs are to be heard attributable to disturbance of function, or the signs are those of a localized valvular disease. The last event is of constant occurrence when the malignant endocarditis is a recurrent attack.

The course of acute endocarditis varies extremely in accordance with the etiology, the previous condition of the patient, and the various complications. Although cases of simple or verrucous endocarditis generally recover from the immediate symptoms, chronic valvular endocarditis is the usual outcome. Malignant endocarditis may prove fatal within a few days, or, as in typhoid fever, within a few weeks, or, as in chronic pyæmia, the course may extend over a period of months. Simple endocarditis may suddenly assume a malignant type. The embolic disturbances in simple endocarditis may be severe in virtue of the large size of the emboli and the situation of the organ obstructed. The embolism of malignant endocarditis is more serious, although the emboli are usually smaller. Their number and infectious qualities are the chief causes of their danger, since the suppurative inflammation of serous membranes is likely to result from their frequent presence near the surface of organs.

DIAGNOSIS.—Since the physical examination of the heart may give no evidence of a localized lesion, the diagnosis of acute endocarditis is often made only at a post-mortem examination. In the diseases in which endocarditis occurs as a complication, attention may be directed to the heart by palpitation, præcordial discomfort, and dyspnœa out of proportion to the febrile condition present. If a murmur is heard its significance is to be determined by the characteristics stated. The existence of the endocarditis may first be made apparent by the recognition of the symptoms of embolism of the general arterial system. The probable simple or septic course of the endocarditis often may be inferred from the etiology even before the characteristic septic or pyæmic symptoms arise. The endocarditis complicating acute rheumatism, chorea, small-pox, typhoid fever and diphtheria, scarlet fever and debilitating diseases, is likely to be of a simple character, while the endocarditis present in traumatic, puerperal, and gonococcal infections is often of a malignant type. The endocarditis occurring in pneumonia is generally simple, but may be malignant. A considerable enlargement of the spleen and cutaneous and retinal hemorrhages are evidences of the malignant nature of the endocarditis. Since the symptoms of malignant endocarditis are those of a toxæmia, either a septicæmia or a septico-pyæmia, and the course of malignant endocarditis may resemble that of typhoid fever, malarial fever, or acute tuberculosis, it frequently becomes necessary to exclude these diseases. The onset of typhoid fever is usually more gradual, the range of temperature is more typical, and functional disturbance of the heart is not a conspicuous feature. Malarial fevers are to be excluded by the examination of the blood or by the inutility of quinine. In acute general tuberculosis the onset is more gradual, there is often evidence of a localized tuberculosis, and the predominant signs are those of pulmonic disease.

PROGNOSIS.—The prognosis of simple acute endocarditis is generally favorable as to the immediate outcome, although eventually serious, since this disease is usually productive of an incurable valvular deformity, and it is also to be remembered that grave results from embolism may occur. Furthermore, the prognosis is to be guarded, since simple endocarditis quickly may become septic. The prognosis of malignant endocarditis, although extremely grave, is not absolutely hopeless. The immediate attack, if recovered from, leaves behind a permanently damaged heart.

TREATMENT.—In the great majority of cases endocarditis is due to an infection which can be little modified by treatment. If, however, it is possible to affect the cause of the endocarditis, no time should be lost in so doing. Thus, an antitoxin may be used in diphtheria; or, if the case be of rheumatic origin, ammonium salicylate should be given for the first forty-eight hours in sufficient doses to produce pronounced cinchonism. Subsequently great caution is necessary in the use of the salicylates, on account of their markedly depressing influence. In some cases of sthenic endocarditis in the earlier days of the disease calomel should be exhibited.

When the endocarditis is fully developed, the treatment must be symptomatic. If there be sthenic cardiac excitement, aconite should be employed ; when, as is usually the case, the tendency is to cardiac weakness, it is a very dangerous remedy. Digitalis is to be used even in the earlier stages of the disease when there is great cardiac embarrassment with irregular heart-action ; but in cases which are not septic digitalis is not often required until the later stages of the disease, when the symptoms of cardiac failure become threatening or the acute endocarditis is passing into valvular disease and it is important to favor compensatory hypertrophy. When heart-failure is imminent, diffusible stimulants, such as alcohol, ether, and camphor, may be used : strychnine is important. For the control of pain morphine may be given hypodermically. It is better to repeat small doses, at short intervals if necessary, than to exhibit large single amounts.

Local treatment is of little, if any, avail. If the application of ice, or of Leiter's tubes with cold or with hot water, be grateful to the patient, it may be resorted to. Mustard plasters are to be employed in crises of suffering and distress. It is very doubtful whether blisters have any influence upon the disease, and they may add greatly to the suffering of the patient.

During the whole course of an endocarditis absolute quiet should be insisted upon, with most careful nursing to prevent exertion or exhaustion. The patient should not be allowed to get out of bed for any purpose. The food should be light and nutritious.

CHRONIC ENDOCARDITIS.

Etiology.—Chronic endocarditis commonly results from the persistent and progressive character of the tissue-changes arising in acute endocarditis, especially in the verrucous variety, since malignant endocarditis is so often fatal. In the rare cases of recovery from malignant endocarditis permanent alteration of the endocardium also results : hence the etiology of chronic endocarditis is largely that of acute endocarditis, the majority of the cases being the result of acute articular rheumatism. In addition, importance is to be attached to syphilis, gout, alcohol, and lead, and also to the complex and indefinite disturbances occurring in advancing years, among which may be included the effects of extreme or prolonged strain of the heart.

Morbid Anatomy.—A parietal and a valvular form of chronic endocarditis occur, but the latter alone is of especial clinical importance. Two anatomical varieties of chronic valvular endocarditis are to be recognized, the one a fibrous endocarditis, the other a chronic ulcerative endocarditis. Fibrous endocarditis is manifested by thickening, induration, adhesion, and contraction of the valve crescents or cusps. The valves thus become shortened, and the septa between adjacent crescents may be so diminished that two of the crescents are apparently fused into one, and the cusps of the auriculo-ventricular valves may be unified by adhesions. In addition, lime salts may be deposited in the deformed valves, in consequence of which they become rigid. The calcification takes place throughout the valve or at limited portions at the free edge or in the region of attachment, and the lime salts may also be deposited in thrombi attached to the surface of the valve. As a result, the valves may have a mulberry-like or jagged surface, the valve-tendons may become thickened, indurated, adherent, and shortened, and the insertion of the mitral valve, in which such changes are frequent, may be represented by a calcareous ring. In chronic ulcerative endocarditis, necrosis and fatty degeneration take place in the thickened valve, and the disintegrated portions are washed away in the blood-current, leaving more or less extensive loss of substance, the atheromatous ulcers.

Chronic valvular endocarditis of congenital origin affects the right side of the heart, especially the tricuspid valve. Acquired endocarditis is usually limited to the left side of the heart, although in rare instances the right side may be affected. The mitral valve is oftenest diseased, and next in frequency is inflammation of the aortic valve. The tricuspid valve is occasionally the seat, but the pulmonary valve is rarely inflamed. The aortic and mitral valves may be simultaneously affected, and the mitral and tricuspid likewise. In rare cases the aortic, mitral, and tricuspid valves are diseased, while in extremely rare instances all the four valves are inflamed. Disease limited to the mitral valve is more frequent in early life, while late in life the aortic valve is oftener diseased, largely owing to the frequency of chronic endaortitis after fifty years of age.

The effect upon the heart depends upon the duration, degree, and result of the valvular affection, and is manifested by dilatation of the cavities and hypertrophy of the wall, the one or the other predominating according to the nature of the functional disturbance of the valve: these alterations are found in one cavity or in one side of the heart according to the valve conspicuously altered. In extreme cases of hypertrophy the weight of the heart may be increased threefold or fourfold, and the thickness of the ventricular wall may be three or four times the normal. Papillæ and trabeculæ are enlarged, and the myocardium is increased in density and of a dark-red color, becoming grayish-red as compensation fails. The endocardium, particularly of the cavity longest exposed to the effects of the valvular disease, becomes thickened, and the wall of the dilated auricle, especially of the left auricle in mitral stenosis, may be largely fibrous.

SYMPTOMS.—The symptoms of chronic endocarditis are those of chronic valvular disease of the heart, and are of gradual development in consequence of the progressive nature of the valvular deformity and the simultaneous compensatory hypertrophy of the wall of the heart. The existence of a chronic endocarditis is often recognized on physical examination before any symptoms arise, or is discovered when cardiac symptoms appear in the course of an acute recurrent endocarditis.

The effect of the thickening, contraction, calcification, or perforation of the valves is such deformity as to interfere with their function or to produce an obstruction at the orifices which they guard. Either the valves become incompetent to close the orifice, and hence permit a regurgitation of the column of blood, or a narrowing of the orifice is occasioned, causing obstruction to the passage of blood. The incompetence of the valves to close the orifice is usually designated *insufficiency*, while obstruction to the passage of blood through the orifice is indicated by the term *stenosis*. Often the alterations of the valves are such that the orifice is narrowed and the valves are unable to close the opening, hence incompetence and stenosis coexist. A distinction is drawn between organic, relative, and functional insufficiency of the valve. In the first the inability to close the orifice is the result of anatomical changes in the valves or at the orifice. In relative insufficiency the normal valves are unable to close the orifice, owing to its dilatation with the dilatation of the heart, whereas in functional insufficiency the papillary muscles are so weakened, usually in consequence of fatty or fibrous degeneration, as to be unable to hold the valve-curtains in place.

The immediate effect of incompetence of the valves or of stenosis of the orifice is an overfilling of the auricle or the ventricle, which is able to empty itself only by increased work. If the valves are insufficient, the ventricle or the auricle, according as the crescentic or the cuspid valves are affected, becomes distended not only by the blood which normally enters in sufficient quantity to fill it, but also by the return of blood already expelled which leaks through the incompetent valves. In stenosis additional work is demanded of the heart to force through a narrowed opening the normal quantity of blood contained by the cavity. This additional work is accomplished by means of an increase of the heart-muscle, to which the term compensatory hypertrophy is applied. That such hypertrophy may take place it is necessary that the digestion be relatively normal, that sufficient and suitable nourishment be had, and that the various functions of the body be in good working order. Since the valvular lesions tend to produce increased deformity in the course of time, the hypertrophy also progresses, and as long as it is sufficient little or no disturbance to the comfort of the individual results. If, however, digestion fails, or food is defective in quality or quantity, or repeated unusual demands are made upon the heart, the muscular fibre degenerates, and the cavities of the heart become incompetent to expel their contents. Since a normal quantity of blood then cannot be forced through the left side of the heart, it accumulates in the pulmonary veins and capillaries, thereby producing an increased resistance to the passage of blood through the right ventricle into the pulmonary artery. The inability of the right ventricle to empty itself causes an accumulation of blood in the right auricle and also in the general venous system. The ultimate effect, therefore, of all valvular disease of the heart is failing compensation and venous congestion throughout the body.

The laboring heart then beats appreciably and sometimes tumultuously, the bronchial mucous membrane becomes swollen from the congestion of its blood-vessels, and the distended pulmonary capillaries project into the alveoli, which are thus prevented from receiving the necessary quantity of air, and rapid and short breathing results, indicative of the attempt to aerate the increased quantity of blood in the lungs. This cardiac dyspnœa is therefore chiefly due to mechanical causes interfering with the normal rapidity of the flow of blood through the lungs and with the admission of air for its oxygenation. It is therefore of earlier and more constant occurrence in mitral disease than in affections of other valves. Both inspiration and expiration are impeded, and the frequency of respiration is accelerated. As the mechanical difficulties increase in consequence of the progressive weakening of the compensatory hypertrophy, the difficulty of breathing becomes more apparent, and is constant, instead of being occasional and produced only by obvious causes.

The dyspnœa is not infrequently manifested in the form of *cardiac asthma*, which occurs more frequently in aortic than in mitral disease, and occurs in fibrous myocarditis or in simple dilatation of the left ventricle. The attacks are occasioned by some obvious exciting cause, as unusual physical exercise, mental or moral disturbance, an attack of indigestion, prolonged exposure to cold, or may occur without any appreciable exciting cause. The patient is not infrequently aroused from sleep by a sense of substernal constriction, and the asthmatic attack rapidly progresses, anxiety, orthopnœa, cyanosis, sweating, and coldness of the skin being present. The pulse is quickened and of diminished tension. Often moist râles are to be heard in the posterior and lower portions of the lungs, and dry râles are present elsewhere. There is usually little or no expectoration, although in extreme cases acute œdema may occur and a bloody frothy fluid be raised. The paroxysm may last an hour or more, gradually lessening in severity until it fades away. The attacks recur frequently or after long periods of intervening freedom from disturbance, according to the cause and the effects of treatment.

The especial variety of dyspnœa to which the term *Cheyne-Stokes breathing* is applied occurs also in the failing compensation of cardiac disease, especially in aortic affections, fibrous myocarditis, and fatty degeneration. In this variety of dyspnœa the patient ceases to breathe for an interval of fifteen to thirty or more seconds, when the breathing begins, becoming rapidly and progressively quickened for an interval of several seconds, after which the respirations are fewer and longer and finally cease, the inspirations being resumed at intervals of a minute, more or less. During the period of arrested breathing the pulse is rapid and weak, but becomes slower and stronger with the beginning of respiration. Cheyne-Stokes breathing occurs during sleep or when the patient is awake, and is continuous or paroxysmal. Its occurrence is usually indicative of a late stage in failing compensation, although we have known it to be continued at intervals for a period of two years. It is frequently relieved by appropriate treatment.

The difficulty of respiration is added to in the course of time by a transudation of serum from the blood-vessels, which accumulates in the alveoli and bronchi, thus further interfering with the admission of air. The alveolar epithelium is desquamated, red blood-corpuscles pass through the walls of the blood-vessels, the blood coloring matter precipitates, and the lung becomes dense, of an iron-rust color, and contains abundant blood and serum, the condition known as *brown induration.* Rupture of the pulmonary vessels also occurs, portions of the lung becoming engorged with blood and presenting a state of infarction resembling that caused by embolism.

As the hypertrophy of the right side of the heart fails to force blood through the lungs, congestion of the peripheral venous system results. The distended jugular veins are apparent as dark-blue lines or cords, which are in constant motion in part from the transmission to the distended vein of the impulse of the auricular contraction, in part from recurrent diminution in volume during inspiration. An actual venous pulsation synchronous with the systole of the heart exists when in tricuspid insufficiency the dilatation of the veins is sufficient to produce incompetence of the valves at the entrance of the jugular into the innominate vein. The smaller veins become dilated and tortuous, and the capillaries are so distended with blood that the skin, especially of the face, ears, fingers, and toes, becomes of a blue color. The nutrition of the tips of the fingers and toes is affected, and they become club-shaped. The first effect of the venous congestion of the liver is to produce such enlargement of this gland that the anterior border may be found below the level of the navel. The enlarged liver may receive and transmit the impulse from the aorta, which is to be distinguished from the expansile pulsation due to venous engorgement in tricuspid insufficiency. With the persistence of the congestion destruction of the liver-cells and moderate increase of the fibrous tissue take place, finally resulting in the formation of the small pigmented liver known as the *nutmeg liver.* The obstruction to the passage of blood through the liver causes passive congestion of the radicles of the portal vein in the walls of the stomach and the intestine, and chronic catarrah of the gastro-intestinal mucous membrane follows, which interferes with digestion and thus checks the effectual nutrition of the hypertrophied heart.

In the early stage of portal obstruction the spleen is moderately enlarged, while in the later stage its density becomes increased, the process being similar to that which takes place in the liver.

The check to the flow of blood from the kidneys interferes with the transudation of fluid through the glomeruli and causes the kidneys to become dense and purple, a condition to which the term *cyanotic induration* is applied. The quantity of urine is diminished, and its specific gravity is increased. There is an abundant brick-dust sediment, with occasional red blood-corpuscles, and perhaps a trace of albumin, which characteristics of the urine are indicative of a chronic passive congestion of the kidneys.

As the result of prolonged congestion of the peripheral venous system, a transudation of fluid from the veins eventually takes place, and dropsy appears. The fluid in *cardiac dropsy* is usually first observed in that part of the body from which blood is returned to the heart with the greatest difficulty,—namely, in the feet, especially about the ankles. The œdema, therefore, in ambulatory patients, is at first noticed at the end of the day, disappearing during the night; but with the increasing weakness of the heart the dropsy increases, and affects the thighs, the walls of the chest and abdomen, and the scrotum. Eventually fluid appears in the peritoneal, pleural, and pericardial cavities; finally it is poured into the lungs, and then often proves the immediate cause of death.

Among the most important of the complications of chronic valvular endocarditis is embolism. The formation of thrombi is promoted by the stagnation of the blood and an alteration of the surface over which the blood passes: hence they are present in those parts of the heart in which the current moves the slowest, namely, in the auricular appendages and in the intertrabecular recesses, which, in the dilated heart particularly, form pockets sunk into the wall. They arise also in the widened recesses above the valves of veins, especially of the legs and in the dilated and sacculated pelvic plexus of veins. The thrombi, becoming dislodged, are carried along as emboli, and produce the various phenomena of embolism according to their place of origin and distribution. Venous emboli and those from the right side of the heart, which are obviously the more numerous owing to the greater surface from which they may arise, follow the distribution of the pulmonary artery, and according to their size either produce sudden death when unable to pass into the primary divisions of the pulmonary artery, or give rise to large or small wedge-shaped masses of embolic infarction of the lungs. Emboli transferred from the left side of the heart also cause sudden death if too large to pass through the mitral or the aortic orifice, and when small may reach the brain, causing aphasia or hemiplegia, or pass into the spleen and produce a localized splenitis, more rarely obliterate the splenic artery and give rise to a secondary thrombosis of the splenic vein extending to the superior mesenteric vein, usually ending in fatal hemorrhagic infarction of the intestine. If the emboli are carried to the kidneys, focal necrosis occurs, which may involve an entire kidney if the trunk of the renal artery is obstructed, the symptoms being those of an acute hemorrhagic nephritis, or the emboli may be carried into the arteries of the extremities, producing temporary functional disturbance or necrosis and gangrene.

PROGNOSIS.—The prognosis of valvular endocarditis is more dependent upon general conditions than upon the valve concerned or the nature of its lesion. While compensation is satisfactory the immediate prognosis is favorable. The existence of causes which tend to enfeeble compensation makes the prognosis correspondingly serious. In the valvular endocarditis of children hypertrophy and dilatation rapidly progress, and compensation is earlier than in the endocarditis of adults, in whom compensatory hypertrophy is of more gradual occurrence and keeps a more uniform pace with the disturbances to be overcome. The endocarditis of elderly people, so frequently the result of arterio-sclerosis, may exist for years with but little disturbance, provided the general nutrition is relatively normal. The habits, exposure, and occupation of the person diseased are also important in prognosis. Excessive action of any sort, mental, moral, or physical, increases the demand upon a heart readily displaced from a condition of equilibrium, and sudden or prolonged strains often produce extreme disturbances of balance. Pregnancy and parturition act as continuous or temporary causes of increased work of the heart, and repeated child-bearing tends rapidly to weaken compensation. If puerperal infection follows childbirth, the localization of the bacteria upon an already diseased valve is of frequent occurrence.

Disease of the valves, whether acute or chronic, local or general, increases the gravity of the prognosis of any disease, but especially of pneumonia, typhoid fever, or other severe or prolonged infection, of fibrous myocarditis, and of prolonged anæmia. The danger is less immediate provided the degenerated myocardium can be stimulated by the action of such a remedy as digitalis or strophanthus.

Opinions differ as to the prognosis of chronic valvular endocarditis in relation to the valve affected; but it is generally agreed that disease of the valves of the right side of the heart earlier proves fatal than that of the left, in consequence of the more immediate production of cyanosis and dropsy, the usual terminal symptoms of valvular endocarditis. Of mitral or aortic disease occurring before middle life, the former is sooner associated with evidence of failing compensation than the latter, and in general compensatory hypertrophy of the right ventricle yields earlier to disturbance of nutrition than hypertrophy of the left ventricle. Writers particularly disagree as to the more serious import of stenosis or insufficiency of the aortic and mitral valves. This difference of opinion probably depends in large part upon the fact that stenosis of the orifice is generally, if not always, associated with incompetence of the valve, the degree of which varies within wide limits, whereas primary insufficiency of the valve is usually unaccompanied by obstruction at the orifice. Sudden death after middle life in the presence of effective compensation is more likely to occur in aortic insufficiency in consequence of the frequent simultaneous disease of the coronary arteries. Sudden death, however, is not infrequent in mitral stenosis, but when it does occur it is the result of embolism of the valve-orifice, or of the pulmonary artery from thrombi formed in consequence of the sluggish circulation in the later stages of failing compensation. The prognosis is always more unfavorable in combined valvular disease than in the affection of a single valve.

MITRAL INSUFFICIENCY.

The commonest of all the valvular affections is mitral insufficiency, which may occur alone or in combination with stenosis. The immediate effect is the regurgitation of blood into the left auricle during the contraction of the left ventricle. This tends to prevent the reception of the normal amount from the pulmonary veins and to produce a diminished flow from the ventricle in systole. The auricle thus becomes overdistended, its cavity dilated, and its wall hypertrophied, although compensation is chiefly obtained by hypertrophy of the right ventricle. The signs of mitral insufficiency are, therefore, a systolic murmur, resulting from the regurgitation of the blood-current, and to be heard loudest at the apex. The murmur varies in intensity, but is increased on slight exertion, even on change of position, especially from the horizontal to the vertical. When loud it is to be heard elsewhere, especially in the axilla and the back. In rare instances it may be heard at the left of the sternum directly over the valve, and is sometimes continued into the diastole. According to the degree of alteration of the valve the murmur may or may not be accompanied by the first apex-sound. The area of cardiac dulness is increased, especially in the transverse direction, and in extreme cases may extend from beyond the left mammillary line to the right of the sternum. In young persons with a yielding thoracic wall a bulging of the præcordial area may exist. In consequence of the hypertrophy and dilatation of the right ventricle the apex-beat is intensified and perceptible over a greatly enlarged area, and the pulmonic second sound to be heard near the sternum in the second left intercostal space is strongly accentuated, on account of the increased pressure against the wall of the pulmonary artery. The aortic second sound is enfeebled from a diminished pressure against the aortic wall, and may be inappreciable at the apex. The pulse is of diminished volume and tension. There may be no symptoms for years, during which the compensation is sufficient, except on exertion, when temporary shortness of breath and a quickened pulse occur. As compensation fails cyanosis occurs.

Systolic murmurs often loud in character may be heard at the apex and be transmitted into the axilla independently of organic disease of the mitral valve. Such murmurs, functional or hæmic, occur in a variety of diseases, even in robust persons in vigorous health, as shown by Prince in his examination of candidates for the position of fireman or policeman, but are not associated with increase in the area of cardiac dulness.

MITRAL STENOSIS.

Next in frequency to mitral insufficiency is stenosis of the mitral orifice, the immediate effects of which are essentially the same as those of insufficiency,—namely, dilatation and hypertrophy of the left auricle and hypertrophy and dilatation of the right ventricle. A diastolic murmur occurs as the left auricle forces blood into the ventricle, ending with the production of the first sound, increased on exertion, and is heard loudest at the apex. This murmur often increases in intensity at the end of diastole, and is perhaps first heard at this time; it is, therefore, often called presystolic. The diastolic murmur is often associated with a systolic murmur from the frequent combination of insufficiency with stenosis. The area of cardiac dulness is increased laterally, and may extend from the right of the sternum to the mammillary line. The apex-beat is usually felt in the fifth intercostal space in the vicinity of this line. The second heart-sound is frequently reduplicated, and Guttmann considers this an important diagnostic sign of mitral stenosis, since the duplication is at times to be heard during the temporary absence of the murmur. In consequence of the hypertrophied right ventricle there is accentuation of the pulmonic second sound. Another important sign of mitral stenosis is the purring thrill, increasing on slight exertion, and to be felt near the apex. The pulse is weaker than in mitral insufficiency, and is usually irregular. The signs of congestion of the pulmonary and of the peripheral veins occur earlier than in mitral insufficiency.

AORTIC INSUFFICIENCY.

In aortic insufficiency the ventricle becomes distended by the admission of blood during the diastole both from the left auricle and from the aorta. An abnormally increased quantity of blood is thus constantly being received, which is to be expelled only by increased work: hence the ventricle becomes hypertrophied and the whole heart is enlarged. The physical signs are a prolonged diastolic murmur, continued downward towards the apex, heard with greatest intensity in the midsternal region, and sometimes extended to the right and left of the sternum. The second sound of the heart is either obscured by the murmur or is absent in consequence of the disease of the valves. A systolic murmur due to an associated narrowing of the orifice is sometimes also to be heard, especially at the second right intercostal space, although it may be very faint. At the apex a systolic as well as the diastolic murmur is to be heard, either transmitted from the aortic orifice or resulting from organic or functional mitral incompetency. In the latter case the first heart-sound is not present at the apex. On auscultation of the carotid and subclavian arteries the diastolic murmur is sometimes transmitted, also a double murmur when existing at the aortic orifice, and the second sound, if present, is transmitted. If the arteries are compressed, a murmur is readily produced, and on pressure upon the femoral artery a double murmur and even two sounds may be heard. A systolic sound is to be heard on auscultation of the arteries, even of those at the wrist.

The area of cardiac dulness is increased, especially in length, and may extend as low as the seventh rib and beyond the left mammillary line. The apex-beat may be found outside the nipple in the sixth intercostal space, and is perceptible over a wide area, heaving of the chest being associated with each beat of the heart. The visible and palpable arteries are dilated and tortuous, and pulsate strongly. The contraction of the powerful left ventricle causes the arteries to be at once distended to the maximum, and an immediate fall of the tension results from the inability of the valves to hold the column of blood. This pulse, the *water-hammer* or *Corrigan pulse*, is often more readily recognized at the wrist by holding the arm upright. The capillaries are frequently distended and visibly beat, as may be seen in the pulsating flush of the cheeks or of the bed of the nail made anæmic by pressure upon the nail, or of the everted lower lip through a piece of glass pressed upon the mucous membrane.

Aortic insufficiency usually exists without symptoms for many years, owing to the efficient compensation caused by hypertrophy of the left ventricle. Palpitation is often not apparent, except on exertion, until compensation fails, when the attacks become more frequent. The palpitation may be associated with throbbing headache and wakefulness, and the patient be unable to lie on the left side. A feeling of faintness, especially on assuming the erect position, dizziness, and flashes of light occur. There is frequent pain referred to the region of the heart and sometimes extending into the left arm (angina pectoris), and dyspnœa is very troublesome. These symptoms are more and more easily induced by exercise or indigestion; finally the mitral orifice becomes dilated, relative insufficiency occurs, and the symptoms of venous congestion of the pulmonary and body veins result. Death not infrequently takes place before relative insufficiency of the mitral valves occurs, either suddenly from cardiac paralysis, or more gradually from cerebral hemorrhage or arterial embolism.

AORTIC STENOSIS.

Aortic stenosis is the least frequent of the valvular affections of the left side of the heart, and, like mitral stenosis, is usually combined with some degree of incompetency. The effect upon the ventricle is the same as that from aortic insufficiency,—that is, dilatation and hypertrophy result, but the hypertrophy predominates over the dilatation. A systolic murmur is heard loudest near the third right costal cartilage and extends into the large arteries of the neck. The murmur is at times accompanied by a thrill, which when loud is to be heard throughout the cardiac area. The second aortic sound is often combined with a diastolic murmur, from the associated insufficiency. The area of cardiac dulness is increased downward and outward, but the increase is moderate in comparison to that resulting from aortic insufficiency. The apex-beat may be powerful and heaving or feeble and inconspicuous, according to the condition of the myocardium and the degree of hypertrophy. The pulse is of small volume, of increased tension, and usually of diminished frequency. During compensation aortic stenosis produces no symptoms. With failing compensation palpitation, headache, dizziness, and faintness occur, followed by cardiac pain and dyspnœa as in aortic insufficiency.

TRICUSPID INSUFFICIENCY.

Tricuspid insufficiency when organic is usually the result of a fœtal endocarditis, although in rare instances it may be due to endocarditis acquired after birth, in which case disease of the mitral or the aortic or of both valves is associated with it. Relative insufficiency of the tricuspid valve is frequent, and is the usual result of mitral disease and sometimes of aortic endocarditis. Tricuspid insufficiency also follows diseases of the lung which prevent the flow of blood through the pulmonary artery, such as emphysema, fibrous pneumonia, and bronchiectasis. The blood regurgitates into the right auricle, backs into the venæ cavæ, and produces engorgement of the veins of the body, with the effects already mentioned. A systolic murmur is to be heard at the right of the lower part of the sternum and may extend outward from this region. The intensity of the second pulmonic sound is rather diminished than increased; any resulting hypertrophy of the right ventricle is usually moderate, hence the transverse area of dulness is but slightly increased, and the apex-beat, though forcible, is neither diffused over a wide area nor much displaced. Especially important in diagnosis is the evidence of passive congestion of the veins of the body, characterized by the jugular pulse, which is most forcible in the right internal jugular vein and when the patient is on his back. Less frequent is the recognition of expansile pulsation of the liver from passive congestion of the branches of the hepatic vein. The symptoms of tricuspid insufficiency are cyanosis, dropsy, and the modifications in the secretion of urine already mentioned in the symptomatology of valvular endocarditis. These symptoms occur alone or are associated with those of respiratory disturbance, according as tricuspid insufficiency exists as a primary or as a secondary condition, independently or associated with pulmonary or other valvular lesion.

TRICUSPID STENOSIS.

Tricuspid stenosis is extremely rare, and is usually of congenital origin, though sometimes acquired after birth, in which case it is associated with mitral or aortic or both mitral and aortic disease. In consequence of the obstruction to the flow of blood through the tricuspid orifice the right auricle is dilated and hypertrophied and the peripheral venous system becomes congested. A diastolic or a presystolic murmur is to be expected loudest near the ensiform cartilage, and is thereby to be distinguished from the murmur of mitral stenosis, which is to be heard near the apex. The effects of tricuspid stenosis are those of tricuspid insufficiency, but are of more rapid occurrence.

DISEASE OF THE PULMONARY VALVE.

Endocarditis of the pulmonary valve is of extreme rarity, usually congenital, although possibly of traumatic origin from a severe strain after birth. A diastolic murmur loudest at the right of the midsternal region concealing the second sound of the heart is to be expected in *pulmonary insufficiency.* This murmur is sometimes associated with a systolic murmur, which is to be differentiated from the murmur of aortic stenosis by the predominance of the symptoms of dilatation and hypertrophy of the right ventricle and of disturbances of the pulmonary circulation. A palpable thrill has been recognized to the right of the sternum. *Pulmonary stenosis* is of more frequent occurrence, and is usually associated with a perforated septum, patent ductus arteriosus, and other alterations of the heart, to which attention has been called on page 637. In pulmonary stenosis a systolic souffle is to be expected at the left of the sternum near the third costal cartilage, accompanied by palpable thrill and associated with hypertrophy of the right ventricle.

TREATMENT OF CHRONIC HEART DISEASE.

Although chronic heart diseases vary greatly in the character and seat of the lesion, yet when viewed from a therapeutic stand-point all cases have so much in common that we have thought the treatment could be made most clear by a general discussion under one heading.

There are three conditions of the heart as to power: first, that in which the power is excessive; second, that in which the power is normal; third, that in which the power is below the norm. For the sake of brevity, in this article the heart in which the muscle is excessive is spoken of as hypertrophied; that in which the muscle is weak, as dilated: this entirely independently of the question whether the internal cavities of the heart are or are not increased in size.

Although an altered valve or valve-opening can in no way be restored to its integrity, except in functional insufficiency, yet severe valvular disease may exist for many decades without giving serious inconvenience to its subject; the reason being that there has been such increase in the power of the heart-muscle as to compensate for the valvular disease. Thus, if there be such a leak at a valve as shall require x increase of power to overcome the loss of blood, it is plain that if the heart-muscle has gained exactly x power, little immediate evil will result from the diseased valve: such a heart is spoken of as having undergone "compensatory hypertrophy." If, however, the heart has gained only half x power, although it is absolutely hypertrophied,—*i.e.*, enlarged and increased in power as contrasted with its normal self,—yet it is relatively dilated; that is, it is decreased in power in proportion to the work required of it: such a heart may be spoken of as having undergone "absolute hypertrophy," "relative dilatation." It must, however, be understood that the terms relative and absolute hypertrophy and dilatation are not used, and that when in this article a dilated or weak heart is spoken of the meaning is a heart weak for the work required of it.

The diagnosis of the cardiac condition which is useful for the purposes of the therapeutist has to do not so directly with the nature or seat of the valvular lesion as with the relations between the increase of work and increase of power. The usual clinical diagnosis of the cardiac lesion is chiefly of practical value in assisting in making up the therapeutic diagnosis.

A feeble murmur to the therapeutist is certainly as alarming as a loud murmur, because its feebleness very frequently depends upon lack of propulsive power, whereas a very loud murmur may in part be due to the fact that the blood is driven with force over the diseased valve. The indications of strength in the heart are strength and width of cardiac impulse, loudness and regularity of the heart-sounds, and fulness, force, moderate slowness, and regularity of the pulse. On the other hand, feebleness of the heart-sounds and of the impulse, rapidity, feebleness, and irregularity of the pulse, coldness of the extremities, and especially venous engorgement, are evidences of failing heart-force. Whenever there is a marked tendency to passive congestion of the lungs, to congestion and enlargement of the liver, to dropsical effusions of cardiac origin, heart-power is failing, and, whatever the valvular lesion may be, the treatment is that of cardiac dilatation.

The only resources which we have for the treatment of *cardiac hypertrophy* consist in the avoidance of such violent exercises as shall call the heart into excessive activity, and in the use of veratrum viride and aconite. The action of these two remedies upon the heart is very similar: aconite is, however, more generally useful, because it is less apt to disturb the stomach, and because its influence is somewhat more persistent; from one to two drops of the official tincture may be given three times a day. Larger doses are sometimes required, especially in times of excessive cardiac excitement; although few patients will bear persistently more than three drops a day.

In the treatment of *cardiac dilatation* rest to the heart is of the utmost importance, and must be enforced with a rigor proportionate to the extent of the heart-failure; in severe cases prolonged confinement to bed is essential, the secondary bad effects of the confinement being overcome, if necessary, by the use of massage and electricity; sometimes avoidance even of intellectual excitement is so necessary that isolation and all the procedure of the rest-cure should be insisted upon. (See page 402.) In milder cases of heart-failure it may only be requisite to avoid violent exercise, such as hill-climbing, running up-stairs, etc. The opinion put forth by certain clinicians that a weak heart can be made strong by systematic exercise of the heart we believe to be founded upon the confounding of cases of true heart-weakness with those in which the heart is secondarily weak from the deposition and invasion of its tissues by fat.

It is always important to attend carefully to the diet in a case of cardiac dilatation; usually concentrated nutritious food, with only a moderate amount of farinaceous articles, is the best; but when an active gouty diathesis, or a tendency to obesity, or other indication for the use of a special diet exists, the best results are to be obtained by the use of the diet best fitted for the needs of the system. Tobacco must be forbidden. Coffee is often deleterious. Malt liquors are to be used with great caution.

The drugs which are directly useful in cases of dilatation are caffeine, the nitrites, strophanthus, digitalis, and strychnine. It is essential for the proper use of these drugs that the differences in their physiological actions be thoroughly understood. Caffeine is very feeble and uncertain in its cardiac action, and is never of any value in cardiac dilatation save as an adjuvant to the more positive cardiants; as a diuretic it is the most active of the heart drugs, and is therefore especially useful when there is a tendency to suppression of the renal secretion or to dropsical effusion. The nitrites when in small dose paralyze the inhibitory fibres of the vagi, and probably also stimulate the heart-muscle, so that very violent cardiac movements result; the condition of stimulation, however, passes at once into one of dangerous sedation if the dose be increased beyond a certain point. Moreover, the nitrites, by paralyzing the vessel-walls, and probably also the vaso-motor centres, widen the blood-paths and lower arterial pressure. Evidently their use in chronic cardiac dilatation is very limited. Of the nitrites the amyl salt acts in the course of a few seconds after its exhibition, and releases the system from its influence in a few minutes. It is of no value in heart diseases save in certain brief crises. Nitroglycerin is distinctly less fugacious in its influence; nevertheless, the action of a full therapeutic dose is manifested within two minutes after its ingestion, and ceases in about forty minutes: so that when given for a continuous effect nitroglycerin must be administered at least every hour.

Strophanthus is primarily a muscle-poison, and is useful because the muscle-fibres connected with circulation first feel its action. It is a powerful and very certain stimulant to the heart-walls, and probably also to the coats of the arterioles. It is absorbed rapidly, and acts quickly and persistently, but not with the peculiar permanence of digitalis. It should be given every four to six hours: the dose of the tincture is five to eight minims; of the active principle strophanthin the dose is 0.0002 gramme. In severe cases of cardiac failure strophanthus is less positive and certain in its action than is digitalis. Although it is undoubtedly capable of causing death as a poison, we have never seen other ill effects than gastric irritation, which invariably when an attempt is made to push the remedy in ascending doses soon becomes so severe as to create intolerance.

Digitalis is absorbed very slowly and eliminated still more slowly, so that its influence upon the circulation may continue for days after the cessation of its administration. It acts as a powerful stimulant to the heart-muscle, to the peripheral pneumogastric nerves, to the vaso-motor centres, and to the muscle-fibres in the walls of the arterioles. As the circulation of the heart-wall suffers even more than the general circulation of the body when there is heart-failure, any cardiac drug which increases the activity of the circulation affects most favorably the circulation in the heart-wall, that is, the feeding of the heart-muscle, which in many cases has been at the same time overworked and underfed. Of all known drugs, however, digitalis is the most powerful in its permanent tonic effect upon the heart-nutrition. As has been shown by Gaskell, the period of diastole is that of repair of the heart-structure, and there seems to be no doubt that the pneumogastric nerve has a trophic function, so that in the case of dilated heart digitalis brings more food (blood) to the heart-walls, prolongs the periods of structural upbuilding of the heart-muscle, stimulates the trophic nerve which dominates cardiac nutrition, and by strengthening inhibition quiets nervous irritability. As a diuretic digitalis is inferior to strophanthus.

The only disagreeable effect which is often produced by digitalis is disturbance of digestion. It is certainly true that the drug is capable of a cumulative action, although this is denied by recent authorities. In a case of pleuritic effusion in which digitalis had been given for ten days or more in large doses, no effect was apparent until one morning the pulse dropped from about 100 to 80; the remedy was withdrawn, but the pulse steadily fell until four days after the withdrawal of the digitalis it had reached 40 per minute, and at that rate it remained for several days before it began to mount towards the norm. The cumulative action of digitalis is to be chiefly feared when the drug fails to produce increased diuresis, and has been noted at various times immediately after tapping, for ascites, a patient who has been using digitalis very largely. The poisoning in these cases is without doubt produced by the absorption into the blood of liquid which has been lying in the tissues, the withdrawal of external pressure from the abdominal vessels having brought about increase of lumen and consequent taking in of fluid. Nevertheless, the cumulative action of digitalis is a rare phenomenon, and probably never becomes dangerous provided the drug be withdrawn at the oncoming of the first symptoms.

The slowness of absorption and of elimination of digitalis affects very materially the method of administration. Whenever there is a cardiac crisis and haste is necessary, hypodermic injections of the tincture should be employed. Ordinarily it is best to give the drug at intervals of from four to eight hours. Failure to obtain effects from it may be due to lack of boldness in administration. It should always be given cautiously, the pulse carefully watched, and the remedy withdrawn as soon as the beat falls below 85 (that is, if very large doses are being given) ; no more of the drug being administered until its effects begin to vanish.

There is much evidence in medical literature as to the superiority of the infusion over the tincture, but this apparent superiority rests simply upon the fact that ordinarily the infusion is given in much larger proportional dose than is the tincture. The tincture is ten times the strength of the infusion, so that six minims (ten to twelve drops) are equal to one fluidrachm of the infusion. The dose of the tincture may be set down as from five drops to a fluidrachm, that of the infusion from a fluidrachm to a fluidounce. Digitalin is a complex substance : as kept in the drug-stores it is of various composition, and cannot be relied upon as a representative of digitalis. Digitoxin has been used by some clinicians, but the experiments of Kobert indicate that it does not thoroughly represent digitalis, and we have never employed it.

Strychnine is a valuable drug in cases of heart-failure as an adjuvant to digitalis or strophanthus. Its influence upon the circulation is not, however, comparable to that of the remedies just mentioned, and its effects are never very pronounced. It may be given in much larger doses than those commonly used ; ordinarily in a bad case with cyanosis one-fifteenth or even one-twelfth of a grain every four hours may soon be reached by ascending doses without the production of any symptoms of strychnism.

Cocaine resembles strychnine very closely in its action, and in bad cases it may be well to give it alternately with strychnine in one-quarter-grain doses.

In regard to what may be called the "minor heart drugs," ammonia is sometimes useful in a crisis ; it is, however, such a powerful local irritant that it can scarcely be safely used in sufficient dose to produce distinct immediate effect : most of the restoration of heart-function which is apparent under its influence is really reflex, due to irritation of the peripheral nerves in the nasal or other mucous membrane. At best the influence is so fugacious that it is only in a crisis that the drug is of any use.

Cactus, convallaria, and adonidin have failed us utterly, and seem to have no practical value. Sparteine has been very highly recommended by Pawinski and Clarke as having especial power in controlling nervous palpitation, even in Graves's disease. The official sulphate may be given in doses of one-fourth grain, increased cautiously to two grains, if necessary, every six to eight hours. Our experience with it has not been very favorable.

As adjuvants to the more powerful remedies, alcohol, ether, or Hoffmann's anodyne may be used freely in cases of sudden heart-failure. They may be given hypodermically, but produce much local irritation, and are so readily absorbed from the stomach that it is usually preferable to give them by the mouth. In Germany camphor is very much used as a rapidly acting heart-stimulant by subcutaneous administration; the ten per cent. solution in olive oil of the German Pharmacopœia may be injected in doses of one-half to one fluidrachm, or a solution of camphor in ether may be employed.

Success in the treatment of heart disease depends largely upon the management of the secondary conditions produced by the cardiac lesion. The portal circulation very frequently becomes the seat of excessive congestion, which may manifest itself chiefly in derangement of the function; with it there may be a pronounced increase in the size of the liver. Under these circumstances there is no remedy of equal rank with the mercurials. The vegetable cathartics, which are alleged to have active cholagogue power, are of very little value. Sodium phosphate, especially the mixture of sodium sulphate and sodium phosphate with potassium iodide (see formula 19), is often of service. Nitrohydrochloric acid is sometimes useful. All these drugs, however, are entirely secondary to the mercurials. Very frequently purgative doses of calomel bring the greatest relief; it may even be that the calomel is followed by a favorable action of the digitalis which had previously been without avail. Perhaps more often the continuous use of small doses of corrosive sublimate (one-twentieth to one-eightieth of a grain) has a very happy effect. In many cardiac cases iron is indicated for the relief of the secondary anæmia; under such circumstances usually the tincture of ferric chloride with corrosive sublimate gives the best result.

Treatment of Special Conditions.

Early Weeks of Valvular Disease.—It is especially important when an acute endocarditis is passing into valvular lesion that great care be exercised to favor the development of compensatory hypertrophy. Excessive hypertrophy is a very rare phenomenon when the valvular lesion has been rapidly induced by an endocarditis. Rest, fresh air, carefully graded exercise, high feeding, and the habitual use of digitalis in small doses are the means at our disposal for increasing the working power of the heart which has been damaged by acute inflammation. Digitalis is in these cases much superior to strophanthus, on account of the greater trophic influence which it exerts upon the heart. There are practitioners who condemn the use of digitalis in the early or formative stage of cardiac valvular disease. Nevertheless, its power for good is in many cases at this time greater than at any other, and it is certainly when properly used incapable of harm in most cases in which the valvular lesion has been produced by an endocarditis. It is so rare for compensation in such cases to become excessive that the danger of cardiac hypertrophy may be considered almost nothing; if, however, there should be signs of excessive cardiac growth, digitalis should be omitted. In no instance should it be given in such dose as to produce an habitual pulse of under 75. When under care compensatory hypertrophy has been obtained, or when in any case an organic cardiac murmur associated with compensatory hypertrophy is unexpectedly discovered, the administration of heart tonics should be avoided, unless at intervals when temporary derangement of the health brings the circulation below the normal level.

The question whether an individual should be told that he has a heart disease or not is often of somewhat difficult solution: probably in the majority of cases it is better to give a careful warning, along with the statement that cases of heart disease often live comfortably for decades of years, because in no other way can the patient be led to the well-regulated life that is necessary for his continuous good. Under these circumstances violent muscular exercise should be interdicted; care should be exercised in allowing the patient to go into high altitudes; daily tepid or cold baths should be taken, the Turkish and very hot baths being avoided as dangerous if there is distinct heart-weakness. Tobacco is not allowed; alcohol should be used very moderately, if at all.

Acute Cardiac Dilatation.—An acute attack of cardiac dilatation or failure, if of severe type, may be due to the blocking up of a branch of the coronary artery. If the branch is a large one, a fatal result is inevitable; if it is small, recovery may occur, though the symptoms at first may be very severe. Severe attacks of sudden cardiac failure may occur in valvular disease with or without apparent cause; they are to be recognized by the heart-pangs, shortness of breath, irregular forced respiration, rapid feeble broken pulse, and evidences of venous congestion. When these symptoms develop with great rapidity, when signs of venous engorgement are pronounced, and orthopnœa exists with cyanosis, the taking of twenty to thirty ounces of blood by venesection has long been recommended by standard authorities. We believe this teaching to be correct, but it is plain that such venesection cannot be repeated, and that its use must be reserved for crises in which the dilated feeble heart is so close to the point of giving up the struggle to move the large amount of blood in the body that the effect of the more usual treatment cannot be waited for. (See page 675.) In some cases, especially when there is a tendency to dropsical effusion, it is possible to reduce the volume of blood sufficiently by rapid free purgation. The ordinary treatment of acute heart-failure with backing up of the blood in the pulmonary vessels, resulting in pulmonic œdema with frothy bloody expectoration, may be summed up as follows: first, the use of absolute rest; second, the administration subcutaneously of small doses of morphine and atropine to quiet the nervous system; third, the free administration of alcoholic stimulants by the mouth, the guarded use of nitroglycerin, which dilates the general blood-vessels of the body and probably lessens the amount of work necessary for the left ventricle, the hypodermic injection of strychnine, the inhalation of oxygen gas, which may increase oxidation in the blood and certainly does not strongly support the patient morally, and, finally, the free use of digitalis. Owing to the ease with which the stimulating action of nitroglycerin passes into cardiac sedation, large doses of nitroglycerin are very dangerous. Of all the drugs digitalis is the most efficacious: it must be used in large doses. From twenty to thirty minims of the tincture may be given every two hours if required; in a crisis thirty minims of the tincture may be given immediately hypodermically: the danger of local effects is lessened by not injecting more than fifteen minims in one place.

In the advanced stages of chronic cardiac disease, when the heart seems entirely unable to do its work, relief will often be afforded by the use of a drachm or more of tincture of digitalis every twenty-four hours, the remedy being withheld for one or two days each week to prevent accumulation, and the dose lowered whenever it can be done without suffering. It should be remembered that these large doses are justifiable only when life cannot otherwise be made comfortable. We have frequently seen patients who for many weeks had suffered all the horrors of orthopnœa, notwithstanding various treatment, including the moderate use of digitalis, put upon their feet by massive doses of the drug and even enabled to resume their vocations. It is essential, however, to diminish the dose as soon as possible; in our experience, patients who have been kept going by the use of these very large doses of digitalis have in the end almost invariably dropped dead. It is not probable that these sudden deaths were due to any direct action of the drug upon the heart; they were probably the result of the use by the heart of all the forces within its power. Such cases are frequently paralleled in heart diseases where the patient has remained comfortable and suddenly expired without digitalis having been given. In any event, in our opinion, months of ease with sudden death at the end are to be preferred to a probable shorter period of excessive suffering.

Valvular Lesions.—The treatment of all forms of valvular lesions is simply that of the accompanying muscle condition. The seeming truth in the old teaching that digitalis is not useful in diseases of the aortic orifice is due to the fact that aortic disease is in the majority of cases of gradual development, and is, therefore, very prone to be accompanied with completely compensatory or excessive hypertrophy. Again, digitalis is most satisfactory in mitral insufficiency because in these cases the opening in the closed valves is apt to be small, irregular, and much choked up by vegetations or fimbriations. If under such circumstances by the use of digitalis the amount of blood thrown out at a single stroke and the power of ejection are simultaneously increased, friction increases at the rough, narrow chink in the closed mitral valve much more rapidly than it does at the wide, open, smooth, aortic orifice, so that a much smaller percentage of blood flows back through the valve than was the case before the exhibition of the digitalis. Cases of mitral insufficiency are occasionally met with in which the heart-muscle is distinctly feeble and compensation failing, but in which digitalis in full dose increases very greatly the heart-pang and general distress. In most, if not all, of these cases it will be found that the left auricle is excessively dilated and is unable to withstand the increased strain thrown upon it by the forcible regurgitant flow under the use of digitalis. In all the cases of this character which we have seen the result has proved that the situation was from the first desperate and without remedy.

Treatment of Symptoms.

Cardiac palpitation and distress are usually best controlled by appropriate cardiants: in excessive hypertrophy with throbbing, aconite; in the distressing pain in heart-failure, digitalis; when with the pain there is high tension, as when arterio-sclerosis coexists with cardiac disease, nitroglycerin is often most serviceable. The local application of ice-bags and of various counter-irritants, such as mustard plasters and flying blisters, sometimes gives relief. Hoffmann's anodyne is temporarily efficient; a teaspoonful of it should be given in a wineglass of ice-cold water. Hypodermic injections of morphine, with atropine, are most valuable and efficacious subduers of heart-pain. (See also Angina Pectoris, page 684.) Insomnia and the horrible night restlessness which sometimes occurs in heart disease are to be met by the cautious use of potassium bromide, of trional, of sulphonal, of chloralamide, of paraldehyde, and of other of the minor somnifacients. An alcoholic potation, especially if combined with camphor, sometimes suffices. Chloral is very effective in producing sleep, but is certainly dangerous in full dose when there is cardiac weakness, and if used habitually is apt to weaken still further the force of the circulation: it should therefore usually be avoided. Cough and hæmoptysis may ordinarily be left to nature, but if they become severe should be controlled in the ordinary method; a moderate hæmoptysis is in many cases beneficial, by temporarily relieving pulmonic engorgement. Indigestion and vomiting are usually to be treated by remedying the hepatic and general portal congestion. Bitter tonics are rarely of service. When the vomiting is severe, creosote, hydrocyanic acid, bismuth, cerium oxalate, cocaine, and the other well-known anti-emetic remedies may be tried. Very often the chief reliance must be upon opium suppositories. Excessive cardiac dyspnœa or orthopnœa, being the direct result of failing heart-power, is to be chiefly combated by the use of cardiac tonics and stimulants, aided, if the lungs be violently congested, by the use of dry cups or other forms of counter-irritation. Strychnine and cocaine are indicated ; the inhalation of oxygen gas rarely, if ever, does any good.

For getting rid of dropsical effusions in cardiac cases, purgatives, diaphoretics, or diuretics are employed ; of these the first named are the most efficient, but they are very exhausting to the patient. The choice of purgatives lies between the salines, notably sodium or magnesium sulphate or potassium and sodium tartrate ; the last of these is the most pleasant to take, but is the least powerful. Salines, if used at all, are best given in concentrated solution. Compound jalap powder (thirty grains) is a very effective hydragogue cathartic, which may, however, be made more serviceable by the addition of two drachms of cream of tartar to the dose. Elaterium acts very kindly if given with extract of belladonna (one-sixth of a grain of each) and repeated every four to six hours as necessary. Of these purgatives we have preferred the last ; other practitioners habitually use the salines ; either remedy may be employed.

Diaphoretics are not usually effective in cardiac dropsy. The hot bath, whether in the form of the vapor bath (Russian), or the dry-air bath (Turkish), or the simple hot-water bath, is dangerous, and should be used cautiously or be altogether avoided if the heart is very weak.

Diuretics often act happily, provided the circulation of the kidneys can be, in a measure, restored by cardiac tonics. Of these cardiants strophanthus is very active as a diuretic, but is as such inferior to caffeine, whose heart-action is, however, very feeble. Potassium bitartrate, an ounce a day in a pint of infusion of juniper or of water containing two or three ounces of the compound spirit of juniper, is the most active of the sedative diuretics, soothing rather than irritating to the kidneys. Sodium and theobromine salicylate, and pilocarpine hydrochlorate (one-twentieth of a grain every two hours), are active diuretics which do not irritate the kidneys. Scoparius in full dose is one of the most certain of its class, but is somewhat irritant ; it is best given in the form of the decoction, made by boiling for ten minutes one ounce of the dried broom-tops with a pint of water and straining ; dose, one-half pint to a pint during twenty-four hours. Squill is irritating to the kidneys, but very useful in many cases of cardiac dropsy, especially in the old pill of one grain each of calomel, squill, and digitalis, taken three or four times a day. The Jendrassik method of using calomel may succeed after other remedies have failed : five grains of calomel are to be exhibited every hour until fifteen are taken, or three grains of the calomel may be given every four hours until free diuresis is obtained or severe intestinal disturbance is induced.

In some cases of cardiac dropsy relief may be obtained by mechanical means. Thus, œdematous legs may be bandaged with canton flannel, or, if the weather be cold, in wool flannel. Paracentesis abdominalis is rarely required: puncturing the legs, the introduction of Southey's tubes, and the making of moon-shaped incisions below the internal malleoli, are procedures which may be forced upon the practitioner, but are to be put off as long as possible: strict antiseptic precautions should always be taken in their performance.

CARDIAC NEUROSES.

PALPITATION.

By this term is understood an increased beat of the heart, producing sensations disagreeable to the patient. Palpitation occurs in healthy persons of nervous temperament as well as in sufferers from disease of the heart, in whom it is one of the serious symptoms of failing compensation. Palpitation as a cardiac neurosis is more common in women than in men, and is especially likely to be excited at the age of puberty, during the climacteric, or in connection with disturbances of menstruation. The prolonged use of tea, coffee, tobacco, or alcohol, mental overwork, sexual excesses, digestive disturbance, pelvic disease, exophthalmic goitre, chlorosis and anæmia, and prostration during convalescence from severe acute disease, act as predisposing causes. The immediate paroxysm may be produced by mental or physical excitement, by an attack of indigestion, or without any obvious excitant. Palpitation occurs at irregular intervals and lasts for a longer or shorter time. The accelerated beating of the heart is usually apparent on inspection of the carotids and of the cardiac region, but the action of the heart, though rapid, may be feeble, and the patient complain of sensations of faintness. The increased pulsations are often associated with a feeling of substernal oppression, perhaps of choking. The face is generally flushed, though sometimes pale, and the expression is anxious. On examination of the heart during the attack the sounds, especially the second, though usually accentuated, are sometimes feeble, and systolic murmurs may be heard at the base. Da Costa has called attention to recurrent attacks of palpitation and præcordial pain, particularly among soldiers, but also to be found among other persons. He applies the term *irritable heart* to this condition, attributing the attacks to strain and overaction of the heart, and states that among soldiers they are especially likely to follow fatiguing marches, and that permanent dilatation may result. Palpitation independent of organic disease, though disagreeable, is rarely dangerous.

TREATMENT.—In the treatment of palpitation of the heart not due to disease of the viscus, it is essential first to discover, and then, if possible, to remove, the cause. When this has been done, in a large proportion of cases no further difficulty is experienced, so that the palpitation does not require any direct treatment. If, however, at any time there is distress with the palpitation, camphor or Hoffmann's anodyne may be given in full dose, and in some cases the persistent administration of small doses of tincture of aconite or of tincture of digitalis will be found useful. Theoretically the choice between these two remedies is to be made in accordance with the condition of the heart as to power; but practically in many cases the final selection must be the result of trial, that medicament being selected which affects most satisfactorily the individual case.

In irritable heart the avoidance of all exertion or emotion that would provoke cardiac excitement should be the basis of the treatment. The patient should be made to understand that the affection is serious and may lead to permanent alteration of structure, so that the temporary inconvenience of prolonged rest shall be willingly endured. In perhaps the majority of these cases the best results are to be obtained from the continuous administration of aconite; but when there is any tendency to failure of heart-power digitalis should be used in such doses as may be required.

TACHYCARDIA.

Rapid beating of the heart is usually considered to be the result of a disturbance of innervation of the cardiac ganglia, attributable either to disease of the ganglia themselves or to paralysis of the pneumogastric or irritation of the sympathetic nerves. Martius considers that tachycardia is due to a sudden dilatation of the heart, which is frequently associated, the pulsations being quickened that the heart may be emptied. The immediate causes of tachycardia are the same as those productive of palpitation, and in addition organic disease at the origin or in the course of the pneumogastric nerve is important. Like palpitation, its occurrence in disease of the heart belongs to the stage of failing compensation.

In *paroxysmal tachycardia* attacks of rapid beating of the heart, the pulse rising perhaps above two hundred beats, continue to recur for an hour or more at frequent intervals during a period of weeks, months, or years, the patient being free from disturbances in the mean time. The attacks are accompanied by nausea, prostration, and anxiety, and are sometimes relieved by belching of gas or by an evacuation of the bowels. H. C. Wood reports the case of a physician eighty-seven years old in whom violent attacks had frequently recurred for fifty years without causing any disturbance of health or interfering with a very active life. Paroxysmal tachycardia must be considered to be a distinct neurosis whose pathology is at present inexplicable; though persistence of the attacks may result in permanent dilatation. In the case above mentioned the paroxysms could be immediately aborted by rapidly drinking a tumbler of very cold ice-water. No known treatment has any effect upon the recurrence of the attacks.

BRADYCARDIA.

Bradycardia, or slow beating of the heart, is attributed to irritation of the pneumogastric nerve, or to paralysis of the branches of the sympathetic nerve which accelerate the action of the heart, or to irritation or paralysis of the nerve-centres within the heart. It is more common in men than in women, and in adults than in children. A physiological is to be distinguished from a pathological bradycardia. The former (normal bradycardia) is found in certain people, and often in old age. In like manner the frequent occurrence of bradycardia after delivery is hardly to be regarded as pathological, and during convalescence from fever it is on the border-line between the physiological and the pathological variety.

Bradycardia as a symptom of disease may be due to a variety of lesions affecting the nervous system and the heart, or to a toxæmia. Among the diseases of the nervous system are meningitis, hemorrhages, intra-cranial tumors, affections of the cervical portion of the spinal cord, mania, melancholia, and paralytic dementia. More often the lesions are present in the heart, and include sclerosis of the coronary arteries, chronic myocarditis, fatty degeneration of the heart, and aortic and mitral disease. According to Dehio, the persistence of bradycardia despite the use of atropine is evidence of its cardiac origin. The toxic causes are tea, coffee, tobacco, alcohol, opium, digitalis. carbonic acid gas, lead, the poisons present in uræmia and diabetes, and the bile-acids in jaundice. The bradycardia in convalescence from acute infectious diseases, as typhoid fever, acute rheumatic arthritis, pneumonia, diphtheria, erysipelas, malaria, and in diseases of the digestive tract, as ulcer or cancer of the stomach, has been attributed both to toxæmia and to exhaustion. In like manner its occurrence in starvation is a result of prolonged exhaustion. In normal bradycardia the pulse-rate rarely falls below forty, but in abnormal bradycardia the beat may be less than ten strokes. According to the degree of slowness of the pulse and the corresponding quantity of blood distributed at each systole are the character and frequency of associated symptoms. These usually occur in paroxysms induced by mental or physical excitement, perhaps by acute indigestion, and the attacks continue for several minutes or for a half-hour or more. Cerebral anæmia often follows, manifested by fainting, loss of consciousness. or epileptiform convulsions. Indeed, so-called *senile epilepsy* is a frequent result of paroxysms of bradycardia in elderly persons with sclerosis of the coronary arteries or fibrous myocarditis. The respiration may be simultaneously diminished, and sometimes assumes a Cheyne-Stokes character. When the attack is recovered from, a considerable degree of exhaustion ensues. Bradycardia is of grave prognostic importance when due to disease of the heart or brain, although this symptom may be continued over a period of several years. Moreover, as in several cases reported by the elder Flint, bradycardia may end in death, and a careful post-mortem examination may fail to detect any cause. These are probably diseases of the cardiac ganglia entirely beyond our ken. When due to cardiac disease, dilatation is the uniform result of the disturbed action of the heart. If death ensues, it may be sudden or gradual from cardiac insufficiency.

TREATMENT.—Whenever a cause can be assigned for an existing bradycardia it should be removed, if possible; further than this the treatment is very unsatisfactory. Whenever there is any feebleness of the heart, rest and total avoidance of heart-strain should be rigidly enforced, and under such circumstances strophanthus may be very carefully tried. In those cases which may be for the present denominated "essential bradycardia," in which no explanation of the symptoms can be made out during life or detected after death, there is no known remedy. For the slowness of the pulse-beat due to pneumogastric irritation large doses of atropine may be indicated; but in a case in which the pulse fell to four a minute, and in which on three or four occasions the patient was pronounced dead, neither belladonna nor any other known cardiant had the slightest apparent influence, although given very freely: finally, under rest, recovery of the heart-rate occurred.

ARHYTHMIA.

Alterations of the rhythm of the heart are considered to result from faulty innervation, and may be found in persons otherwise normal as well as in those diseased. The pulse of defective rhythm is usually spoken of as intermittent or irregular. Baumgarten divides the causes of irregularity into those originating in the brain or heart and those of reflex origin. The intra-cranial causes are cerebral concussion, hemorrhage, softening, abscess, meningitis, and psychical disturbances. Those proceeding from the heart are the result of disturbed nutrition or of the action of poisons. The disturbances of nutrition occur in fevers, wasting diseases, coronary sclerosis, fibrous myocarditis, valvular disease, and cardiac strain. The abuse of tea, coffee, tobacco, and alcohol, and the action of various drugs, especially digitalis, belladonna, and aconite, are among the toxic causes, and the reflex causes are to be found in blows upon the abdomen, acute and chronic digestive disturbances, and diseases of the kidney. Numerous variations in the irregularity of rhythm are to be recognized. In the *paradoxical pulse* of Kussmaul the pulse is accelerated and less full during inspiration than in expiration, sometimes stopping at the end of prolonged inspiration. This variety occurs not only in fibrous pericarditis and mediastinitis, but also when there is abundant exudation, and in mediastinal tumor and in obstruction of the air-passages. It may be observed in sleeping children. In the *intermittent* pulse the beats of the heart are not transmitted to the wrist, and in the *deficient* pulse the heart occasionally fails to contract, and thus a beat is lacking from time to time. An *alternating* pulse is present when the volume of every other beat is full or diminished. In the *bigeminal* and *trigeminal* pulses every second or every third beat fails to reach the wrist, hence every two or every three beats are separated by an abnormally long interval. In *delirium cordis* the beat of the heart is wholly irregular in force, in frequency, and in the interval between the beats.

Occasional irregularity of the pulse is less indicative of a serious disturbance than is persistent irregularity; when occurring in infants or elderly people it is of no necessary pathological significance, marked degrees of irregularity being compatible with prolonged and active life. Even in cardiac disease the arhythmical pulse may be present at a time when there is satisfactory compensation, though usually it is a sign of failing compensation. The extreme irregularity of delirium cordis, however, occurs in the later stages of failing compensation, and is a grave prognostic sign, since it not infrequently ends in sudden death. Cardiac arhythmia calls for no treatment other than that of the condition upon which it depends.

ANGINA PECTORIS. NEURALGIA OF THE HEART.

Definition.—A paroxysm of intense pain in the region of the heart, associated with a sense of impending death.

The term angina pectoris applies rather to a group of symptoms than to a definite disease, usually occurring after middle life, and more frequently among men than among women. The most frequent cause is sclerosis of the coronary artery, but it may result from chronic aortic stenosis or insufficiency, aneurism of the arch of the aorta, fatty degeneration of the heart, or chronic adhesive pericarditis. A distinction is drawn between true angina and pseudo-angina, the latter occurring more frequently in young adults of nervous temperament, especially in the neurasthenic and hysterical, although the causes of the immediate attack may be the same as in true angina. The attacks of pseudo-angina are also occasioned by the abuse of tea, coffee, or tobacco, and by lead poisoning, and are more likely to occur in persons suffering from prolonged disturbance of digestion. Nothnagel has applied the term *vaso-motor angina* to attacks of substernal constriction and pain referred to the heart, associated with a pale, bluish-gray color of the cool skin and a sense of muscular stiffness. This condition is assumed to be the result of a spasm of the peripheral arteries, and is apparently simply a variety of pseudo-angina in which vaso-motor disturbances are conspicuous.

Symptoms.—The severer attacks of angina pectoris are usually preceded, perhaps for years, by what are essentially milder attacks,—namely, a sense of oppression beneath the sternum, slight pain at the apex, and some shortness of breath on slight exertion. Such discomforts are induced by mental or physical excitement or by indulgence in alcohol or tobacco, or follow slight disturbance of digestion. These milder attacks of angina gradually increase in severity until typical paroxysms occur.

Under the effect of similar causes, sudden, unexpected, often intense pain arises, referred to the region of the heart and associated with a sense of oppression in the midsternal region. The pain is primarily referred to the cardiac plexus of nerves, from which it radiates frequently into the left shoulder, neck, and arm, in the latter often following the course of the ulnar nerve to the fingers, which, as well as the arm, are perhaps benumbed. More rarely the pain extends into the right arm or shoots up and down the chest, sometimes extending to the lower extremities. During the attack there are great anxiety and fear of impending death. The patient grasps a support if within reach, and remains stationary, fearing to draw a long breath, although there is no real dyspnœa. The face is pale, the skin is moist, and the pulse is usually increased in frequency, weak and irregular, but the second aortic sound may be of increased tension. The attack rarely lasts longer than a minute or two, although it may be frequently repeated during a series of hours. The patient may feel faint or lose consciousness, and the paroxysm not infrequently ends with belching, vomiting, or an evacuation of the bowels. In the course of a few minutes after the pain has disappeared the patient may feel as well as usual, or hours of exhaustion may follow. Although in general the physical examination of the heart is negative, dilatation, especially of the left ventricle, is likely to occur if the angina is continued over a period of years. The symptoms of obstruction of the pulmonary circulation then are manifested, and the attacks of angina may be associated with the signs of acute insufficiency of the left ventricle,—namely, dyspnœa and a frothy, bloody sputum from extreme congestion of the lungs.

Diagnosis.—The symptoms of angina are sufficiently characteristic, but their significance largely depends upon the evidence of associated organic disease of the heart, of its vessels, or of the aorta. True angina occurs more frequently among men beyond middle life in consequence of unusual mental excitement or physical exertion, is of short duration, and at times is associated with a pulse of increased tension from arteriosclerosis. Pseudo-angina takes place especially at night, and chiefly among neurasthenic or hysterical young women or men suffering from dyspepsia. The attacks last for a half-hour or more, pain and fear of death being less conspicuous than a sense of constriction and palpitation, and end with the frequent desire for micturition. The cases of especially doubtful diagnosis are those of pseudo-angina with evidence of cardiac disease, when it may be necessary to reserve one's opinion until treatment has made the distinction clear between the true angina, dependent upon organic disease, and the pseudo-angina, which practically is an exaggerated form of nervous palpitation.

Prognosis.—The prognosis of true angina, whether mild or severe, is always serious, since progressive organic lesions are the usual cause, and increasing dilatation, perhaps myomalacia, of the left ventricle, is the result. Mild attacks may suddenly terminate fatally from thrombotic or embolic obliteration of the coronary artery, from rupture of the heart, or without any obvious anatomical lesion. The pathological importance of obstruction of the coronary artery is demonstrated by the experimental production of complete closure of the left coronary artery, the effect of which is paralysis of the left ventricle, followed by dilatation and immediate death, or by rapid breathing, congestion, and œdema of the lungs. On the other hand, attacks of angina associated with cardiac lesions may continue for many years, and the patient with suitable care and proper treatment may live to a good old age. The prognosis in the individual case is the better provided there is no evidence of disease of the aorta, of the aortic valves, or of the myocardium, and the pulse is regular and without high tension. The prognosis is also more favorable the fewer and the milder the attacks, especially when the patient is able largely to control his surroundings.

TREATMENT.—The energy of treatment in heart-pang should always vary with the severity of the symptoms. During a mild paroxysm large sinapisms over the cardiac region may be used, and even in the severest attacks are commonly employed along with mustard foot-baths, though there is no reason for believing that they affect the condition. The inhalation of amyl nitrite will frequently cut short an attack. Nitroglycerin is equivalent to amyl nitrite, except that it is not quite so prompt and is more lasting in its effects. The only other effective anodyne remedy is morphine given hypodermically, one-fourth to one-half grain; with it should be associated atropine (one-hundred-and-fiftieth to one-hundredth of a grain), and in cases of weak heart strychnine (one-twentieth of a grain). When the heart is extremely feeble the nitrites must be used with great caution, but in many cases the patient should be taught to carry amyl nitrite or a solution of nitroglycerin—spiritus glonoini—with him, in a small vial containing a single dose, or, in the case of the amyl salt, in glass pearls which can be broken and their contents immediately inhaled. Nitroglycerin tablets also may be used. Authorities advise the administration of two or three doses of nitroglycerin daily as a preventive of attacks: if, however, nitroglycerin is used to ward off attacks it should be given not less frequently than every two hours.

The treatment between the paroxysms should be directed against the underlying cardiac condition: the administration of silver nitrate, zinc sulphate, potassium bromide, and various other alleged nervines, as advised in some text-books, is the outcome of despair.

Violent exercise, smoking, the abuse of alcohol, and all excesses whatever must be sedulously avoided. Emotional storms, such as a fit of anger or the excitement of marital or irregular coitus, have in many cases precipitated a fatal attack. A steep ascent, a late arrival at a railway train, a rush to a trolley-car, may end in sudden death. Very dangerous is the attempt to walk against a cold high wind, as in the case of a noted

Philadelphia preacher, who, leaving his pulpit, briskly started northwest in the face of the wind, grasped a hand-railing, and was dead.

In hysterical or neurasthenic pseudo-angina it is very important that the patient should be made to understand that the attacks are not angina. Hoffmann's anodyne and asafetida will sometimes suffice; but not rarely morphine is imperative, notwithstanding the danger of the narcotic habit. Between the paroxysms the neurasthenic condition should be very carefully treated.

CHAPTER IV.

DISEASES OF THE ARTERIES.

ARTERIO-SCLEROSIS.

DEFINITION.—An affection of the arteries characterized by circumscribed or diffuse thickening of the intima and by degenerative or inflammatory changes in the media and sometimes in the adventitia.

The term arterio-sclerosis was introduced by Lobstein to indicate the alterations frequently found in the larger arteries, especially in the aorta. Later, one feature of the alterations, the formation of a pap-like material, *atheroma,* was made prominent, and the entire process was designated atheromatous degeneration. Virchow then applied the term *endarteriitis chronica nodosa sive deformans* to the arterial changes, but Gull and Sutton made it conspicuous that the analogous alterations of the blood-vessels of the kidney in chronic fibrous nephritis were a part of a general affection of the blood-vessels, to which they applied the term arterio-capillary fibrosis. Although at present there exists a considerable difference of opinion as to the unifying of all the chronic arterial changes that are found in advancing years under one head, the intimacy of relation between them is not to be denied; they are usually regarded as manifestations of arterio-sclerosis.

ETIOLOGY.—Evidences of arterio-sclerosis are so often found after middle life that its absence in men beyond the age of fifty is exceptional. Its occurrence in women, however, is far less frequent. Heredity is generally considered as an important remote cause, although the immediate causes are far more significant. Among the latter are to be recognized over-eating, the excessive use of alcoholic drinks. lead poisoning, sedentary habits, repeated extreme stretching of the walls of the arteries in laborious occupations, and the factors of importance in the etiology of gout, chronic rheumatism, and diabetes. The relation between chronic fibrous nephritis and arterio-sclerosis admits of a threefold interpretation: first, that both the nephritis and the arterio-sclerosis are the results of the same cause; second, that the nephritis causes a toxæmia from the insufficient elimination of the products of tissue metamorphosis, which act as a cause of the arterio-sclerosis; and third, that the arterio-sclerosis causes the nephritis. Syphilis is the most frequent etiological factor in what may be called premature arterio-sclerosis,—that is, when occurring previous to the age of forty. Tuberculosis also is a frequent occasion of localized arterio-sclerosis, especially in the arteries of the pia mater, and of late years etiological importance has repeatedly been assigned to acute infectious diseases.

MORBID ANATOMY.—The alterations occurring in arterio-sclerosis are either circumscribed or diffuse. The former are especially found in the largest arteries or in the immediate vicinity of localized lesions affecting the small arteries. The diffuse changes are present in both the large and the small arteries, and it is this variety in particular that is especially important in human pathology. The gross appearances of circumscribed arterio-sclerosis are seen to best advantage in the aorta in the condition called by Virchow nodular endaortitis, by Councilman nodular arterio-sclerosis. Circumscribed, rounded, slightly elevated, grayish-white or opaque white patches project from the intima, of which they represent a localized thickening, and increase in density and opacity in the course of time. The patches may be few or many, and when abundant tend to become confluent, thus involving a large surface. They are frequently found at the origin of both large and small branches of the aorta, and often produce narrowing of the orifices, especially of the coronary, renal, intercostal, and lumbar arteries. These patches, the arterio-sclerotic plates or nodules, are due to a proliferation of the cells of the intima, the result, according to Virchow, of an inflammation of the deeper layers, but, as held by Köster, of a primary inflammatory process proceeding from the vasa vasorum of the media and adventitia. Thoma considers that the weakness of the middle coat which is so marked a feature in this disease is rather a cause than a result, and that the wall yields first, thus leaving a space, occupied in the distended condition of the artery by the sclerotic plates. The latter thus represent a compensatory hypertrophy of the intima serving to retain the normal calibre of the vessel to a greater or less degree.

These "pseudo-cartilaginous plates" undergo retrograde changes, being transformed sometimes into atheromatous patches, abscesses, and ulcers, at other times into calcareous plates. A necrosis of the cells, at first in the deeper layers of the thickened intima, takes place, and there results a granular detritus of an opaque yellow color and of soft pap-like consistency in which fat-drops and crystals of cholesterin are found composing the atheromatous material. If the degenerative changes extend to the surface, the superficial layer of endothelium gives way, and the blood-current removes more or less of the atheromatous material from the cavity, the wall of which not infrequently serves as a place of origin for thrombi. The deposition of lime salts frequently takes place both in the arterio-sclerotic patch and in the necrotic tissue. Thus are produced the osteoid plates, the surface of which is smooth towards the arterial canal, rough, perhaps jagged, towards the middle coat. Usually these various manifestations of the arterio-sclerotic process are combined, and are associated with such loss of elasticity of the middle coat that the aorta is dilated and in extreme instances somewhat convoluted.

In diffuse arterio-sclerosis the alterations of the larger vessels are essentially the same as those already described,—namely, cellular hyperplasia of the intima and degenerative changes with loss of elasticity and contractility in the media, the tendency being rather to extensive alterations of the wall than to circumscribed lesions. More important is the occurrence of an endarteritis in the smaller arteries, especially in those of the brain, heart, and kidneys. Councilman states that it is present also in the liver, in consequence of which a marked increase in density results. A like alteration of the blood-vessels in the brain was first described by Friedländer as an obliterative endarteritis, and was attributed by him to syphilis, but similar changes are to be found there and elsewhere in consequence of tuberculosis, and in connection with fibrous tissue formation from various causes, notably in the so-called organization of a thrombus. The more extensive this diffuse endarteritis the greater the destruction of the organ especially affected; but the question still remains open whether parenchymatous destruction precedes and causes or follows and results from the arterial changes. In diffuse endarteritis necrosis and calcification in the thickened intima do not occur in the smaller vessels, but are limited to arteries of the size of the radial.

There exists a considerable difference of opinion as to the relation between tortuous arteries and those altered in consequence of arterio-sclerosis. The splenic artery, the iliac artery, and others as small as the temporal arteries, not infrequently become elongated and varicose in advancing years, the *serpentine aneurism;* but such abnormality of the visible external arteries is no evidence of the existence of arterio-sclerosis of the aorta or of its branches. In the larger arteries a deposition of lime salts often occurs in the muscular media, in consequence of which it becomes transversely ribbed. A similar infiltration of lime salts may take place in the arteries of the extremities, transforming them into rigid tubes, often compared to pipe-stems. Such changes are generally considered as degenerative, and in no way necessarily connected with the proliferative changes in arterio-sclerosis. They may be combined with the latter, however, and the tortuous splenic artery may show circumscribed sacculated dilatations due to endarteritis, the dilated portions being diffusely infiltrated with lime salts as in the case of the calcified plates of the aorta. In arterio-sclerosis, especially of the coronary arteries of the heart and of the arteries of the extremities, the secondary deposition of lime salts may cause rigid tubes as in calcification of the media, but the earthy salts are deposited rather in the intima than in the middle coat.

Hypertrophy of the heart, especially of the left ventricle, is constantly associated with arterio-sclerosis. Some pathologists believe that the loss of arterial elasticity and contractility calls for increased work on the part of the heart to drive the blood to the periphery of the body; others think that a primary hypertrophy of the heart causes stretching and weakening of the media and thickening of the intima, tending to restore the previous calibre of the artery. The appearances of the heart are those either of idiopathic hypertrophy or, in case there is sclerosis of the coronary arteries, of hypertrophy combined with fibrous myocarditis and with dilatation. Indeed, as already stated, the common cause of fibrous myocarditis is sclerosis of the coronary arteries.

SYMPTOMS.—Nodular arterio-sclerosis of the aorta is not usually productive of symptoms. Sometimes the dilatation of the arch is sufficient to be manifested by an increased area of dulness at the right and upper portion of the sternum and by visible or palpable pulsation at the sternal notch. In such cases an inequality of the pulses recognizable at the wrist may be due to the narrowing of the orifice of an innominate artery. It is also possible that the muscular pains and weakness of the trunk so often complained of by elderly people may be the result of interference with the circulation through the intercostal and lumbar arteries in consequence of a narrowing of their orifices by sclerotic patches. If the sclerotic plates are situated at the origin of the coronary or renal arteries the nutrition of the heart or of the kidneys is likely to be disturbed, and brown atrophy of the heart or simple atrophy of the kidney result. The slowly progressing enfeeblement of the heart, the transitory and slight albuminuria, even when associated with an occasional hyaline cast, in old people, thus meet with a satisfactory explanation.

Diffuse arterio-sclerosis not only causes hypertrophy of the heart to overcome the obstruction, but also interferes with the nutrition of the organs especially concerned. The gradual progress of the cardiac hypertrophy is compensatory, and symptoms of arterio-sclerosis are unlikely to arise until the heart presents signs of failing compensation or localizing symptoms call attention to the brain or kidneys. During this period of progressing arterio-sclerosis the signs of cardiac hypertrophy are present,—namely, downward and outward displacement and abnormally powerful beat of the apex, accentuation of the second aortic sound, and a pulse of high tension, full, regular, and sometimes slow. The especial characteristic is the cord-like feel of the radial artery when under considerable pressure of the finger, a sign not to be confounded with the extreme rigidity indicative of calcification, which is characteristic rather of senile and degenerative changes than of the productive as well as degenerative lesions of arterio-sclerosis. A tortuous course of the radial artery, unless associated with a pulse of high tension, is not a necessary indication of diffuse arterio-sclerosis. As the hypertrophied heart weakens, the tension of the pulse may be lowered, and cough and shortness of breath result from the weakened heart not being able to overcome the obstruction to the flow of blood. Diffuse arterio-sclerosis of the coronary arteries of the heart produces myomalacia and fibrous myocarditis, manifested by præcordial discomfort, perhaps ending in attacks of angina pectoris, in dyspnœa, which may be asthmatic or may present the Cheyne-Stokes characteristics, and, eventually, in acute œdema of the lungs. Arterio-sclerosis of the cerebral vessels is indicated by preliminary attacks of headache or dizziness, perhaps followed by evidence of cerebral hemorrhage or of cerebral softening. Even temporary attacks of localized disturbance of cerebral function may occur in arterio-sclerosis, especially when the latter is dependent upon syphilis or associated with nephritis. If the arterio-sclerosis occurs predominantly in the kidney, the symptoms are those of a slowly progressing fibrous nephritis; slight attacks of dyspnœa, indigestion, headache, muscular weakness, disturbance of vision, frequency of micturition, and thirst give evidence that the renal arterio-sclerosis has occasioned such destruction of tissue that a state of renal inadequacy exists. Arterio-sclerosis of the vessels of the extremities is less a cause of gangrene than is calcification of the muscular media. *Senile gangrene* when not of embolic nature is to be regarded as due rather to arterial calcification than to arterio-sclerosis, while *diabetic gangrene* may be the result of the obliterating tendency of arterio-sclerosis.

DIAGNOSIS.—The presence of nodular arterio-sclerosis is to be assumed in elderly people, though sometimes it is absent in them. Incipient diffuse arterio-sclerosis is to be suspected from a pulse of persistent high tension, and its presence is to be considered probable when the signs of idiopathic hypertrophy of the heart are also present. The diagnosis of a sclerosis of the coronary arteries of the heart is essentially that of a fibrous myocarditis, and the renal arteries are to be considered sclerotic when there is evidence of a chronic fibrous nephritis. The diagnosis of cerebral arterio-sclerosis is based upon the association of symptoms of disturbed cerebral circulation with evidence of arterio-sclerosis elsewhere in the body.

PROGNOSIS.—The prognosis of general arterio-sclerosis is always uncertain, though not necessarily grave. It is uncertain, since rupture of the diseased arteries or aneurisms may ensue. It becomes grave when there is a conspicuous affection of the blood-vessels of the heart or of the kidneys.

TREATMENT.—Although potassium iodide is recommended by some good observers in arterio-sclerosis, we do not believe that the condition is amenable to treatment; in the early stages much can be done to arrest the development of the disease by removal of its cause. Thus, if alcohol, lead, or other poison be producing the disease, the treatment should be that of the chronic poisoning; if the subject be syphilitic, continuous mild antisyphilitic treatment should be employed; if there be a gouty diathesis, this should be actively combated: if there be renal disease, efforts must be directed to overcoming its effects; if, as in many cases, the source of the trouble be over-eating, especially of rich nitrogenous food, with under-exercising, the life-habits should be changed. Very often it is necessary to explain to the patient the condition, so that by a quiet, abstemious life, with abundance of gentle and continuous out-door exercise, the degeneration of the arteries may be delayed. In many cases, especially when the habits of the patient cannot be thoroughly controlled, life will be prolonged and made more comfortable by yearly visits to alkaline purgative mineral springs,—a custom which often may be continued with advantage even in the advanced stages of the disorder. Not rarely the habitual use of the Turkish bath does good by maintaining the activity of the skin. In the advanced stages of the disorder, however, great care must be taken in exposing patients to high heat, especially if there be any secondary cardiac involvement.

As it is impossible to change arteries which have undergone degeneration, the treatment in the advanced stages of arterio-sclerosis is chiefly directed to combating the symptoms of various local disorders as they arise. In apoplectic attacks, with arterial tension, lividity of the face, or marked dyspnœa, venesection may be sometimes necessary for temporary relief.

ANEURISM.

Definition.—The localized dilatation of an artery.

Varieties.—In *true aneurism* all the coats of the artery are present at the outset, although at a later stage it may be impossible to differentiate them. In *false aneurism* the tissues surrounding the artery are pushed aside by blood which has escaped from a torn or cut artery. The *dissecting aneurism* of the aorta is usually regarded as a variety of false aneurism, since at no time are all the coats of the artery contained in the wall. It is the result of a tear into the weakened media through the altered intima in arterio-sclerosis, and the layers of the middle coat are split apart by the force of the blood-current.

When an artery communicates directly with a vein, the resulting dilatation of the vessels is called *aneurism by anastomosis.* If a true aneurism communicates with a vein, the term *arterio-venous aneurism* is applied; while if the communication is established through a false aneurism, the condition is known as *varicose aneurism.*

Etiology.—Aneurisms occur at all periods of life, but especially during the middle third, and more often in men than in women. The essential cause is a diminished resistance of the wall, which may be accompanied by an increased blood-pressure. The diminished resistance of the wall is largely due to premature arterio-sclerosis: hence the importance of syphilis, and perhaps of alcoholic abuse, in the etiology of aneurisms. It should be mentioned that aneurisms may occur without the physical signs of arterio-sclerosis: hence it is assumed that a weakening of the coat represents an early stage of arterio-sclerosis. The wall may be weakened in consequence of the production of an acute endarteritis by an infectious thrombus or embolus, or may be torn by a calcified embolus.

The importance of increased blood-pressure is suggested by the relative frequency of aneurisms among persons exposed to laborious muscular work or severe muscular strain, as sailors and blacksmiths. Evidence in the same direction is presented by the more frequent occurrence of aneurisms of the aorta at its arch, and by the frequent projection of sacculated aneurisms from those points in serpentine aneurism of the splenic artery at which the direction of the current of blood is abruptly changed. Violence is also of importance in etiology, especially in aneurisms of the peripheral arteries. A variety of aneurism of rare occurrence and of unknown etiology has been described by Kussmaul and Maier under the term *nodular periarteritis.* In this affection the intima is forced through a ruptured media, local swellings of the adventitia follow, and thus multiple aneurisms are rapidly produced along the course of the smaller arteries of the body.

MORBID ANATOMY.—The dilatation of the artery is either circumscribed or diffuse. The circumscribed dilatation is cylindrical, spindle-shaped, or globular: hence cylindrical, fusiform, and sacculated aneurisms are discriminated. One or many aneurisms, in rare instances a hundred or more, may be present, varying in size from those not larger than a grain of sand, the miliary aneurisms of the brain, to those which are as large as an infant's head. Diffuse dilatation affects an artery and perhaps its branches over a considerable distance; in consequence of the tortuous course pursued, this variety is called *cirsoid* or *serpentine* aneurism.

At the outset the wall is composed of the three coats of the artery, but as the aneurism increases in size the middle coat disappears, the outer and inner coats become fused, and only a single layer of fibrous tissue remains. Where the three coats are to be differentiated, the appearances of arterio-sclerosis are likely to be found in the intima; fissures, fat-drops, leukocytic infiltration, or fibrous scars are present in the middle coat, and the adventitia is abundantly infiltrated with cells. The circumscribed aneurism communicates with the artery from which it arises by a large or a small opening, which is round or slit-like, and usually contains a lamellated thrombus which sometimes completely fills the sac. The oldest portions of the thrombus, lying upon the wall, are brittle, of an opaque yellowish-gray color, and are covered by elastic layers of a translucent pale-red tint, while the free surface is dark red, and often ribbed. As the aneurism increases in size, displacement of neighboring structures takes place when possible, compression is exercised upon yielding structures, and resistant tissues, especially bone, are eroded and absorbed. The orifices of arterial branches arising in the vicinity of the aneurism may be distorted, and the branches, after being stretched or compressed, may become obliterated. Thrombi frequently are formed in veins which are compressed by the aneurism.

SYMPTOMS.—The symptoms of aneurism depend largely upon the size and situation of the dilatation, and in certain cases there may be no symptoms. In general, the disturbances produced are the result of pressure or of rupture, although the aneurism may first become apparent as a pulsating tumor. Miliary aneurisms of the brain are usually unsuspected until rupture occurs, followed by cerebral hemorrhage.

ANEURISM OF THE AORTA.

The aorta from its origin to its bifurcation is a frequent seat of aneurisms, which are usually divided into thoracic and abdominal aneurisms according to the region concerned. Aneurism is oftenest found at the arch of the aorta, and in that locality, when large, forms a pulsating tumor pressing upon the structures in its vicinity, the symptoms varying according to the part of the arch especially affected.

Aneurism of the ascending portion of the arch may project into the pericardial cavity and produce no symptoms until rupture takes place, when immediate death results from hemorrhage into the pericardium. Aneurisms in this region also cause dilatation of the aortic orifice or shrinkage of the aortic valves, and thus, unless a considerable tumor is formed, give rise only to the signs and symptoms of aortic insufficiency. As the tumor increases in size it is likely to occasion local pain, which is due to pressure upon the cardiac plexus of nerves or upon those of the pleura, pericardium, or skin. The pain, therefore, varies in character, being either anginoid, stitch-like, aching, or neuralgic, and is occasional, paroxysmal, or persistent. Pressure upon the superior vena cava results in venous congestion of the head, neck, and arms, or an innominate vein, usually the right, may be conspicuously compressed, with a corresponding limitation of the cyanosis and œdema to one side or to the other. Perforation of the superior vena cava sometimes occurs, and Pepper and Griffith have called attention to the importance of the sudden development of cyanosis and œdema as indicating this complication. Pressure upon the azygos vein may also take place, giving rise to hydrothorax. Pressure upon the neighboring sympathetic causes dilatation or contraction of the pupil of the right eye according as irritation or paralysis of the sympathetic fibres takes place. The right vocal cord is sometimes paralyzed from pressure upon the recurrent laryngeal nerve.

Aneurism of the transverse portion of the arch has a wider range of pressure-symptoms, since the respiratory and digestive tracts are concerned, as well as the blood-vessels and the nerves. Pressure upon the trachea causes dyspnœa, the breathing at times having a stridulous sound or presenting an asthmatic or a Cheyne-Stokes character. There is not infrequently cough from the catarrhal inflammation of the trachea and bronchi, and a considerable quantity of secretion, at times blood-stained from congestion of the mucous membrane, may be expectorated. Pressure upon a primary bronchus is also productive of cough and dyspnœa associated with feeble respiratory sounds in the part of the lung affected, and may lead to retention of secretion in the bronchi, with resulting putrefaction, ending in gangrene and abscess of the lung. The left vocal cord is frequently paralyzed from pressure upon the left recurrent laryngeal nerve in its course around the arch, and hoarseness, or aphonia, and paroxysms of suffocation may ensue. Pressure upon the pneumogastric nerve is rare, but sometimes happens, causing vomiting and spasm of the œsophagus, with difficulty in swallowing. When the œsophagus is compressed, persistent difficulty of swallowing, especially of solid food, occurs, and the possibility of the presence of an aneurism as the cause of a dysphagia should be eliminated before the use of a sound, since the latter has caused immediate death by perforating an aneurism compressing the œsophagus. The emaciation resulting from aneurismal obstruction of the œsophagus may in rare cases be enhanced by the pressure of the aneurism upon the thoracic duct. The left innominate vein is especially liable to compression, in which case œdema of the left half of the head and neck and of the left arm follows. The orifice of the innominate, the left carotid, or the left subclavian artery, especially of the innominate artery, may be dilated and form part of the aneurism; on the other hand, these vessels are likely, also, to become narrowed and distorted, perhaps obliterated, and may be compressed or obstructed by thrombi. Corresponding variations in the character of the pulsations in them result, especially noticeable in the marked differences sometimes recognized in the radial pulses. The pulmonary artery has been compressed, causing compensatory hypertrophy of the right ventricle, and in rare instances this artery has been even perforated.

Pain is more characteristic of aneurism of the descending portion of the arch and of the thoracic aorta than are symptoms of pressure, although the œsophagus, trachea, left bronchus, and lung may be compressed, and dysphagia, dyspnœa, and cough result. The pain is usually attributed to erosion of the vertebræ, although it is more probably occasioned by a neuritis developed as the spinal nerves become incorporated in the walls of the sac: it is generally referred to the back, in the region of the left scapula. The spinal cord in rare instances has been exposed and compressed, with the production of paralysis. Flint mentions that aneurism of the descending aorta causes a delay in the pulsations in the arteries of the lower extremities as compared with the radial pulse, and Osler states that the femoral pulse may be absent although the blood is distributed to the vessels of the leg.

The physical examination of the thoracic aorta in case of suspected aneurism seeks to establish the presence of a pulsating tumor. When the aneurism is small, or the chest-wall does not yield to its growth, there is no visible tumor. An abnormal area of dulness may be appreciated either at the right or across the upper part of the sternum. If the ascending or the transverse portion of the arch is enlarged, the dulness may be recognized also at the left of the spine in case the descending portion of the arch and the continuous portions of the thoracic aorta are concerned. Eventually the aneurism may so increase in size as to produce a visible pulsatory heaving of the chest-wall near the second and third right costal cartilages, at the sternal notch, or near the spine, according to the seat of the aneurism, and the larger the tumor the more likely is there to be a displacement of the heart downward and to the left. If the sternum becomes eroded, a tumor with expansile and often powerful pulsation projects in the region of the manubrium and extends considerably beyond its borders. The overlying skin eventually becomes livid and necrotic at the place of greatest tension. The tumor is resistant when it contains abundant clotted blood, though readily yielding to pressure when the contents are largely liquid. A systolic thrill as well as a powerful systolic impulse is at times to be felt, and a feeble diastolic beat is not infrequently perceptible. Oliver, Ross, and others have emphasized the value of *tracheal tugging* as a sign of deep-seated aortic aneurism, and it has been asserted that it is never present except when there is an aneurism. According to Grimsdale, however, this sign is of little positive value, since he found it in sixteen per cent. of a number of persons free from aneurism and examined with reference to its presence. The tugging is attributed to the direct pressure during cardiac systole of the dilated artery upon the left bronchus, or upon the portion of the trachea immediately adjoining. The sensation is to be obtained by gently pressing the fingers or thumbs against the cricoid cartilage from below upward while standing either in front of or behind the patient. On auscultation over the aneurism two sounds are usually distinctly heard, one resulting from the distention of the aneurism, the other due to the transmission of the second cardiac sound when the aortic valves are sufficient. There may be no murmurs, or a systolic murmur is to be heard, which is usually of little diagnostic importance unless combined with a diastolic murmur. The latter is indicative of an associated aortic incompetence, but may be produced by an eddy in the aneurism.

In the further progress of aortic aneurism the tendency is towards increased disturbance of respiration, perhaps associated with fever, progressive loss of flesh and strength, and hemorrhage. The bleeding is often the immediate cause of death, and may take place externally or internally. In the former case blood is poured from the mouth by way of the œsophagus, trachea, or lungs, or escapes through the skin. Perforation internally takes place into the pericardial or the pleural cavity, perhaps into the stomach through the œsophagus. The immediately fatal hemorrhage may be preceded by occasional gushes or by slight oozing, separated by intervals of days or weeks. Embolism is an occasional complication, resulting from detachment of portions of the contained thrombus and their transfer into one of the branches of the aorta.

Diagnosis.—The suspicion of an aneurism may be based upon the physical signs or upon the symptoms, but the diagnosis usually requires the presence of both physical and rational signs, and may then be doubtful. An expansile pulsating tumor is not always present, and the expansile pulsation of an unusually high aortic arch or that transmitted from an hypertrophied and dilated heart may be mistaken for the pulsation of an aneurism. The former produces no pressure-symptoms, and the latter is accompanied usually by the signs of aortic insufficiency. A solid tumor of the mediastinum or one at the base of the neck may transmit pulsations, but undergoes no corresponding change of volume. The former muffles the heart-sounds, although interfering but little with breathing and swallowing. Both compress adjacent veins, although affecting the arteries but little.

PROGNOSIS.—Aneurisms of the arch and of the thoracic aorta are always a source of danger, and usually prove fatal, although they may exist for years. Their progress may be checked for a long time by the formation of thrombi, but the process of repair is always incomplete, and it is doubtful if any true aneurism of the arch large enough to produce symptoms has ever been healed.

ANEURISM OF THE ABDOMINAL AORTA.

Aneurism of the abdominal aorta attaining any considerable size usually begins in the vicinity of the cœliac axis, which is often incorporated in the sac. The tumor may be large and prevent the descent of the diaphragm. The lumbar vertebræ are frequently eroded, and then form the posterior wall of the aneurism, being covered merely by a lamellated thrombus. The chief symptom is pain, perhaps followed by numbness, due to pressure upon neighboring nerves or their incorporation in the wall of the tumor. As in thoracic aneurism, the pain may be referred to the back, although the hand of the patient when indicating its seat at times points to the front. There is usually fixed pain in the epigastric region, but shooting pains often follow the lumbar or sciatic nerve. Symptoms of pressure are less significant than in aneurism of the thoracic aorta. Vomiting results from pressure on the pylorus or the duodenum, and pressure upon the colon may interfere with the action of the bowel. Aneurism of the abdominal aorta when sufficiently large to produce symptoms usually continues to increase in size, perhaps with periods of temporary quiescence, until rupture of the wall occurs. Fatal perforation then takes place into the alimentary canal or into the peritoneal or the pleural cavity. The immediately fatal hemorrhage is sometimes preceded by the escape of blood into the retroperitoneal tissues, especially at the left of the spine, causing a rapid increase in the size of the tumor, associated with pain, and suggesting a localized peritonitis. In rare instances the erosion of the vertebræ is so extreme as to lead to compression of the spinal cord and paralysis. Sometimes the superior mesenteric artery is obstructed by embolism or continued thrombosis from the aneurism, acute hemorrhagic infarction of the intestine being the result. The renal arteries, and those of the legs also, may be the seat of the embolism.

DIAGNOSIS.—The diagnosis of this variety of aortic aneurism depends upon the recognition of an immovable rounded or elongated tumor which has an expansile pulsation and may transmit a thrill. On auscultation a systolic, perhaps a double, murmur is to be heard. The femoral as compared with the radial pulse is delayed. The pulsation of the aneurismal tumor may be simulated by the energetic beating of the aorta in neurasthenic, anæmic, or emaciated persons, especially in hysterical women, or by the powerful impulse of the aorta in hypertrophy and dilatation of the left ventricle. The characteristics of the patient, the absence of pain, and the paroxysmal nature of the throbbing indicate the functional disturbance, while the physical examination of the heart makes clear its hypertrophy. Tumors of the stomach, of the liver, or of the head of the pancreas not infrequently transmit the aortic impulse. They are to be excluded by frequent mobility and by a disappearance of the pulsation when the patient is placed in the knee-elbow position.

Aneurisms of the cœliac axis and of its branches, the mesenteric, renal, and iliac arteries, also occur, but are not to be recognized unless forming a pulsating tumor. It is to be remembered, however, that the rupture of abdominal aneurism is one of the causes of intra-abdominal, perhaps of intra-peritoneal, hemorrhage, to be relieved only by surgical treatment.

PROGNOSIS.—The prognosis of this variety of aortic aneurism is exceedingly grave, death usually resulting from perforation and hemorrhage.

TREATMENT OF ANEURISM.—Measures for the cure of aneurism have for their object the obliteration of the sac or more usually of the entire lumen of the affected artery, and are mechanical (that is, surgical) in their nature. The most effective of these surgical procedures, such as ligation or arrest of circulation for a series of hours by pressure on the artery above the aneurism so as to bring on coagulation within the artery, are plainly not applicable to aneurism of the aorta. In a number of cases, however, the carotid, the subclavian, or both vessels, have been tied for aneurism of the aortic arch. Sometimes prolonged relief of the symptoms has been obtained; usually the operation has been without avail: it would seem to be justifiable only when the arteries tied have their aortic origins involved in the aneurism. A second mechanical method which has been used in a number of cases consists in putting into the aneurism horse-hair, fine wire, or a similar foreign body, which shall mechanically bring about a coagulation of the blood within the sac. Again, combined with the use of such materials has been the injection of a few drops of a strong coagulating solution, such as liquor ferri perchloridi. Attempts have also been made to secure coagulation by electrolysis, and it has been asserted that the use of fine silver wire inserted through the hypodermic needle into the sac, associated with subsequent electrolysis (Loreta's method), is sometimes successful. Almost always, however, the induction of these foreign materials into an aortic aneurism has proved disastrous to the patient; nor has any better result attended persistent compression by means of the pad. When the aneurism protrudes through the chest-wall an elastic mechanical support may be used; but permanent compression by an inelastic pad can rarely be borne, and still more rarely does it do good.

In the great majority of cases we are forced to rely upon medical treatment in aortic aneurism, though such treatment seldom if ever suffices for a cure. The object of the treatment is to bring about coagulation in the sac by reducing the force of the blood-current and increasing the coagulability of the blood. The classical method seems to have been first systematized by Valsalva, though perfected and exploited by Joliffe Tufnell; it consists in prolonged absolute rest in the recumbent position, with a restriction of food, and especially of drink, nearly to the minimum necessary for the sustaining of life, and the use of cardiac sedatives; to it may be added various narcotics for the relief of pain and restlessness, and in robust cases even venesection. In order to be effective the rest must be absolute. Two diets are employed: one, known as the "low," consists daily of ten ounces of bread, six ounces of farinaceous pudding, one ounce of butter, and one pint of milk, divided into three meals; fish or boiled meat being added from time to time, if the diet be insupportable by the patient. The "dry" diet consists of four ounces of bread, one-half ounce of butter, and two ounces of milk, for both breakfast and supper, and three ounces each of meat and bread and one ounce of milk for dinner. Many authorities advise repeated venesection.

Theoretically the Valsalva treatment should be continued for from three to four months; practically very few patients can be found to tolerate it for more than six or eight weeks; often the best that can be done is to give from time to time short courses of the treatment. In all cases the return to the normal diet should be gradual. The Valsalva method of treatment is very irksome, and the result is so uncertain that patients may rationally prefer a quicker death to prolonged discomfort. When there is any evidence that the aneurismal sac has a wide mouth, or when the aneurism is fusiform in character, the chances of a good result are greatly lessened. To our thinking, the wisest thing to do in most cases of aortic aneurism is to make the patient comfortable as long as possible, and not to trouble him and his friends with unavailing restrictions.

Potassium iodide is a valuable remedy in the treatment of aortic aneurism, even when there is no distinct syphilitic history. How it acts is not known; it appears often to bring about relief of pain and greatly to favor coagulation of the blood in the sac: ten to fifteen grains three times a day may be given continuously. The only drug which should be used to reduce heart-action is aconite. Veratrum viride is less effective unless given in large dose, and then it is apt to produce vomiting. The bromides, opium, and other narcotics may be administered for the benumbing of the nervous system and the obtaining of sleep. Digitalis, which has been much used by certain surgeons in the treatment of aneurisms, is an especially dangerous remedy. The survival of many patients who have taken it has been due to the drug having been given in such small doses as to be ineffectual: given in large doses it increases not only the force of the circulation, but also the size of the pulse-wave, and thus greatly endangers the tearing away of fibrinous coagula or the rupturing of the sac itself. We have seen the enormous blood-wave produced by digitalis tear open an aortic aneurism, with immediately fatal results.

Under any circumstances the patient with aneurism should lead a quiet life, avoiding emotional and muscular excitement, but not, unless forced by suffering, putting aside all mental work.

The chief aneurismal symptoms requiring treatment are those which are produced by pressure: there is nothing that will relieve the pain caused by an eroding aneurism but morphine. Intense engorgement of the venous system of the head and arms, and violent attacks of dyspnœa with cyanosis, sometimes may require free venesection for relief. Dyspnœa due to pressure upon the trachea is usually the outcome of compression near the bifurcation, and is not to be relieved by tracheotomy: in those rare cases in which bilateral abductor paralysis is caused by pressure on the recurrent laryngeal nerves, as revealed by laryngoscopic examination, tracheotomy may bring relief.

SECTION IV.

DISEASES OF THE RESPIRATORY APPARATUS.

CHAPTER I.

DISEASES OF THE NOSE, LARYNX, TRACHEA, AND BRONCHI.

DISEASES OF THE NOSE.

EPISTAXIS. NOSEBLEED.

Bleeding from the nose is the result of injury either from without, as a blow, or from within in consequence of picking the nose, or of impaction of foreign bodies, or of a fracture at the base of the skull. Local affections of the nasal mucous membrane, as inflammation, ulcer, and polypi, act as causes. Diseases of the blood-vessels, especially dilatation of the veins, and hypertrophy of the heart connected with arterio-sclerosis, are, at times, productive of nosebleed. Epistaxis is infrequent, however, in the chronic venous congestion associated with obstruction to the circulation from chronic disease of the heart and lungs. The ascent into rarefied air, as in mountain-climbing or in a balloon, produces nosebleed. It is of occasional occurrence in young women during menstruation, and its presence, especially periodically, when the catamenia are absent is regarded as a vicarious menstruation. Nosebleed is of frequent occurrence at the outset of typhoid fever, and may occur in the early stage of other infectious diseases. It is of frequent occurrence in chronic anæmia, in leukæmia, in hemorrhagic diathesis, and in hæmophilia. Plethoric persons not infrequently suffer from nosebleed, but the popular assumption that frequent nosebleed is a forerunner of cerebral hemorrhage lacks satisfactory proof.

Bleeding independent of local causes takes place as an oozing, usually from the anterior and lower portion of the cartilaginous septum from one nostril, sometimes from both nostrils. Commonly the blood flows drop by drop from the nasal opening, although it occasionally forms a continuous stream of considerable size, or may be manifested by a projecting clot. The blood may pass into the pharynx, and, when coughed out or swallowed and vomited, be thought to originate in the lungs or stomach. Attacks of nosebleed are rarely sufficient to produce more than temporary weakness. Frequently recurring attacks of epistaxis, however, may result in the symptoms of anæmia, and always demand the search for a local cause in the nostril. Nasal hemorrhage from fracture at the base of the skull may prove a cause of death.

TREATMENT.—In many cases epistaxis can be controlled by simple procedures habitually employed in the household, such as holding the hands up, applying ice or cold pieces of metal to the back of the neck or to the nose, or snuffing up very cold or, preferably, very hot water. The application of a saturated solution of antipyrin or a ten per cent. solution of cocaine is sometimes useful, or solutions of astringents, such as tannic acid or alum, may be injected. Pressure may be applied upon the facial artery as it passes over the lower jaw. The late D. Hayes Agnew stated that he had frequently arrested violent epistaxis, even in cases in which plugging the posterior nares had been insufficient, by making a bougie of a long strip of the rind of bacon, passing it through the nostril, and allowing it to stay there some time. When the epistaxis comes from an ulcerated point that can be reached, the local application of solution of chromic acid or of solid silver nitrate should be made. In any case, if the hemorrhage continues in spite of milder measures, the nares should be plugged both posteriorly and anteriorly.

ACUTE RHINITIS. ACUTE NASAL CATARRH. CORYZA. COLD IN THE HEAD.

ETIOLOGY.—Acute rhinitis is often the result of exposure to draughts of air, to cold or wet weather, and to the local action of inhaled irritants, as dust of various sorts, gas, vapor, or steam. The irritation from bacteria is probably important, as is suggested by the occurrence of epidemics of acute nasal catarrh; indeed, it is not unlikely that "taking cold" from exposure to cold and damp is due to bacterial action, the growth of the bacteria being favored by the circulatory disturbance in the nostrils produced by the exposure. The importance of infection in etiology is also suggested by the occurrence of acute rhinitis as an early manifestation of measles, influenza, and diphtheria; and gonococcal and syphilitic infections may be followed also by acute rhinitis. Inflammation of the nasal mucous membrane not infrequently results from the extension of inflammation of the continuous mucous membrane of the mouth and pharynx.

SYMPTOMS.—Frequent sneezing and increasing obstruction of the nostrils are the significant symptoms of acute rhinitis. These are not infrequently preceded by chilly sensations followed by slight fever, the temperature rising one or two degrees, and by a feeling of general discomfort, perhaps weakness. There is at first a profuse watery secretion from the nasal mucous membrane; later it is slimy and finally opaque yellow. The sense of smell becomes impaired, if not lost, and that of taste is also enfeebled. Herpes of the nostrils or upper lip is not infrequent, and the constant flow of the nasal discharge often causes a maceration of the epidermis, resulting in abrasions upon which crusts finally are formed. The mucous membrane is swollen, reddened, and covered with more or less opaque mucus. Towards the end of the attack it may be eroded and coated or encrusted with pus. Extension of the inflammation to the conjunctiva is frequent, causing redness, swelling, abundant lachrymation, and excessive secretion, and this often dries at the inner canthi. Severe headache and frontal neuralgia follow extension of the rhinitis to the frontal sinuses, and deafness results from a secondary inflammation of the pharynx at the opening of the Eustachian tube. After a few days the symptoms usually disappear, with the exception of the discharge from the nostrils, which may continue gradually diminishing in quantity for a week or ten days.

Although the diagnosis of acute rhinitis is easily made, it is to be remembered that the cause is usually uncertain, since various contagious and infectious diseases begin with nasal catarrh. Caution in diagnosis is especially necessary during epidemics of measles, diphtheria, and influenza. In general acute rhinitis is unimportant, though disagreeable, but the possibility of its extension to the bronchi, especially in the very old and the very young, should be remembered, in which event the outcome may be uncertain. Obstruction to the nostrils so interferes with the nursing of infants that feeding with a spoon is often necessary.

TREATMENT.—Acute coryza does not ordinarily require confinement to the house, but in delicate, very old, or very young subjects it may even be necessary to put the patient to bed. In its formation-period the disease can in many people be arrested or modified by a full dose of quinine taken at bedtime, or by camphor (twenty drops of the tincture every two hours). Often, especially when the attack is accompanied by aching pains or other evidences of a general cold, a free sweat is useful (see formula 7), or the Turkish bath may be tried. Local treatment usually gives great relief. Formula 12 may be used, or bougies made with cacao butter containing one-fourth to one-half of a grain of cocaine may be inserted into the nostrils every four to six hours.

CHRONIC RHINITIS. CHRONIC NASAL CATARRH.

ETIOLOGY.—Constant exposure to the causes of acute rhinitis, especially to irritating dust, and to cold and wet in various trades or occupations, and frequently recurring or unusually severe attacks of acute rhinitis, are the usual causes of chronic inflammation of the nasal mucous membrane. Especially likely to become chronic is acute rhinitis extended into the frontal or ethmoidal sinuses or into the antrum of Highmore. Chronic rhinitis is frequent in scrofulous children, and importance in etiology is to be attached to the construction of the nose, especially to the presence of a deviated septum, which by permanently narrowing the passages permits the retention of inflammatory causes and products.

Symptoms.—Two varieties of chronic rhinitis are recognized, the hypertrophic and the atrophic, according to the nature of the predominant changes of the mucous membrane, but hypertrophy and atrophy are frequently combined. In *hypertrophic rhinitis* the nasal mucous membrane is reddened and swollen, often to such an extent that the space between the nasal septum and the turbinated bones is obliterated. The swelling of the mucous membrane may be general or limited to the anterior or to the posterior nares. The swollen mucous membrane, especially at the posterior end of the lower turbinated bones, may be lobulated, papillate, or project as a polypus. Breathing through the nostrils is obstructed, and the patient becomes a mouth-breather, as in chronic follicular pharyngitis, a condition which often accompanies chronic rhinitis. The expression is dull and the voice is nasal. Taste and smell are lost, and hearing is often impaired from extension of the inflammation to the pharynx. There is abundant muco-purulent secretion, which, especially in children, is seen to ooze from the nostrils, causing eczema of the nose and lips. Crusts formed of the dried secretions and perhaps containing blood are frequent both within the nose and upon the lip. Slight epistaxis often occurs, and is frequently excited by picking at the nose to relieve irritation from the crusts. The septum has occasionally been perforated in the course of time by the picking finger.

In *atrophic rhinitis* the mucous membrane is thin and dense from atrophy of the glands and sclerosis of the interstitial tissue. It is covered with crusts of a green or gray color, producing a constant sense of irritation, in consequence of which the patient frequently picks at the nose and removes the crusts, often with the production of bleeding. The nasal cavities are of large size. The especial characteristic is the extremely fetid discharge, *ozæna*, the odor from which often is not recognized by the patient. The ozæna may be associated also with a deep-seated ulceration, in which case syphilis, tuberculosis, or an impacted foreign body is likely to be the cause. Chronic rhinitis is greatly benefited by treatment, which should be early instituted to prevent a possible deafness, persistent neuralgia, or retarded mental development.

Treatment.—The treatment of both hypertrophic and atrophic rhinitis is chiefly surgical, consisting in the application of various remedies and the removal of hypertrophied or abnormal parts. The results of the continuance of the local disease are so serious, and the treatment is so complicated and special, that in a treatise like the present it is impossible to do more than refer to special works upon the matter, and to say that unless the practitioner shall have had sufficient training in the use of instruments and in the making of local applications to the nose, it is better to refer the case directly to the specialist. Palliation can be obtained by the use of douches containing various antiseptic or deodorizing solutions, which should usually be distinctly alkaline. (See formulas 13 and 14.)

AUTUMNAL CATARRH. HAY FEVER. ROSE COLD.

DEFINITION.—A periodical affection of the naso-pharyngeal mucous membrane, often ending in asthma, and produced in certain persons by special irritants.

ETIOLOGY.—The occurrence of a peculiar form of acute catarrh at the end of May or early in June and lasting three or four weeks has long been recognized both in this country and in England, though rarely seen upon the Continent. This affection has been designated June cold, rose cold, hay fever, or hay asthma, and its origin is attributed to the pollen of certain grasses and cereals. Morrill Wyman first made conspicuous the more serious autumnal catarrh closely allied to June cold in method of origin and symptoms. Essential in the production of both is a nervous temperament, exposure to the exciting causes, and excessive sensitiveness of the nasal mucous membrane. The nervous temperament is often inherited, since successive generations in certain families are sufferers. The exciting causes are present in the atmosphere towards the end of May and about the 20th of August. According to Wyman, they are less frequent in the cities in June than in September. The sufferer in the United States may escape an attack when in Europe. The sensitiveness of the nasal mucous membrane is often sharply localized, either accompanied with or independent of an obvious lesion, as shown by the relief which has rapidly followed the local treatment of the sensitive spot. The exciting causes are to be found not only in the pollen of certain grasses and cereals, especially in new-mown hay, but also in the emanations from various flowers, and even from fruits. The inhalation at other times of various irritants often produces an attack of coryza or asthma in sufferers from the periodical catarrh.

SYMPTOMS.—At or about the stated dates the patient notices an itching in the mouth, nose, or throat, and a sense of fulness or weight in the frontal region. In the course of a day or two there is itching of the eyelids, which are puffy, and the nasal mucous membrane becomes swollen, reddened, and so irritated that a violent attack of sneezing results, which is accompanied by a profuse watery discharge from the nostrils, often continuing throughout the day. Paroxysms of sneezing occur, at first in the morning, and later at irregular times, and are accompanied by more or less febrile disturbance and a sense of prostration. The appetite is poor, and the senses of taste, smell, and hearing are blunted. The throat becomes sore, and in the course of a fortnight the bronchial mucous membrane is irritable and a dry cough is frequent. At this time asthmatic paroxysms are likely to occur. The immediate symptoms of the disease cease at the end of a month, or earlier in case of frost, and the patient, weakened more or less in mind and body, rapidly recovers.

According to Wyman, autumnal catarrh differs from the June catarrh essentially in the greater severity of the symptoms. It is distinguished from simple catarrh by the itching of the eyes, nose, and throat, the profuse discharge, protracted course, terminal asthma, persistent periodicity, and the production of the paroxysm by definite irritants.

PROGNOSIS.—The liability to the June or summer catarrh usually disappears after the age of forty. The autumnal catarrh, on the contrary, ordinarily persists throughout life, even to extreme old age. The severity of the paroxysm diminishes in advancing years, and occasionally varies from time to time in the course of years.

TREATMENT.—The treatment of hay fever based upon the theory that the disease is a neurosis, to be controlled by the use of arsenic and measures for the upbuilding of the nervous system, is of no avail, general treatment having no value other than that of sustaining the system against the exhaustion produced by the local disease. It is maintained by various specialists that local treatment will suffice to cure a large percentage of cases,—a statement which, however, still needs confirmation. The local curative treatment consists in the surgical removal of deformities, destruction by cauterization of sensitive portions of the mucous membrane, and the making of various local applications. It requires great skill in the use of instruments, and, therefore, especial training on the part of the practitioner.

The local palliative treatment consists in the employment of certain drugs having the power of benumbing sensitive nerve-endings. Among these may be mentioned potassium bromide, a solution of which (ten grains to the fluidounce) may at first be carefully applied to sensitive spots, afterwards more freely used and also increased in strength. Antipyrin seems also to have some effect, and cocaine will almost invariably give temporary relief. The free use of cocaine in hay fever, and especially the employment of it by means of sprays to be used at the discretion of the patient, has in a large number of cases produced the narcotic habit, and is to be strongly gainsaid. If the cocaine must be used by the patient for relief, it should be in the form of bougies made with cacao butter, which will melt in the nostril: each bougie should contain one-eighth to one-quarter of a grain of cocaine. In this way much smaller amounts of the alkaloid suffice than with the spray, and the systemic excitement is largely avoided. For the relief of asthma the various narcotics employed in asthma may be given. In the excessive violence of the asthmatic paroxysms of hay fever hypodermic injections of morphine with atropine may be required, but their use is attended with danger of the narcotic habit.

The climatic treatment of hay fever is, we believe, almost invariably successful in preventing the attacks during the treatment; in some cases when the attacks have been thus controlled during a series of years the tendency greatly lessens or entirely disappears.

The way in which climatic treatment acts is at present inexplicable. Thus, the disease exists both in America and in Europe, and it is certain that in a large number of American cases the attack is prevented simply by travelling in Europe, while it is stated by European physicians that the European can avoid the attack by coming to America. A certain degree of elevation above the sea is often effective: thus, in Europe relief is often obtained by going to the high Alps, and in America by going to the higher regions of the Alleghanies, as the summits of the Catskills, or to Whitefield, Mount Washington, Bethlehem, Franconia, or other localities in the White Mountain district. The Rocky Mountains furnish probably innumerable localities of exemption.

Mere "northing" also brings relief to many: thus, cases escape by going to Cape Breton. Life in the primeval forest—at least in the so-called "North Woods," the primeval forest of Northern America—is an almost sure preventive: hence the Adirondacks, Maine, the Canadas, afford relief to many. We have seen the invalid going out of the North Woods during the hay fever season gradually in twenty-four hours develop his hay fever as he passed from the dense forest through the clearings to the open country. Mackinac Island and Georgian Bay are resorted to with asserted success, but in our experience only greatly lessen the severity of the attack.

Usually residence on the sea, in a vessel, or on a small barren island is prophylactic: hence Beach Haven and Fire Island are noted American resorts. In islands situated close to land hay fever subjects may be comfortable when the wind is off the sea, but immediately develop distressing symptoms when the land breeze blows.

DISEASES OF THE LARYNX.

ACUTE LARYNGITIS.

ETIOLOGY.—Primary and secondary forms of acute laryngitis are to be recognized. The former is the result of exposure to cold and wet, and of the action of irritants inhaled or swallowed. The occurrence of epidemics of laryngitis suggests that at times the irritant may be infectious. Excessive strain of the voice, whether sudden or prolonged, is also a frequent cause. Secondary laryngitis results from the extension of inflammation from the naso-pharyngeal or the tracheal mucous membrane, especially when occurring in the sequence of acute infectious diseases, as scarlet fever, measles, diphtheria, influenza, whooping-cough, variola, erysipelas, and typhoid fever, or of chronic infection, as syphilis and tuberculosis. Secondary laryngitis also occurs as the result of injury to the larynx or neighboring parts, and of inflammation of the pharynx and the neck.

Laryngitis accompanied by the formation of a false membrane constitutes the *fibrinous laryngitis*, *membranous* or *pseudo-membranous croup*, of authors. It is usually diphtherial, but the bacillus of diphtheria may be absent and other bacteria, especially streptococci, be present. A fibrinous laryngitis may occur in various infectious diseases, notably scarlet fever, which are wholly independent of true diphtheria; or it may result from the inhalation of irritating gases, vapors, and steam, or the local action of caustics.

VARIETIES.—Acute laryngitis is either superficial or deep-seated, and the inflammatory changes may be limited to definite portions of the larynx, as the epiglottis, the vocal cords, or the hypoglottic region, or all parts of the larynx may be simultaneously diseased. The superficial varieties of inflammation are the catarrhal and pseudo-membranous, the former being characterized by redness and swelling of the mucous membrane, and by the presence of a mucous or muco-purulent secretion, and occasionally of ecchymoses and erosions. The characteristics of pseudo-membranous laryngitis are mentioned in detail in the article on Diphtheria. Phlegmonous laryngitis is the deep-seated variety of acute laryngitis, and is characterized by a serous, fibrino-serous, or cellular infiltration of the submucous tissue, tending towards resolution, suppuration, or necrosis with gangrene. The region below the vocal cords is the part of the larynx in which the phlegmonous inflammation is especially likely to occur.

SYMPTOMS.—Acute laryngitis is either mild or severe from the outset, although, especially in children, mild symptoms may assume rapidly a severe type. Acute catarrhal laryngitis, or acute laryngeal catarrh, is the variety of most frequent occurrence, and is characterized early by a tickling or burning sensation in the larynx, accompanied by a dry cough. The voice soon becomes husky or hoarse, and in the course of a few days may be reduced to a whisper, in consequence of swelling and impaired mobility of the vocal cords. The cough, although at first dry, is later followed by the raising of a viscid or opaque yellow sputum, small in quantity, and perhaps streaked with blood. In young children in whom the glottis is small the cough is high-pitched, the inspiration is noisy, and spasm of the glottis is frequent during the paroxysm of cough, in consequence of which the face becomes purple in the attempt to force air through the contracted glottis. Attacks of dyspnœa associated with hoarseness and a barking cough—*false croup*—are frequent, especially at night, awaking the child from sleep, and are induced both by spasm and by the accumulation of secretion in the glottis. These attacks not infrequently occur for a few nights in succession, the child during the daytime being in apparent comfort.

In acute catarrhal laryngitis of the adult there are but little elevation of temperature and but slight constitutional disturbance. In children, on the contrary, there are often fever, headache, and some prostration.

In severe acute laryngitis, which is usually either membranous or phlegmonous, the obstruction to the larynx is so considerable as usually to cause marked dyspnœa. In the phlegmonous variety the disturbance of breathing may progress with great rapidity, a condition of apparent comfort changing in the course of a few minutes to one of serious, if not of fatal, dyspnœa. The attack is announced by a chill, followed by a temperature of 102° or 103° F., with severe pain referred to the larynx and aggravated by coughing and swallowing. The larynx is tender to the touch.

DIAGNOSIS.—The local irritation, dry cough, and hoarse voice are sufficiently characteristic of acute laryngitis, and the use of the laryngoscope will show redness of the affected part, and perhaps swelling of the mucous membrane, which in severe laryngitis may be limited to the hypoglottic region. In non-diphtherial cases of pseudo-membranous laryngitis the symptoms at the outset are those of an acute catarrhal laryngitis, but there are a constant barking or crowing character to the cough and persistent hoarseness of voice. After two or three days the respiration becomes more difficult, and paroxysms of coughing occur, associated with symptoms of suffocation, whence the term croup. The presence of pseudo-membrane in the pharynx, or the expulsion of membrane from the larynx, warrants the diagnosis of this form of laryngitis; but persistent suppression of voice between the paroxysms and continuing evidences of laryngeal obstruction are of most serious import. False croup suddenly occurs, especially at night, in children comparatively free from serious disturbance of the larynx during the day and at bedtime. The rapid relief following treatment is opposed to the course of laryngeal obstruction in diphtheria or croup.

PROGNOSIS.—Acute catarrhal laryngitis is a disease lasting from a few days to a fortnight. The prognosis in adults is favorable. In young children the laryngeal catarrh is likely to extend into the trachea and bronchi, and then may end in a severe if not dangerous bronchitis. The possibility that relatively mild forms of acute laryngitis both in the adult and in the child may suddenly become dangerous by the production of œdema of the glottis is always to be remembered. The prognosis in false croup is usually very favorable; in pseudo-membranous it is very serious.

TREATMENT.—Unless the attack of acute laryngitis be very severe, the patient should be confined simply to his room, not necessarily to bed. Talking and all use of the voice should be strictly forbidden. The diet should be light but nutritious. In the beginning of the attack the potassium citrate mixture (see formula 16) may be freely used. With it, if there be fever, aconite should be given. Judicious purgation by salines may be advantageous.

The local applications are internal and external. At first the patient should simply inhale steam (vapor) or atomized water (preferably slightly alkalinized), or lime water, at short intervals. Later, when secretion has been in a measure established, ammonium chloride solution (five to twenty grains to the ounce) may be freely used with the atomizer, or compound tincture of benzoin (twenty to thirty drops), vaporized in hot water or in an inhaler, may be employed. Very late in the disease, when secretion is free, the vapors of terebene or of oleum pini sylvestris are sometimes serviceable. Externally, ice-bags may be applied around the throat, but usually the hot-water pack is preferable. Several thicknesses of flannel wrung out of hot water should be wound around the neck, and the whole covered with oil-silk and a towel. A few drops of turpentine upon the flannel will render this application actively counter-irritant.

In the false croup of childhood, an emetic of ipecacuanha, aided by the hot bath, will ordinarily suffice to put an end to the immediate paroxysm. If it does not, potassium bromide with chloral may be exhibited, or a whiff of ether or of chloroform may be given. Hot moist applications, or mustard plasters, around the throat are sometimes of service. Between the paroxysms the child should usually be kept in the house, and should be treated for catarrhal laryngitis, with the addition of certain drugs to overcome the tendency to spasm. Potassium bromide, being entirely safe, should be freely administered. Chloral, cautiously employed at bedtime, is often of service. A dose of castor oil should usually be given directly after the first attack. In pseudo-membranous croup the treatment should be that of diphtheria without antitoxin. Emetics should be freely used, to remove, if possible, the membrane.

CHRONIC LARYNGITIS.

ETIOLOGY.—Frequent or prolonged exposure to the causes of acute laryngitis is important in the etiology of the chronic variety. Of especial significance are the trades demanding exposure to a dust-laden atmosphere or the professions requiring continuous or excessive use of the voice. Chronic passive congestion of the mucous membrane from obstruction to the circulation through the heart or lungs may cause chronic laryngitis, and the excessive use of alcohol or tobacco is important in etiology. Tuberculosis and syphilis as causes of laryngitis are considered in the articles on these diseases. Chronic laryngitis is a disease of adults, and occurs oftener in men than in women.

SYMPTOMS.—There is a sensation of tickling or pricking in the larynx, likely to be followed by a paroxysm of coughing, and aggravated by the use of the voice, a dusty atmosphere, or a sudden change of temperature. There is a frequent tendency to clear the throat, and the coughing results in the raising of a small quantity of dense opaque gray mucus. In consequence of swelling of the vocal cords or weakness of their muscles, the voice is husky, variable, and may eventually be in whispers. Difficulty of swallowing is sometimes present. In chronic hypoglottic laryngitis the mucous membrane and submucous tissue may be so thickened as to cause persistent dyspnœa. On laryngoscopic examination the mucous membrane is usually swollen, of a dark-red color, and the surface may be granular or eroded. At times circumscribed or diffuse thickening of the posterior portion of the cords, *pachydermia laryngis,* is to be seen. As a rare condition the mucous membrane is found atrophied. Chronic laryngitis is an affection usually greatly relieved by treatment, but relapses are frequent, from the difficulty of avoiding exposure to the causes.

TREATMENT.—In chronic laryngitis it is essential to treat carefully any adenoid growths or deformities or enlargements about the nasal passages, and to see that the subject avoids the use of tobacco, alcohol, and very rich foods, as well as abstains from loud or excessive talking and from living in overheated rooms. Usually in chronic laryngitis, as in recurring acute laryngitis, exposure of the neck and frequent ablutions in cold water are beneficial. The general health should be attended to, and local applications made to the mucous membrane of the larynx. These applications cover almost the whole range of local alteratives and astringents,—silver nitrate, iodine and glycerin, potassium chlorate and potassium bromide, zinc salts, bismuth preparations, tannic acid, etc. Any ulcerations should be carefully touched with silver nitrate or other appropriate substance. In tubercular cases iodoform is especially valuable. It may be insufflated two or three times a day in conjunction with a little morphine or cocaine, after the larynx has been cleansed with a slightly antiseptic spray.

When ulceration about the epiglottis interferes with swallowing, the application of cocaine before the meal may afford relief for a time, but usually it soon loses its power. According to Wolfenden, when the epiglottis is so destroyed that swallowing becomes almost impossible, milk may be sucked up through rubber tubing when the patient hangs his head downward over the side of the bed.

In syphilitic laryngitis antisyphilitic remedies must be carefully used in addition to local applications. In many cases tracheotomy should be resorted to. It not only affords relief, but sometimes seems to benefit the larynx permanently by putting it out of use.

ŒDEMA OF THE LARYNX.

ETIOLOGY.—Swelling of the mucous membrane of the larynx from the presence of a serous fluid representing the exudation of inflammation or the effusion of dropsy takes place particularly in the epiglottis, in the aryepiglottic folds, and sometimes in the false cords. The inflammation may originate in the larynx, especially from its injury from within or from without, or may extend from neighboring parts, as the pharynx, the neck, or the submaxillary region. It is often an accompaniment of tuberculosis, syphilis, and cancer of the larynx or the pharynx. The dropsical effusion may be due to local causes, as pressure on the jugular veins by tumors of the thyroid, enlargement of the lymph-glands of the neck or mediastinum, and aneurism of the arch of the aorta. Œdema of the larynx may be due also to the general causes of dropsy, as disease of the kidneys, heart, and lungs. It is said to have followed the use of potassium iodide.

Symptoms.—Dyspnœa from obstruction of inspiration and expiration and a sense of constriction referred to the larynx are the characteristic symptoms. The dyspnœa often rapidly progresses, and may prove fatal within a few hours, or even in a few minutes, unless relief is obtained. The œdematous epiglottis may be seen if the back of the protruded tongue is depressed, or may be felt with the tip of the finger.

Treatment.—When in laryngitis there is a tendency to œdema of the larynx, the case must be carefully watched. Ice may be used externally, and also by the mouth. The internal medicaments and the local applications to the larynx should be much the same as in acute catarrhal laryngitis. Stimulating substances may, however, be used somewhat sooner than in the ordinary cases. As soon as there is pronounced œdema, cocaine should be used as a local anæsthetic, and the epiglottis be well scarified. The practitioner should always be ready to perform tracheotomy at a moment's notice. Nearly all the deaths that have occurred from the disorder could have been prevented by an early operation.

TUMORS OF THE LARYNX.

Of the tumors which occur in the larynx, the myxoma, fibroma, lipoma, chondroma, angioma, adenoma, and cyst are benignant, while the sarcoma and cancer are malignant. Fibroma is oftenest found, especially as the papillary fibroma or papilloma of the true cords, and may be present at birth. It not infrequently occurs in the course of chronic laryngitis and in the vicinity of ulcers of the larynx. Tumors arise from any portion of the larynx, but especially from the vocal cords or their immediate vicinity. They are usually rounded, often pedunculate, the surface frequently warty, and sometimes attain a considerable size.

They may be so situated and so small as to produce no symptoms. As a rule, the first manifestation is hoarseness, which persists for a long time and may end in aphonia. Cough is frequent when the tumor is situated in the vicinity of the glottis, and dyspnœa arises if the size of the tumor is such as to obstruct the larynx or its shape and mobility permit sudden closure of the glottis, under which circumstances spasms of dyspnœa threatening suffocation result. Pain and difficulty in swallowing are rare. The prognosis is favorable except in the case of malignant tumors, as the growth early produces laryngeal symptoms and removal with the aid of the laryngoscope is easy.

Treatment.—The treatment of tumors of the larynx is surgical removal.

DISEASES OF THE TRACHEA AND BRONCHI.

Inflammation of the trachea, *trachitis*, rarely occurs independently of disease of the larynx or bronchi; hence the primary symptoms are those of a laryngitis or of a bronchitis. Affection of the trachea is indicated by the occurrence of pain or tenderness in the course of this tube.

When the inflammation of the mucous membrane of the respiratory tract is limited particularly to the bronchi, the condition is known as bronchitis. According to the localization of the inflammation the distinction is drawn between bronchitis of the larger and medium-sized tubes, simple bronchitis, and bronchitis of the smallest tubes, capillary bronchitis. Simple bronchitis is either acute or chronic; capillary bronchitis when not tubercular is always acute. A further distinction is based upon the nature of the product of the inflammation. Usually it represents an increased quantity and modified quality of the secretion which normally appears on the surface of the membrane, and hence is then designated catarrhal bronchitis. Rarely a cast of a bronchus and its branches formed of fibrin or other material represents the inflammatory product, and the condition is then known as membranous or fibrinous bronchitis.

ACUTE BRONCHITIS. ACUTE BRONCHIAL CATARRH.

Etiology.—The growing tendency at present is to regard micro-organisms as the immediate cause of acute bronchitis. This is generally admitted in epidemic bronchitis and in the bronchitis of influenza, whooping-cough, diphtheria, tuberculosis, measles, and erysipelas. It is considered probable in the bronchial inflammation of variola, typhoid fever, malarial fevers, and syphilis. Since various pathogenic bacteria have been found repeatedly in bronchi free from disease, predisposing or favoring causes are obviously necessary. Most common of these is exposure to frequent and sudden changes of temperature and moisture, which oftenest occurs in the spring and fall, at which seasons cases of acute bronchitis are most numerous. The inhalation of dust, or of irritating gases, vapor, or steam, is also to be included among the causes. Conditions interfering with the freedom of respiration, whether deformed thorax, disease of the lungs, heart, or kidneys, gout, rickets, or scrofula, and the weakness due to alcoholism or occurring in infancy and in old age, are also favoring causes.

Morbid Anatomy.—The inflamed mucous membrane is reddened and swollen, from congestion of the blood-vessels and from the presence of a serous and cellular exudation in the mucous membrane. The surface is covered with secretion, which varies in characteristics according to the stage of inflammation. When the smaller bronchi are inflamed the secretion exudes on pressure, and patches of atelectasis and nodules of broncho-pneumonia are frequently associated.

SYMPTOMS.—In simple bronchitis the attack often begins with sneezing and hoarseness, indicative of irritation of the nasal and laryngeal mucous membrane, or at the outset the symptoms are referred to the bronchi, and consist of a sense of constriction or a tickling or a raw feeling beneath the sternum or in the region of the trachea. If the inflammation is localized in the trachea or in the bronchus or the bronchi of the right or the left lung, the seat of the discomfort corresponds. With these incipient symptoms there may be slight fever, perhaps preceded by chilly sensations, backache, muscular pains, and weakness. Cough is of early occurrence, is at first occasional, dry, and annoying, and in nervous persons is often paroxysmal and unduly violent. It is sometimes so excessive as to cause vomiting. In the course of a day or two a glairy secretion is raised, at first in small quantity. As the cough loosens the secretion increases in quantity, becomes opaque and eventually yellow, and not infrequently contains specks or streaks of blood. The sputum consists of a hyaline or fibrillated material in which are mucous and pus corpuscles and pavement, cylindrical, and ciliated epithelium. Red blood-corpuscles are often seen with the microscope when there is no suggestion of blood from the appearance of the sputum. On physical examination of the chest the sounds on percussion are essentially normal. On auscultation there is no decided alteration of the respiratory murmur. Râles are present, at first dry, sonorous, or sibilant, and changing in quality, even disappearing after coughing. As the formation of secretion increases, moist râles are to be heard, both coarse and fine, during inspiration and expiration, and are accompanied by dry râles. The coarse râles are to be recognized on palpation, and are not infrequently heard at some distance from the patient.

In mild bronchitis relief to the discomfort usually occurs as the secretion becomes profuse and easily expelled. The disease lasts a week or ten days, and the discomforts are generally insufficient to compel the patient to stay in bed.

Capillary bronchitis is present when the inflammation extends to the small bronchi, and as a rule is diffused over a wide area in both lungs. The smaller bronchi may be inflamed at the outset, or the attack may begin as a simple bronchitis, becoming progressively worse as the inflammation extends into the bronchioles. The severe symptoms indicative of capillary bronchitis may exist, therefore, from the beginning or develop in the course of simple bronchitis. Dyspnœa, often extreme, especially in infants, is the conspicuous feature, manifested by rapid and superficial breathing, movements of the nostrils, and exaggerated use of the accessory muscles of respiration. The cervical muscles become prominent. The lower ribs are retracted on inspiration. The skin is of a bluish tint, and the jugular veins are distended. The cough is frequent and short, and the secretion is moderate in quantity, and is usually swallowed by young children. The temperature is 103° F., or more, and the pulse quick and feeble. The patient takes but little nourishment, loses strength rapidly, is restless, may be drowsy or delirious, and, if a child, may have convulsions. Percussion is likely to be negative, the dulness from areas of broncho-pneumonia and atelectasis being concealed by the deep seat or by the hyperresonance of neighboring portions of the lung. The respiratory murmur varies in quality in different parts of the chest, is either feeble or harsh, and the expiration is prolonged. It is often obscured by numerous fine moist râles, diffused or circumscribed, heard both on inspiration and on expiration, especially in the lower half of the lungs. Coarse moist râles and dry râles are also to be heard, but are less characteristic than the fine moist râles of the localization of the disease in the smaller bronchi. Capillary bronchitis usually lasts two or three weeks, and is often a cause of death in old persons and in infants. The severe forms of capillary bronchitis end in broncho-pneumonia, and will be further considered in the article on broncho-pneumonia.

DIAGNOSIS.—Simple bronchitis is readily recognized by the cough, the variety of râles, and the slight constitutional disturbance. The distinction between simple bronchitis and a mild attack of influenza during the occurrence of an epidemic of the latter is often arbitrary, and is based upon the absence of the characteristic symptoms of influenza as the disease progresses. The distinction between simple bronchitis and capillary bronchitis is one of degree. Mild symptoms indicate localization of the inflammation in the larger bronchi; dyspnœa, high fever, and abundant fine moist râles indicate its seat in the smaller bronchi. In infants in whom cough may be slight and expectoration absent, dyspnœa and fever may be the only prominent symptoms. The distinction between capillary bronchitis and lobular pneumonia or broncho-pneumonia is one of inference, based upon the severity and duration of the disease.

TREATMENT.—The treatment of acute bronchitis should vary with the intensity of the attack. In mild cases it may be necessary only to keep the patient in-doors, but when the symptoms are severe confinement to bed should be enforced, whilst the air should be rendered moist by means of the steam atomizer or other device. A light, nutritious, supporting diet should be given. Free external counter-irritation is very important; it should be applied alternately over the back and the chest. The turpentine stupe or the mustard plaster may be used, but ordinarily it is better to make a poultice containing one part of mustard to from five to seven parts of flaxseed-meal, which may be left on for many hours. In children the so-called jacket-poultice is very valuable; it consists of thin flannel made into a bag of such shape that it can be applied closely around the chest and kept in place by tapes tied over the shoulder and in front; to prevent sagging of the contents to the bottom of the bag, a line of stitching should be run lengthwise through the centre. The jacket-poultice should be put on as hot as can be borne;

when there is fever it will not get cold for many hours, and need not be changed for from eight to twelve hours. In many cases five to ten per cent. of mustard may be put in the flaxseed-meal with advantage. When the skin becomes sore, or when it is not convenient to use the jacket-poultice, many practitioners wrap the child's chest in cotton or woollen batting, and cover this with an oil-silk jacket, so that by retention of the perspiration a moist application can be obtained.

The keeping in of the heat of the body by these appliances is a very serious objection to their use when there is high fever; the cotton batting is probably more objectionable than is the poultice; indeed, when the bodily temperature is high, cold applications (ice poultices or compresses wet with cold water) to the chest are preferable to either the poultice or the batting. When bronchitis persists and becomes subacute, the official pitch plaster, or the official pitch plaster with cantharides (warming plaster), placed between the shoulders, often acts very well. When the disease locates itself and remains obstinate in one lobe of the lung, even though the presence of catarrhal pneumonia cannot be demonstrated, a large blister will often be found of the greatest service. When there is great bronchial irritation, relief may be afforded by the inhalation of watery vapors; but drug inhalations have little value in acute bronchitis.

In the first stage of acute bronchitis the chief indication is to favor secretion and thereby aid in resolution. In the robust patient a forming bronchitis may sometimes be aborted by the administration of sufficient doses of veratrum viride (one drop of the fluid extract) or tartar emetic (one-twelfth to one-eighth of a grain) every half-hour until free vomiting is induced. Such treatment is too violent, however, for ordinary purposes, but we have used it with success in robust public speakers or other persons who felt compelled to fulfil some business engagement in a short time. After the vomiting has been induced and the relief of the bronchitis obtained, alcoholic stimulants may be used. In the case of children or feeble adults neither veratrum viride nor tartar emetic should be administered for bronchitis.

The most effective general remedy is the potassium citrate mixture (see formula 16), with ipecacuanha, apomorphine, or tartar emetic, in accordance with the nature of the individual case. One ounce of potassium citrate may be exhibited within the twenty-four hours; sometimes a single half-ounce of the salt given at one dose in the evening will put an end to a forming cold. Flaxseed tea and other demulcent drinks are often grateful to the patient.

When fever exists, aconite may be added to the potassium citrate mixture. Antipyrin, phenacetin, and other drugs of the class are often useful, and may be employed with greater freedom than in infectious diseases. When, however, there is any feebleness, care must be exercised in their use. If the temperature remains above 103° F., there should be no hesitancy in the external use of cold sponging or of cold baths if necessary; with children the tepid bath (90° F.) is usually preferable to cold sponging.

In the second stage of bronchitis, when there is secretion with relaxation, the stimulant expectorants are useful. Ammonium chloride may be considered to lie half-way between the sedative and the stimulant expectorants, and therefore to be especially useful in the opening period of the second stage. It should be given at intervals of not less than two hours, in doses of from ten to fifteen grains; it is often preferred simply dissolved in water, or may be exhibited in accordance with formula 17. With it may be at first combined ipecacuanha or apomorphine. Senega and squill are of very doubtful value: we have never been able to perceive any distinct influence from them.

At a later period, after the establishment of free secretion, certain volatile oils are very effective. Of these oil of eucalyptus may usually be first employed, as the least stimulating; oil of garlic, in the form of syrup of garlic, is often very useful in the protracted bronchitis of children. Terebene is one of the most valuable of the class. Oil of sandal wood, of copaiba, or of cubeb may be tried if other remedies fail or have been used until the lungs have become accustomed to them. Compound tincture of benzoin (five to ten drops on sugar) every two hours is sometimes useful. Terebene and the various oils are best given in capsules (three to five minims each). Creasote, or guaiacol or its carbonate, is often very effective when the bronchitis becomes subacute and persists with free expectoration. (The treatment of capillary bronchitis is given in the article on Broncho-Pneumonia, page 763.)

The proper treatment of the symptom "cough" involves the understanding of the value and object of cough. This act is necessary for the relief of the lungs from various exudations. Cough may be produced by a nervous or an inflammatory irritation of the mucous membrane at a time when there is nothing to be coughed up, or if there be exudation to be expelled the amount of cough present may be in excess of what is needful. In either of these cases the cough becomes an evil, to be done away with or checked as far as practicable. On the other hand, when there is excessive exudation, and especially if there be at the same time great weakness of the patient, as in infancy or in old age, the cough may not be sufficient to bring about relief, in which case it must, if possible, be increased or replaced in some way. It is plain that the amount of cough which the patient has, taken by itself, is not a sufficient guide as to whether the cough is excessive or not. It is the relation between the cough and the work to be accomplished which must be considered by the practitioner. Cough can sometimes be allayed by the use of inhalations of vapor or fumes of medicated sprays which lessen the irritation of the mucous membrane. Belladonna may be used in this way. (See Asthma.) When the cough is of laryngeal origin, cocaine is a valuable local remedy. In most cases excessive cough is to be checked by drugs which benumb the central nervous system: of these the most powerful is opium, which is, however, equally powerful in checking the secretion of the respiratory mucous membrane as well as in deranging digestion. When, therefore, as in the early stages of a bronchitis, it is desired to increase bronchial secretion, opium should not be employed. Again, excessively violent cough is especially seen in hysterical neurotic subjects, most of whom do not bear opium well, and with whom when the narcotic is borne there is danger of the formation of the opium habit. Hyoscyamus is free from all objection and is much used; it is, however, comparatively feeble and uncertain in its action, and must be given in full doses to accomplish anything. The bromides and antipyrin are, we think, more decided in their influence than is hyoscyamus. Codeine is much used by certain practitioners. In some cases chloroform given by the stomach in the cough mixture acts most happily. Prussic acid has been much used. The action of such agents as chloroform and prussic acid lasts only for a very short time, and to exert anything like a continuous influence the drug must be given at intervals of not more than an hour.

In neurotic or hysterical subjects a bronchial irritation may be maintained for weeks or months simply from a cough which is produced by a hyperæsthesia of the mucous membrane. In such cases we have seen recoveries rapidly follow the withdrawal of all expectorant remedies, and the free use of the antispasmodics just mentioned, alone or in combination. Travel, with its change of air and scene, is especially useful in these cases.

Whenever in acute bronchitis it is perceptible that the strength is waning, strychnine and cocaine should be administered. In the old, the alcoholic, and the feeble this condition is especially liable to arise. The strychnine may be added to the cough mixture, but in bad cases should be given hypodermically. An excellent plan is to give it and cocaine alternately every two hours, increasing the dose from one-thirtieth (strychnine) and one-fourth (cocaine) rapidly until as much as one-sixteenth and one-half grain respectively are given, if it be found necessary.

CHRONIC BRONCHITIS. CHRONIC BRONCHIAL CATARRH.

ETIOLOGY.—Chronic bronchitis is essentially a disease of elderly people, and represents the results of repeated exposure to the causes of acute bronchitis. It is likely to occur in persons suffering from chronic disease of the heart, lungs, or kidneys, in scrofula, in gout, in eczema, and in alcoholism. In winter exacerbations are frequent; in summer remissions occur.

MORBID ANATOMY.—The mucous membrane of the affected bronchi either is thickened, reddened, and the surface velvety, perhaps eroded, or is thin, smooth, and shining. Not infrequently there is a simultaneous occurrence of hypertrophy of the mucous membrane of the larger bronchi and atrophy of that of the smaller tubes. When there is abundance of the secretion it readily escapes from the cut tubes. The muscular and elastic fibres in the wall are frequently hypertrophied and distinctly project into the canal. Dilatation of the bronchial tubes, peribronchitis, and emphysema are frequently associated.

SYMPTOMS.—Persistent cough is the conspicuous symptom of chronic bronchitis, and usually becomes aggravated with the approach of winter. It is mild or violent, constant or in paroxysms. The latter often occur at the beginning and end of the day, and may take place at night, disturbing the sleep of the patient. Violent paroxysms of coughing may be associated with dyspnœa, and the face become of a bluish tint, the superficial veins be distended, and the accessory muscles of respiration be brought violently in play. The physical examination of the chest, except for the recognition of râles, is practically negative unless emphysema or bronchitis is present. Sonorous and sibilant râles are to be heard the more abundantly the less the secretion, while moist râles, coarse and fine, are often present, especially in the lower and posterior portions of the lung. In general the course of chronic bronchitis extends over a period of many years, and the health is not materially impaired until emphysema, bronchiectasis, fibrous pneumonia, and dilated heart occur as complications, when dyspnœa, cyanosis, and dropsy are likely to result.

The varieties of chronic bronchitis usually recognized are designated according to the quantity or quality of the discharge from the bronchi. In *dry catarrh* the smaller bronchi are affected, the secretion is scanty, tough, opaque gray. Dyspnœa is constant, and paroxysms of coughing are violent. The physical signs are those of emphysema combined with dry râles. When the chronic bronchial catarrh is associated with abundant purulent secretion the condition is known as *bronchorrhœa*, and a pint or more may be expectorated in the course of a day. The sputum varies in appearance according as mucus or pus predominates, and clumps of green or yellow pus may be present in a more gelatinous and transparent material. The more profuse the secretion the more likely is bronchiectasis to be associated. In bronchorrhœa there is usually progressive loss of flesh and strength, and dropsy not infrequently occurs towards the end of life. The term *serous bronchorrhœa* is applied when the expectoration is profuse, viscid, and clear, resembling mucilage. There is usually but little constitutional disturbance, and the patient may attain extreme old age. *Putrid bronchitis* results from the occurrence of putrefaction in the bronchial secretion, and is to be discriminated from the putrefactive condition occurring in gangrene of the lung by the absence of elastic fibres, and from that occurring in phthisical cavities by the absence of the bacilli of tuberculosis. A febrile temperature occasionally is present, the breath is fetid, and gangrene of the bronchial wall or foci of gangrenous broncho-pneumonia sometimes result. Several instances have been reported of abscess of the brain as a complication.

In general there is progressive loss of flesh and strength, although the course of the disease is rapid when complicated with pulmonary gangrene.

TREATMENT.—In the management of a case of chronic bronchitis, if possible, the patient should be sent to a warm, dry, equable climate, where out-door life can be enjoyed at all times. This is especially true of those cases in which each winter brings a recurrent prolonged bronchitis, the so-called "winter cough." If it is impossible to change the habitation of the person suffering from chronic bronchitis, every precaution should be taken to avoid exposure; heavy under-flannels should be worn. Frequently the tendency to take cold can be modified by the daily use of the cold bath, commenced during the summer months and continued, and by the continuous administration for months of minute doses of arsenic (one-hundredth of a grain *ter die*), which remedy is also of value in the treatment of formed chronic bronchitis.

Tonics, nutritious food, wine, cod-liver oil, regulated exercise, and all drugs and procedures which tend to strengthen the bodily health and vitality, are of the greatest service in chronic bronchitis.

In attempting to modify the disease directly by drugs, the practitioner must select his expectorant according to the condition of the mucous membrane. In an acute exacerbation, with great dryness of the mucous membrane, the potassium citrate mixture may be employed. When, however, it is desired to increase secretion for a longer period, potassium iodide is preferable. The iodide may also be used in very small doses (five to ten grains a day) for weeks at a time, in order to aid in the absorption of exudations which have taken place into the mucous membrane. When there is excessive secretion, gallic acid (five to ten grains four times a day) may be very serviceable. In old cases, with nervousness, and especially when there is a tendency to abdominal flatulence from atony of the bowels, asafetida often gives temporary relief.

The most generally useful expectorants are, however, the volatile oils. Of these, terebene, oil of eucalyptus, oil of sandal wood, oil of copaiba, oil of cubeb, and even oil of turpentine, may be from time to time employed. Benzoic acid, pure or in the form of compound tincture of benzoin (five to ten drops every three hours on sugar), is an occasional remedy. Creosote is among the most valuable of the drugs; it should be given in capsule or emulsion, in slowly increasing doses, until thirty minims a day are taken or the limit of gastric tolerance is reached. Sulphuretted hydrogen has been especially used by respiration, and many springs are now furnished with respiratory chambers. It may, however, be given in solution. It is often efficacious when there is much expectoration in a chronic bronchitis. From two to four ounces of the saturated watery solution may be administered by the mouth four or five times a day or until the breath has a perceptible odor. In the use of expectorants it is essential to avoid disturbing the digestion, and also to vary the drug from time to time according as the mucous membrane of the lung appears to become accustomed to one remedy.

When in chronic bronchitis there is failure of the respiratory function, either from retention of secretion or from loss of the functional power of the lung by anatomical alteration, strychnine and cocaine are the chief reliances; only under rare circumstances is the attempt to clear the lungs by the use of emetics justifiable.

Counter-irritation in chronic bronchitis should be limited to paroxysms of exacerbation or to the occasional use of pitch or other irritant plasters. Inhalations are frequently of value; terebene, compound tincture of benzoin or other benzoic acid preparations, and various stimulating volatile substances of balsamic or terebinthinate nature may be used from time to time with advantage.

In the advanced stages of chronic bronchitis there is almost always dilatation of the right heart, so that the treatment of cases eventually resolves itself largely into the treatment of chronic cardiac failure.

MEMBRANOUS BRONCHITIS.

Definition.—A localized inflammation of the bronchi, usually chronic, and characterized by the expulsion of an arborescent cast of a bronchus and its branches.

Fibrinous casts are formed in the bronchi frequently in diphtheria, in fibrinous laryngitis of non-diphtherial origin, and in pneumonia. Hemorrhagic casts are at times formed from the aspirated blood in nose-bleed and cut-throat, and in the occurrence of pulmonary hemorrhage. In membranous bronchitis, however, inflammation of the bronchial mucous membrane is the primary condition, and has been designated plastic, exudative, fibrinous, and polypoid bronchitis or bronchiolitis.

Nothing definite is known with reference to the causation of this affection. It occurs usually in adults, often in the vigorous and healthy, but frequently in pulmonary tuberculosis. It has been observed also in connection with cardiac disease, and a number of cases have been reported in which herpes zoster, pemphigus, and impetigo were associated. Cases of acute membranous bronchitis have been reported from time to time characterized by the sudden occurrence of fever accompanied with cough and a sense of substernal constriction. The physical signs are those indicative of a localized acute bronchitis, and the more exact nature of the disease is made evident by the expulsion of a bronchial cast, following which relief, perhaps merely temporary, is experienced. In the instances reported the occasional combination of a membranous tonsillitis and the frequent death from suffocation or pneumonia suggest that cases of diphtheria and pneumonia may have been regarded as acute membranous bronchitis.

Chronic membranous bronchitis is the variety commonly encountered, and recurrent attacks are likely to take place at intervals of weeks or months over a period of years. Cough is the conspicuous feature, is sometimes paroxysmal, and generally is not sufficiently severe to interfere with the habits and occupation of the individual. Attacks of dyspnœa may immediately precede the expulsion of the cast, although often there may be no difficulty of breathing at any time. During the occurrence of dyspnœa there is frequently more or less cyanosis. The physical signs are those of bronchitis, and, although respiration in the affected portion of the lung is likely to be feeble or absent, the physical examination of the chest is usually negative, with the exception of the signs of a bronchitis. The expulsion of the bronchial cast is the essential feature, and is at times associated with more or less hemorrhage. The cast ordinarily appears as a rounded, flesh-like mass, in part, perhaps, composed of mucus and blood, and when unfolded in water assumes an arborescent character. The size of the cast varies in accordance with that of the bronchus affected. The cast is either hollow or solid, homogeneous or laminated, and is composed of fibrin, more rarely of inspissated mucus, leukocytes, red blood-corpuscles, and Charcot crystals. The spiral fibres of Curschmann have been found in the cast.

Although the prognosis of chronic fibrinous bronchitis is usually favorable, it is to be remembered that in nearly one-half of the cases reported as fibrinous bronchitis tuberculosis has existed, and the prognosis should be guarded until evidence of the latter disease has been repeatedly sought for in vain.

TREATMENT.—The treatment is that of chronic bronchitis.

BRONCHIECTASIS.

DEFINITION.—Dilatation of the bronchi.

ETIOLOGY.—Bronchiectasis is the result of conditions which produce a weakening of the wall of the bronchus, and an increase of the atmospheric pressure against it, especially from persistent coughing. Most important in the causation of weakness of the wall is chronic inflammation of the bronchi, whether occurring in chronic catarrhal bronchitis, in tubercular bronchitis, or in consequence of the presence of foreign bodies. Bronchiectasis also occurs as the result of the obstruction of a tube by the pressure of tumors, the obliteration of alveoli in fibrous pneumonia, or interference with their expansion by chronic pleurisy. The atmospheric pressure is then exercised upon the unobstructed spaces in the lung, and a collateral, compensatory or vicarious bronchiectasis results, usually associated with emphysema. Bronchiectasis is essentially a disease of adult life, but at times is found in infants even at birth. This congenital bronchiectasis, in which numerous cysts containing a thin liquid are found, usually in one lung, is regarded as the result of syphilis, although it is probable that in certain instances it represents irregularities of development from unknown causes.

MORBID ANATOMY.—Bronchiectasis generally affects bronchi of medium size and their smaller branches, and the alterations may be present throughout both lungs, in which case the lower lobes are more likely to be especially affected. They are oftenest limited, however, to one lung, perhaps to a single lobe. The dilatation is uniform, cylindrical, or circumscribed (fusiform or saccular), the fusiform dilatations often being varicose from the alternation of dilated portions with those of relatively normal calibre. As a result of the dilatation, the diameter of the affected portion of the bronchial tube is variously increased, and in saccular dilatation numerous cavities of various size are formed along the course of the bronchus. The cavities may be blind from the obliteration of outgoing branches, and the connection with the main trunk is also sometimes destroyed. Isolated cavities of bronchial origin thus are formed in the lungs. Neighboring cavities may become confluent from absorption of the lung-tissue, giving rise to trabeculated sacs as large as the fist. Dilatation of the smaller bronchi is often made evident by the ease with which their course is followed with the scissors. The wall of the dilated bronchus is either hypertrophied or atrophied, the two conditions not infrequently occurring in different parts of the same lung. The hypertrophied mucous membrane is thickened, sometimes corrugated, perhaps covered with villi, and of a dark-red color from the presence of numerous dilated blood-vessels. When atrophied, the wall is thin, smooth, and shining, often not sharply defined from the surrounding lung-tissue. As the wall of the bronchus becomes thin the epithelium is flattened, the cartilage is absorbed, the muscular tissue and elastic fibres in part disappear and in part are widely separated. The peribronchial tissue becomes thickened and fibrous. The dilated bronchi contain thin, opaque gray, muco-purulent fluid; in the sacculi is a denser secretion, which may be an opaque gray mucus or inspissated caseous material, sometimes infiltrated with lime salts. If the contents are inspissated the wall is usually thickened and contracted, indicating a tendency to obliteration of the cavity. Saccular dilatations which are entirely disconnected from the bronchus from which they originate may appear as cysts containing a clear fluid. Ulceration of the wall is rare, but may occur, especially when the contents of the dilated bronchus are retained and become putrid. Gangrene of the wall then may result and extend to the neighboring lung-tissue.

SYMPTOMS.—For a long time the symptoms and signs of bronchiectasis are those of chronic bronchitis, and are not likely to suggest dilatation of the bronchi unless localized at some particular part of the lung. The symptom which is especially characteristic of bronchiectasis is cough occurring in paroxysms and followed by the ejection of a large quantity of sputum. The cough usually occurs in the morning, and not infrequently is induced by a change of position, as perhaps in turning from one side to the other, or in assuming the upright position after a night's rest. More than twenty ounces of secretion may be raised from the lungs in twenty-four hours. When allowed to settle it is usually thin, of a dirty-gray color, and frequently is of a disagreeable odor, which in certain cases is distinctly putrid. A sediment is formed also composed of pus-corpuscles, with which are small grayish particles containing fattily degenerated cells, crystals of fat acids, numerous red blood-corpuscles, and bacteria; hæmatoidin crystals also are sometimes present. The upper layer is a thin liquid covered with a brownish froth. In diffused bronchiectasis the physical signs are those of a chronic bronchitis, while in circumscribed bronchiectasis they are those of a cavity, manifested by amphoric resonance, cavernous breathing, and coarse gurgling râles, alternating with dulness and feeble or absent respiration in the same place according as the cavity is full or empty.

After many years there is likely to be progressively increasing dyspnœa, aggravated on slight exertion, and then associated with cyanosis. The finger-tips are clubbed, the *hypertrophic osteoarthropathy* of Marie, as in chronic tuberculosis and in obstruction to the pulmonary circulation from cardiac disease. The diagnosis is based upon the peculiarities of the cough and expectoration and their occurrence for a long time without other symptoms. It is certain only when the signs of a cavity are present. In such cases tuberculosis is eliminated by the absence of bacilli, gangrene by the history and the absence of shreds of lung-tissue, and abscess by lack of evidence of antecedent pneumonia. If the signs of a cavity are absent, bronchiectasis may be overlooked.

The prognosis is usually favorable as to length of life, but death may suddenly occur from hæmoptysis, or rapidly from gangrene of the lung if the secretion becomes putrid. As a rule, diffused bronchiectasis is eventually complicated with emphysema, dilatation of the heart, and cardiac incompetency, with their symptoms and result. In other cases, in the course of years the patient becomes weak and thin from the persistent cough accompanied eventually by continued fever, the symptoms being those of a pulmonary phthisis.

TREATMENT.—There is no known method of reducing bronchial dilatation. The administration both by the mouth and by inhalation of terebene and various volatile oils, especially of sandal wood or of creosote, sometimes is effective in checking excessive secretion. Osler recommends that with a suitable syringe there be introduced into the trachea twice a day a drachm of a solution of ten parts of menthol and two parts of guaiacol in eighty-eight parts of olive oil. When the cavity is situated near the surface of the lung it may sometimes be advantageously opened through the chest-wall and drained.

BRONCHIAL OBSTRUCTION.

Obstruction of the trachea and bronchi may result from external or internal causes. The common external cause is pressure from tumors, especially those of the thyroid, and of the cervical and mediastinal lymph-glands, and aneurism of the arch of the aorta. Cancer of the œsophagus, vertebral and intra-pulmonary tumors, abscess of the mediastinum, enlargement of the heart, and pericardial exudation also may produce pressure from without. The internal causes of obstruction include foreign bodies, membranous exudations from acute inflammation, and strictures from chronic inflammation or syphilis and tumors.

In obstruction of the trachea there is a sense of constriction in the region affected, with difficulty of breathing, especially of inspiration, which is prolonged and perhaps harsh. According to the degree of obstruction is the severity of the dyspnœa, which may become so great as to be associated with conspicuous action of the accessory muscles of respiration, although the larynx changes its position but little.

Bronchial obstruction is generally unilateral, and the result depends upon the size of the bronchus obstructed and the degree of the obstruction. The symptoms of obstruction of a small bronchus may be those of a localized bronchitis. If a large tube is obstructed, the air is both admitted and expelled with difficulty, inspiration being more interfered with than expiration. If the obstruction is complete, atelectasis results, and there is shrinkage of the corresponding half of the chest, with compensatory emphysema of the other lung and corresponding distention of that half of the thorax. Prominence of the cervical muscles and retraction of the supraclavicular and intercostal spaces and of the lower ribs indicate the severity of the dyspnœa. In the region of incomplete obstruction sonorous râles are to be heard, and may be recognized on palpation of the chest. The respiratory murmur is feeble or absent in the region supplied by the obstructed bronchus, and is exaggerated elsewhere.

PROGNOSIS.—The prognosis depends upon the nature and the cause of the obstruction, which may be such as early to cause death, or in case of the obstruction of a small or medium-sized bronchus may be directly recovered from, although bronchiectasis and emphysema may permanently result.

TREATMENT.—When the bronchial obstruction is due to a tumor, this, if possible, should be removed; foreign bodies may be coughed up, but if they can be located, by the Röntgen rays or otherwise, they call for surgical interference. Medical treatment should be advised in case of enlargement of the thyroid or of mediastinal lymphomata, as recommended in the articles on these subjects. The appropriate treatment for syphilis should be used when this disease is suspected as a cause of the obstruction. If the exudation of pericarditis is sufficiently large to produce symptoms of obstruction, it may be removed by paracentesis. The dyspnœa from inoperable or incurable causes of obstruction is to be relieved by inhalations of oxygen or by the administration of codeine or morphine.

ASTHMA. BRONCHIAL ASTHMA.

DEFINITION.—A functional disturbance of the respiratory apparatus manifested by sudden attacks of dyspnœa, with intervals of comparative freedom. Expiration is more interfered with than inspiration, and the sputum is usually characteristic.

The term asthma is used often to include various spasmodic affections of respiration, especially the attacks of dyspnœa occurring in laryngitis or in consequence of the pressure of tumors in the vicinity of the trachea and bronchi, *e.g.*, so-called *thyroid* asthma and *thymic* asthma. Attacks of dyspnœa occurring in disease of the heart or blood-vessels are usually designated *cardiac* asthma; these, and the probably closely allied uræmic dyspnœa, so-called *renal* asthma, and the attacks of dyspnœa in hysteria often due to spasm of the diaphragm and sometimes termed asthma, are to be regarded as symptoms of the diseases in which they occur, the disease asthma being limited to the peculiar paroxysms of disturbed breathing excited more especially by conditions limited to the respiratory tract. Although it is asserted that the dyspnœa is due usually to spasm of the involuntary muscles in the bronchial wall, it is urged also that a congestive swelling of the bronchial mucous membrane may occur and mechanically disturb breathing. Probably of especial importance as a mechanical cause of the dyspnœa is the peculiar material expelled towards the end of the attack and regarded as casts of the bronchioles.

ETIOLOGY.—The sensitiveness of the nervous apparatus which makes the patient liable to attacks of asthma is considered to be often congenital, since asthma frequently occurs in certain families, especially those in which neurasthenia, hysteria, epilepsy, neuralgia, and gout are common. It is observed that asthma not infrequently occurs in persons suffering from saccharine diabetes and lead poisoning. Bronchial asthma may be present at all periods of life, but more frequently in the adult, and is found in men oftener than in women. Of late years especial importance has been directed to the presence of local disease of the respiratory mucous membrane, especially in the nose, pharynx, and larynx, as favoring the production of asthma.

The immediate cause of the attack is often a direct or reflex irritation of the nerves of respiration, especially the branches of the pneumogastric nerve. The exciting causes appear to be intimately connected with climate. Residence in a given locality may be helpful to the one and injurious to the other. The influence of climate does not depend upon the degree of moisture or the range of temperature, but oftener upon peculiarities of the individual, since a moist climate and cold weather are beneficial to some and injurious to others. Attacks of asthma are frequently brought on by the direct inhalation of irritating particles, whether as dust or as odors from flowers or even animals. In certain cases the attack of asthma is regarded as the result of a reflex irritation of the nerves of respiration from disease of the stomach or of the intestinal tract, hence *dyspeptic* or *nervous* asthma, or from the pelvic organs, and even in consequence of pregnancy, hence *uterine* asthma.

SYMPTOMS.—Asthma is a disease of gradual development, and there is nothing suggestive of this affection until the occurrence of a paroxysm of dyspnœa. The existence of a sensitive respiratory mucous membrane is indicated often by the frequent occurrence of attacks of bronchitis, and there may be occasionally a sense of thoracic constriction. The immediate asthmatic attack usually takes place at night, and is sometimes preceded for several hours by a sense of substernal constriction, frontal headache, or digestive disturbance. The patient is roused from a sound sleep with a sense of suffocation. He sits upright, and breathes violently, but not with increased frequency, the inspirations usually being short and deep and the expirations prolonged. In other cases inspiration may be relatively easy and expiration especially labored. The respiration is noisy from the numerous sonorous and sibilant râles which are to be heard even at a remote distance from the patient. The accessory muscles of respiration are firmly contracted, the chest is fully expanded, and the patient leans forward, sits astride a chair, or by other change of position endeavors to expand the chest to its utmost. As the dyspnœa increases, the face is at first red, then of a bluish tint, and finally pale; the skin is cool, there is profuse sweating, and the pulse becomes rapid and weak. There is increased resonance on percussion, from excessive distention of the lungs, which overlap the heart and depress the diaphragm. On auscultation coarse and fine musical râles are to be heard throughout the chest both on inspiration and on expiration, more abundantly during the latter. The attack of dyspnœa continues for minutes or hours, relief often being experienced by the expulsion of a characteristic sputum. The cough is at first slight and dry, but becomes paroxysmal and forcible in the efforts to raise secretion, the presence of which in the lungs is often made evident by the substitution of moist for dry râles. The sputum is viscid, grayish white, scanty, or profuse, and contains the spiral threads discovered by Curschmann. Some of these may be large enough to be seen with the naked eye, while others require the use of the lens. When the fibres are examined with the microscope they are found to be composed of a gelatinous mass twisted about a homogeneous, usually translucent, central thread. The spiral fibres contain mucin, and in the sputum are also alveolar epithelium, eosinophiles, and Charcot crystals, the latter increasing in number the longer the sputum is exposed to the air.

The attack usually lasts several hours, but may terminate in the course of minutes, and the severest attacks may last for days. As the breathing becomes easier the patient feels exhausted, falls asleep, and awakes apparently well, at the most somewhat fatigued. Other attacks are likely to occur in the course of successive days or at intervals of a number of days, during which there is more or less cough and even characteristic sputum between the attacks. Longer or shorter intervals of freedom, lasting months or years, may then follow, or the attacks are of such frequent occurrence that pulmonary emphysema and eventual dilatation of the heart result.

Diagnosis.—Expiratory dyspnœa affecting both lungs is an important characteristic of bronchial asthma, by means of which other causes of recurrent attacks of dyspnœa, except in emphysema, chronic bronchitis, and cardiac asthma, may be excluded. In emphysema the physical examination of the chest and the persistence of dyspnœa are sufficiently distinctive. In cardiac asthma the dyspnœa affects both inspiration and expiration. Râles are absent unless pulmonary œdema occurs as a complication. The discovery of Curschmann's spirals in the sputum at the close of an attack of dyspnœa is to be made practically only in bronchial asthma.

Prognosis.—Asthma, except when it occurs in childhood, is usually a disease lasting for years, and in the bronchial asthma of adults permanent recovery is rare unless the cause is remediable or the patient is able to find and permanently reside in a climate in which he is free from the attacks. The usual termination of this disease is in emphysema and chronic bronchitis, manifested by persistent dyspnœa, chronic cough, abundant muco-purulent sputum, and an hypertrophied right ventricle, which tends to become incompetent with resulting cyanosis and dropsy. In such cases attacks of bronchial asthma may be replaced by those of cardiac asthma, in which inspiration and expiration are alike affected and the physical signs are those of œdema of the lungs.

Treatment.—The treatment of a case of asthma naturally divides itself into the management of the case between and during the paroxysms. As the attacks sometimes depend upon a removable cause, especially upon the presence of polypi or other obstructive lesions of the nose, a careful search for such cause should be made, followed, if opportunity offer, by removal. The urine also should always be examined, to prevent the overlooking of a uræmic origin. As it is probable that in many cases the disease depends, in part, at least, upon an excessive susceptibility of the mucous membranes of the lung to catarrhal inflammation, and as in old cases there is almost always more or less complicating chronic bronchitis, the treatment of chronic bronchitis, especially its climatic treatment, is often very important. Further, in many cases without obvious bronchial catarrh, asthma may be modified by climatic surroundings, so that when it is possible residence in a warm, usually moist, climate should be secured. In scarcely any other disease, however, is it so essential to study the individual characteristics in each case, as at present the relations of asthmatic attacks to climate and locality are entirely inexplicable. As an example, we may mention the case of a great traveller and sufferer from the disease who found freedom from asthma only in the city of Caracas. To some cases the sea-shore, to others the mountains, afford relief; some do best in the city, others in the country; occasionally change of rooms in the house affects the number and violence of the attacks; not rarely sleeping upon feathers or a feather pillow will provoke asthmatic symptoms.

The general hygienic management should be that of chronic bronchial catarrh, though, especially in children, some importance may be attached to restricting the use of meat or other highly nitrogenous foods.

No diet, however, which produces indigestion or flatulence is suitable for the asthmatic. If an almost purely meat diet is the only one digested, it should be the only one allowed. Further, the heavy meal should always be taken in the middle of the day, and the supper should be made very light, so that the digestion may be completed by bedtime.

In some cases of asthma the continuous use of small doses of arsenic for months has pronounced effect. More generally useful is potassium iodide, which should be given for three months in ascending doses up to the point of tolerance. The beneficial effects in the prevention of attacks which sometimes follow the use of grindelia robusta and other balsamic expectorants are probably due to the relief of complicating catarrhs. At times the continuous administration of the bromides, especially of ammonium bromide, with antipyrin or other of the allied chemical drugs, is very useful in lessening the susceptibility of the nervous system. The inhalation of compressed air in the pneumatic cabinet is highly spoken of by some writers.

The remedies which are employed during the paroxysm of asthma are numerous. A hypodermic injection of morphine and atropine or the inhalation of a little ether or chloroform sometimes will abort an attack, but in the use of these agents the danger of the formation of a drug habit is a very important consideration. The most generally useful remedies are the group of vegetable drugs which contain atropine and its allied alkaloids,—namely, stramonium of various species, belladonna, and hyoscyamus. These drugs have the power of paralyzing the motor nerves, and hence act much more favorably in asthmatic or other spasm when applied locally. For this reason they are most effectively used in asthma by means of smoking. For some unknown reason the fumes produced by the burning of potassium nitrate have a most soothing effect upon many asthmatics, and the addition of this salt to the powdered stramonium or other allied herb makes it burn much more steadily and rapidly and at the same time increases the efficacy of the fumes. (See formula 15.) The numerous proprietary powders used in asthma, often with great relief, usually contain a delirifacient herb and the potassium salt. The addition of arsenic still further increases the efficiency of the mixture. (See formula 18.)

The nitrites, especially amyl nitrite taken by inhalation, are exceedingly effective in the height of a paroxysm, but are very apt soon to lose their power. The inhalation of chloroform or of ether will sometimes immediately cut short an attack. Pyridin has been strongly recommended by Germain Sée and others, the fumes of half a drachm to a drachm exposed in a saucer being inhaled. An old remedy, which is especially effective when the asthma is complicated with subacute bronchitis, is tincture of lobelia, which may be taken through the day or at the time of the attack in doses of fifteen drops of the tincture every twenty minutes until it causes vomiting. Chloral often acts well. Hoffmann's anodyne is sometimes of service. Counter-irritation by means of mustard plasters over the whole chest is sometimes serviceable.

CHAPTER II.

DISEASES OF THE LUNGS.

CONGESTION OF THE LUNGS.

An increased quantity of blood may be present either throughout the lungs or in limited portions of them. It occurs under a variety of conditions, and is either transitory or persistent. The distinction is drawn usually between active and passive congestion, the former being attributed to the presence of an increased quantity of blood in the arteries, while passive congestion is the result of an obstruction to the outflow of blood from the lungs.

Active congestion of the lungs may result from prolonged and violent muscular effort, as in running or rowing, in which the action of the heart is excessively increased, or may be caused by a rarefied atmosphere, as in mountain-climbing or balloon-ascensions. It may be due to the inhalation of hot air or other irritants, and to the virus of pneumonia, influenza, and tuberculosis. Acute congestion of the lung sometimes follows the rapid withdrawal of a large quantity of fluid from the chest in pleurisy. An arterial congestion of the lung has been caused by affections of the brain, especially at the base, and in unilateral cerebral lesions the pulmonary congestion has been found on the opposite side of the body. The congestion is called compensatory or collateral when the other lung is prevented from receiving a normal quantity of blood by pressure from without, as in pleurisy or from tumors, or by pressure from within, as in pneumonia, or by embolism of the pulmonary artery. Congestion, perhaps collateral, usually occurs in acute miliary tuberculosis of the lung.

In rapid and general congestion of the lungs there are extreme dyspnœa and a bloody, frothy sputum. Death may occur instantaneously, as in pulmonary embolism or during violent muscular effort, in which case the lungs are excessively distended, and of a dark-red color throughout, and frothy blood escapes from the cut surface. In general, however, the pulmonary congestion is to be regarded as a complication of the disease in which it is present, and is insufficient to produce serious disturbance.

Passive congestion of the lungs is either general or partial. When general it is the result of causes preventing the escape of blood from the lung, especially obstruction to the flow through the heart in consequence of stenosis of the valve or weakness of the parietes. Passive pulmonary congestion follows also obstruction to the venous outflow by intra-thoracic tumors.

General passive congestion of the lungs is usually slowly progressive, and they become distended, heavy, resistant, and of a dark reddish-brown color, which on exposure to the air resembles that of iron rust. The cut surface is relatively dry. To this condition of the lungs the term *brown induration* is given. Microscopic examination shows that the distended capillaries project far into the alveoli, the interstitial tissue is somewhat increased in quantity and abundantly pigmented, and the alveoli contain large epithelial cells in which are red blood-corpuscles and granules of blood-pigment.

Brown induration of the lung gives rise to no especial symptoms while the heart is capable of performing its work. With failing compensation dyspnœa, cough, and a sputum containing blood-corpuscles and blood-pigment are likely to result.

Hypostatic congestion is used to designate partial passive congestion of the lungs which results from an enfeebled action of the heart combined with the persistence of the body in a definite position. It therefore occurs in those affections in which these causes are oftenest associated. Hypostatic congestion is to be expected in prolonged severe infectious diseases, especially typhoid fever and acute articular rheumatism. It is to be found in prolonged coma, as from cerebral hemorrhage, and is likely to occur in the profound weakness due to extensive loss of blood. The condition is also to be observed in extreme distention of the abdomen from liquid, gas, or tumors. The dependent parts of the lungs or of a lung, according to the position which the patient has long held, are of a dark-purple color, are heavy, and contain but little air. The blood-vessels are injected, and there is blood in the alveoli. In extreme cases the cut surface resembles that of the spleen,—*splenization*,—the affected part of the lung is easily torn, and on pressure a thick, bloody fluid escapes. The splenified lung readily becomes inflamed, in which case the condition is known as *hypostatic pneumonia*.

There are no symptoms especially characteristic of hypostatic congestion, the associated feebleness of the respiration and of the pulse being symptoms to be expected in the affections giving rise to the congestion. Careful physical examination is likely to show a slight diminution of resonance in the dependent parts of the chest, feeble or bronchial breathing, and fine moist râles, slowly advancing along the line of gravity. Commonly the condition of the patient is such that physical examination of the chest is inadvisable.

TREATMENT.—The treatment of congestion of the lungs is usually that of the condition which produces it. If it be from heart-failure it is to be met by powerful cardiac stimulants, aided by counter-irritation with turpentine stupes or similar quickly acting powerful rubefacients, or with dry cups all over the back. The cases of cardiac disease in which the intensest pulmonary engorgement exists represent those which are spoken of on page 674, in which free venesection affords the only means of relief, and in which, if the blood cannot be obtained otherwise, even aspiration of the right auricle may be justifiable.

In the hypostatic congestion of typhoid or other low fevers, in addition to the use of alcohol, digitalis, and other cardiac stimulants, and of free counter-irritation, the exhibition of large doses of ergot (to tone up the pulmonic blood-vessels) and of terebinthinate expectorants (oil of turpentine and terebene) is sometimes advantageous.

When the pulmonic congestion is due to a narcotic poisoning, as with morphine, active artificial respiration, or, better, so-called "forced respiration," affords the best chance of recovery.

Active congestion of the lungs, if it really exists, is to be treated, like the first stages of pneumonia, by the use of venesection or local bleeding and of cardiac sedatives,—especially veratrum viride.

PULMONARY HEMORRHAGE.

Hemorrhage occurs from the large as well as from the small blood-vessels of the lung, and may be also the result of the rupture of an aneurism of the aorta into the lung. When large pulmonary vessels are the source of hemorrhage the rupture is usually due to a weakening, perhaps aneurismal dilatation, of the arteries of the walls of phthisical cavities. As a rule, the bleeding comes from small vessels, especially of the congested mucous membrane in bronchitis or from the alveolar capillaries in acute congestion and in pneumonia. Pulmonary hemorrhage also occurs in gangrene, abscess, and cancer of the lung, in pulmonary embolism, in chronic passive congestion, and in purpura. A periodical pulmonary hemorrhage has been observed rarely in women with amenorrhœa, and is regarded as vicarious. Pulmonary hemorrhages connected with disease at the base of the brain are considered to be of vaso-motor origin, and if the lesion is on one side of the brain the pulmonary hemorrhage may be found in the lung of the opposite side of the body. The blood is to be found in the lung as clots in the larger bronchi, or as specks or streaks, due to the inhalation of blood into the bronchioles and alveoli. A pulmonary cavity may be distended with a mass of clotted blood, and in hemorrhage complicating intra-cranial disease circumscribed rounded masses of hemorrhagic infiltration perhaps as large as the fist may be present. *Hæmoptysis* is the symptom significant of pulmonary hemorrhage, although profuse and even fatal bleeding into the lungs may take place without the spitting of blood. Hæmoptysis is generally immediately preceded by a slight irritative cough or by a tickling sensation in the larynx. The mouth is then suddenly filled with a liquid having a saltish or slightly astringent taste and proving to be blood. In severe cases of pulmonary hemorrhage, as from the rupture of an aneurism, the flow of blood may be so rapid that in a few minutes a pint or more of almost pure red blood escapes. Usually a few or several mouthfuls, in all less than an ounce, of blood are ejected, and the blood is bright red and frothy. For several days after the attack of hæmoptysis dark blood intimately mixed with mucus is occasionally coughed up. In the first attack of hæmoptysis the patient, as a rule, appears anxious, and, if the hemorrhage is profuse, collapse rapidly follows, the pulse becoming feeble and the skin cool and moist.

Pulmonary hemorrhage is rarely immediately dangerous unless copious in advanced tuberculosis or from a ruptured aneurism. Death then results in part from the loss of blood, in part from suffocation in consequence of obstruction of the bronchial tubes by the inhalation of blood. Commonly the hemorrhage ceases quickly, the amount of blood lost being insufficient to produce anæmia. Slight attacks of hemorrhage, especially in tuberculosis, are of frequent occurrence, and the patient becomes so accustomed to hæmoptysis as to be but little disturbed by this symptom. Indeed, not infrequently it affords temporary relief to a sense of constriction localized in the chest. Hæmoptysis is to be distinguished from hæmatemesis, since it is associated rather with coughing than with vomiting, and the blood is liquid, red, and frothy, not clotted, dark, or resembling coffee-grounds. The physical examination of the lungs when tuberculosis is suspected as the cause of pulmonary hemorrhage should be postponed until the cessation of the bleeding, lest a recurrent attack should be occasioned thereby. Attacks of hæmoptysis are sometimes feigned to excite sympathy or for other purposes, but the microscopical examination of the red fluid then fails to disclose blood-corpuscles, and the physical examination of the respiratory tract and the history of the case give no evidence of the usual causes of pulmonary hemorrhage.

The treatment of hæmoptysis is sufficiently considered in the articles on Tuberculosis (page 298) and on Chronic Heart Disease (page 676).

THROMBOSIS AND EMBOLISM.

Branches of the pulmonary artery may become obstructed rarely by thrombi, which sometimes originate in the vessel in consequence of disease of its wall or from pressure upon it. The usual cause of obstruction of the pulmonary artery is an embolus composed of clotted blood and brought in the circulating blood either from the right side of the heart or from the systemic veins of the body, especially from the pelvic plexus and from the veins of the legs. The embolus may be so large as to obstruct the pulmonary artery at its origin, but is usually of such size as to enter an intra-pulmonary branch. Its arrest at any particular point is followed by a stoppage of the direct arterial flow into the region supplied by the obstructed vessel and by extreme congestion of the part, ending in hemorrhage, through the collateral circulation. There result in consequence the familiar appearances of hemorrhagic infarction, single or multiple, according to the number of emboli. Wedge-shaped masses are formed, the base usually represented by the pleura, of the size of beech-nuts or Brazil nuts, and in extreme cases composing an entire lobe of the lung.

They are of a dark-red color, dense, relatively dry on section, and free from air. In the later stage of the infarction the nodule becomes paler in color and softer in consistency, and absorption of the extravasated blood is possible. If the embolus comes from a septic thrombus, abscess results, a septic pleurisy being the usual complication. A similar termination at times occurs in bland embolism, from the presence of bacteria upon the respiratory surface within the region of infarction.

The symptoms of pulmonary embolism depend upon the size of the artery obstructed. If a small branch only of the pulmonary artery is concerned, there may be little or no disturbance. When larger branches are affected, respiration is usually temporarily increased. There may be a chill, although there is but little subsequent fever, except in septic embolism. There is slight cough, followed sooner or later by bloody expectoration in small quantities and lasting several days. If the infarction extends to the pleura, localized pain occurs. In small infarctions, or in those situated centrally, the physical signs are usually negative. In large infarctions, circumscribed dulness and friction, feeble or bronchial breathing, and moist râles are to be expected. When the embolus is large enough to obstruct the trunk of the pulmonary artery there are sudden extreme dyspnœa, the admission of air into the lungs being unobstructed, cough, thoracic pain, lividity or pallor, rapidly failing pulse, cold sweats, intense anxiety, and attacks of fainting or unconsciousness, with or without spasms, and death occurs in the course of a few minutes or within an hour or two.

The diagnosis of embolic infarction is based upon the sudden and unexpected occurrence of dyspnœa, the air-tubes being open, followed by cough and occasional hemorrhagic sputa for several days, in a person in whom the condition of the heart or of the systemic veins is such as to permit the presence of a thrombus.

TREATMENT.—There is no special treatment for pulmonic emboli or thrombi. Opium should be given as required to quiet nervous distress, and symptoms, as far as may be, should be met as they arise.

ŒDEMA OF THE LUNGS.

Œdema occurs in the lungs, as elsewhere, in consequence of active or passive disturbances of the circulation or of increased porosity of the wall of the blood-vessels from affections of nutrition or of innervation. It is, therefore, to be found in diseases in which modifications of pulmonary circulation are present and in those in which general dropsy is conspicuous, as acute and chronic nephritis, cardiac disease, and various chronic diseases in which anæmia and impairment of nutrition are prominent. In these as in other affections, even those of acute nature, diffuse or circumscribed œdema of the lungs may occur at the close of life and be the immediate cause of death. Diffuse pulmonary œdema sometimes takes place in consequence of acute pulmonary congestion following embolism of the trunk of the pulmonary artery or the rapid removal of a large quantity of fluid from the pleural cavity. More frequently it is the result of obstructed venous outflow, and is best explained by Welch's investigations, which show that a paralyzed left ventricle prevents the escape of blood from the pulmonary veins in sufficient quantity to make room for that entering from the right ventricle. Capillary engorgement, followed by the transudation of serum and red blood-corpuscles, is the result. In circumscribed œdema the transudation of fluid is dependent upon local as well as upon general causes. If gravity determines the position of the fluid, the œdema is called *hypostatic*. In atelectasis the affected portion of the lung becomes œdematous in consequence of the localized bronchial obstruction and pulmonary congestion, and in *collateral œdema* the fluid is accumulated in the vicinity of a congested or inflamed portion of the lung, and frequently represents an inflammatory exudation.

In œdema of the lungs the effusion from the blood-vessels accumulates in the alveoli and in the interstitial tissue. The affected portion of the lung is distended, dense, heavy, pitting on pressure, and slightly crepitant. It is gray, or reddish gray, and more or less translucent according to the quantity of blood present and the degree of the œdema. On section of the lung abundant fluid is to be squeezed from the œdematous portion, and is the more frothy the more acute the œdema. In chronic pulmonary œdema the fluid contains fewer air-bubbles and more red blood-corpuscles, and the lung-tissue is easily torn. Chronic œdema of the lungs is so intimately associated with chronic congestion, desquamation from the alveolar walls, and the presence of inflammatory irritants, and even of an inflammatory exudation, that the distinction between chronic œdema, chronic passive congestion, and desquamative or catarrhal pneumonia is often arbitrary.

The symptoms of œdema are generally of slow onset, and present but little that is characteristic until there are a sense of more or less suffocation, increasing difficulty of breathing, perhaps associated with cyanosis, a short. dry cough, and a weak pulse. If the œdema occurs rapidly, as in severe pulmonary embolism or thoracentesis, there may be violent dyspnœa and abundant, frothy, perhaps hemorrhagic, sputa. On physical examination of the œdematous portion of the lung there is dulness, the extent and intensity of which depend upon the quantity of fluid present, and the dulness is often comparatively slight, especially in acute œdema. The respiratory sounds are enfeebled, and abundant fine moist râles are to be heard.

Diffuse œdema of the lungs is a condition of serious import, since it usually represents a terminal stage in the disease of which it is a symptom.

Treatment.—The treatment of œdema of the lungs is that of the renal or other condition which produces the attack, with the addition of very active counter-irritation by means of turpentine stupes or of dry cups used freely, and the exhibition, as a rule, of large doses of strychnine and other respiratory stimulants hypodermically. In a large proportion of cases cardiac stimulants are urgently required; digitalis and ether may be used subcutaneously, whilst alcohol, musk, camphor, or Hoffmann's anodyne is given freely by the mouth. In some sthenic cases wet cupping, or even venesection, may be practised with advantage. In uræmic cases with secondary cardiac weakness it must not be forgotten that the hot bath—either the Turkish, the Russian, or the simple water bath—is dangerous, and that even pilocarpine given hypodermically may increase the difficulty by increasing the intra-pulmonic exudation. Great caution is, therefore, necessary in the use of active diaphoretics or diaphoretic measures. The vapor bath, so applied that the patient does not breathe the hot air, is probably the safest and most efficient diaphoretic procedure in these cases. In most cases of œdema of the lungs free serous purging is of great advantage.

ATELECTASIS.

Definition.—An incomplete dilatation of the alveoli and a total or partial lack of air in them.

Etiology.—The immediate cause of atelectasis is obstruction to the admission of air in consequence of the inability of the lungs to expand, or of a mechanical obstruction in the air-passages. The distinction is usually drawn between congenital and acquired atelectasis. In *congenital* or *fœtal atelectasis* the affected portions of the lungs fail to expand, either in consequence of weakness of the infant from disease or premature birth, or from obstruction of the air-passages by foreign material, strangulation, or an abdominal tumor. In *acquired atelectasis* the air is prevented from entering the alveoli in part because of weakness from severe disease, but particularly from obstruction of the bronchi in consequence of inflammation of the mucous membrane, especially when the smaller tubes are concerned, as in capillary bronchitis, or in measles, whooping-cough, and diphtheria. The entrance of air into the alveoli is prevented also by compression of the lung from the presence of liquid or gas in the pleural or pericardial cavity, from intra-thoracic tumors, and from extreme curvature of the spine. Excessive quantities of liquid or gas or large tumors in the abdominal cavity, by opposing the descent of the diaphragm, likewise interfere with the admission of air into the lungs. According to the causes, acquired atelectasis may thus be divided into atelectasis from obstruction and atelectasis from compression.

Morbid Anatomy.—In fœtal atelectasis the posterior and lower portions of the lung are usually affected, and the lobules into which the air has failed to enter appear as dense, dark-blue, solid, more or less wedge-shaped nodules, depressed beneath the pleura. The cut surface is smooth, and but little blood escapes on pressure. In acquired atelectasis the air is removed from the affected portions of the lungs in part by the contraction of the elastic tissue, in part by absorption, and in part by compression of the surface. According to the resulting appearances, the distinction is drawn between the *collapsed* and the *compressed* lung. The collapsed portions of the lung are few or many, more or less wedge-shaped, of a bluish slate color as seen depressed beneath the pleural surface; they are dense, airless, non-crepitant, and are smooth on section. The lobules concerned and those in fœtal atelectasis can be artificially inflated. In the course of time the atelectatic portion of the lung becomes distended by the passage into the alveoli of the liquid and solid constituents of the blood-vessels, and by the desquamation of epithelium from the alveolar wall. The cut surface somewhat resembles that of the spleen: hence the appearances at this stage, as in hypostatic congestion, are designated *splenization*, and somewhat simulate those of lobular pneumonia, which eventually may supervene, but are characterized by less moisture, by more brittleness, and by a pleuritic exudation. The alveoli may remain permanently contracted, and are often obliterated by sclerosis of the interstitial tissue of the lung, in which case an indurated, pigmented, depressed scar remains.

In atelectasis from compression a single lobe or even the entire lung may be involved, and the greater the compression the denser the lung and the paler the color, since both air and blood are expelled. In extreme cases the compressed lung is in a state of *carnification*. It then forms a flattened, cake-like mass situated at the upper and posterior portion of the chest, and of a tough, leather-like consistency, non-crepitant, and of a bluish-gray color intermingled with black specks, which are more or less numerous according to the pre-existing quantity of pigment.

SYMPTOMS.—Atelectasis produces deficient aeration of the blood, but the degree is rarely so extreme as to give rise to characteristic symptoms. These, however, sometimes occur in congenital atelectasis, and the breathing of the infant is then rapid and superficial, the heart is weak, the veins are distended, and the extremities are cold. There are drowsiness and muscular twitchings, and death is a frequent result. The dyspnœa and cyanosis present in the capillary bronchitis of children and adults are in part attributable to existing areas of atelectasis. Protracted atelectasis in the fœtus may interfere with the closure of the ductus arteriosus and of the foramen ovale, and the permanent atelectasis of the adult may give rise to collateral emphysema of the lung and to hypertrophy of the right side of the heart, with the liability to eventual degeneration. On physical examination of the chest, foci of atelectasis may be overlooked or the signs of capillary bronchitis be observed. Extensive atelectasis when superficial gives rise to dulness on percussion, or to a harsh respiratory murmur and increased vocal fremitus, as in pneumonia. It is to be distinguished from the latter disease by the gradual onset, the limitation to the dependent portions of the lung, and the association with an obvious cause.

The prognosis of atelectasis depends upon its cause, and, therefore, is grave, perhaps fatal, in fœtal atelectasis, and is serious according to its extent in the atelectasis of capillary bronchitis. Permanent disablement of the lung may take place in atelectasis from compression.

Treatment.—In many cases of atelectasis all that can be done is to treat the underlying condition and to meet symptoms as they arise. In the new-born, artificial insufflation of the lungs by breathing into the mouth may be tried: if the stomach rather than the lungs is inflated, the practitioner should desist from this method of treatment; intubation and blowing directly into the lungs through a rubber tube would seem to be a rational procedure in these cases. In cases of low fever the patient should not be allowed to lie upon the back, and should from time to time take deep inspirations as a prophylactic measure. In most cases of serious atelectasis it is especially important to support the heart.

EMPHYSEMA.

Definition.—Total or partial enlargement of the lung, sometimes from the presence of air in the interstitial tissue, but usually from dilatation and fusion of the alveoli, resulting in a diminution of the respiratory and vascular surfaces, and producing dyspnœa and cyanosis from imperfect aeration of the blood.

Etiology.—Interstitial emphysema is to be distinguished from essential emphysema both in causation and in results. In *interstitial emphysema* air is present in the interstitial tissue of the lung in consequence of rupture of the respiratory surface, which usually takes place suddenly from a violent expulsive effort when the glottis is closed, as in a severe paroxysm of whooping-cough or in violent vomiting. With the renewal of the increased intra-alveolar pressure more and more air is forced into the interstitial tissue. Interstitial emphysema may result also from the aspiration of air when the lung is lacerated by a broken rib or in cut-throat. *Essential, alveolar*, or *vesicular emphysema* is caused by a weakness of the alveolar wall and an increase of intra-alveolar pressure. The weakness of the wall is especially attributable to deficient or degenerated elastic tissue. The deficiency of elastic issue is, perhaps, of congenital origin, since repeated instances of the occurrence of emphysema have been observed in certain families. Degeneration of the elastic fibres is likely to result either from inflammation of the lung or from prolonged intra-alveolar pressure, as in forced expiration, perhaps also from intensified inspiration, as in the prolonged inhalation of rarefied air. Important in the causation of increased alveolar pressure are chronic and violent coughing, as in chronic bronchitis, frequent attacks of asthma, excessive strain of the voice, but especially those occupations which demand persistent, prolonged expiration, as the use of wind instruments. Emphysema occurs at all periods of life, but is most common in middle age, and its frequency increases with advancing years.

MORBID ANATOMY.—In interstitial emphysema large and small bubbles of air are to be seen beneath the pleura and between the lobules, and follow the course of the interstitial tissue along the bronchi and blood-vessels towards the root of the lung. The inflation of the fibrous tissue may thence extend into the neck and appear beneath the skin as a subcutaneous emphysema, which may be continued over the entire body. If the overlying pleura is also ruptured, the air escapes from the interstitial tissue into the pleural cavity, and pneumothorax results.

The changes occurring in alveolar emphysema are distributed throughout the lungs or are limited to certain portions, in which latter case the emphysema is called *vicarious*, *collateral*, or *complementary*, since it represents the distention of a limited portion of a lung in consequence of the inability of air to enter neighboring parts. The emphysematous lung remains distended from loss of elasticity, is abnormally pale from the presence of fewer blood-vessels, and has a downy feel, which is often the most conspicuous characteristic of the emphysema of old age, *senile emphysema*. The alveoli as seen through the pleura appear unusually large, and trabeculated cavities may be present of the size of a hen's egg, due to the fusion of numerous dilated alveoli. The distention and fusion of the alveoli are most extreme where the lungs most readily yield,—namely, at the apices and along the anterior border. The wall between adjoining alveoli is early perforated and eventually absorbed, and in consequence a destruction of the pulmonary capillaries takes place corresponding to the extent of respiratory surface affected. The mucous membrane of the larger bronchi usually presents the characteristics of chronic bronchitis, and the smaller bronchi are often slightly dilated. The right side of the heart is hypertrophied and dilated, and evidences of chronic endarteritis are frequent in the pulmonary artery. In protracted cases, when degeneration of the right ventricle follows hypertrophy, there are nutmeg atrophy of the liver and cyanotic induration of the kidneys.

SYMPTOMS.—When emphysema has reached a sufficient degree to produce discomfort it is manifested by short, wheezing respiration and by dyspnœa on slight exertion. If the emphysema occurs acutely, as in the interstitial variety, the dyspnœa is quickly followed by cyanosis. In chronic emphysema, however, cyanosis is a late manifestation, and is dependent on the progressing incompetency of the hypertrophied right ventricle. There are frequent cough from the associated bronchial catarrh, disturbance of digestion, emaciation, and eventual dropsy. The patient with pronounced alveolar emphysema has a barrel-shaped chest, except in senile emphysema, in which the chest expands but little on inspiration. The back is rounded, the supraclavicular fossæ are distended by the pulmonary apices, the intercostal spaces are widened and protruded on expiration, and a wreath of dilated small veins is frequent in the region of the insertions of the diaphragm in the thoracic wall. There is increased resonance on percussion, and the resonant area extends abnormally low, both laterally and in the back. Cardiac dulness is largely replaced by the resonance of the superjacent lung, except in senile emphysema. The area of hepatic dulness also is diminished, and the anterior border of the liver often lies below the costal cartilages. On auscultation the respiratory murmur is feeble, and there is prolonged expiration. From the frequently associated bronchitis, moist and dry râles are common. On auscultation of the heart there is accentuation of the second pulmonic sound, and in the later stages of emphysema a systolic murmur due to relative insufficiency of the tricuspid valve is to be heard near the ensiform cartilage. The urine eventually presents the characteristics to be found in chronic passive congestion of the kidneys.

The diagnosis of essential emphysema is readily made from the physical characteristics of the patient, and the prognosis is unfavorable as to recovery, although the disease is not incompatible with prolonged life. If death does not result from intercurrent disease, it usually follows progressive weakening of the heart.

TREATMENT.—The treatment of emphysema is chiefly that of the disease which produces it, hence in a large proportion of cases it is the treatment of chronic bronchitis or of asthma. As the addition of the emphysema to the original disease makes a serious complication, all that is possible should be done to avoid the recurring attacks of the original disease. Thus, the existence of emphysema in a case of chronic bronchitis makes it imperative, if possible, to change the residence to a suitable climate, and the musician may be obliged to give up the use of the wind instrument. As in very many cases of emphysema the right side of the heart is weak, the cardiac tonics play an important part in the treatment of the disease; strychnine, acting as it does simultaneously on the circulation and the respiration, is an especially important remedy, and may be given for a great length of time continuously in slowly ascending amounts until massive doses (one-twelfth of a grain) are reached. It is especially important in these cases to treat the secondary disorders of function produced by the venous engorgement. Digestive disturbances must be carefully attended to.

In Germany the mechanical treatment of Gerhardt is said to be much used in emphysema with marked benefit. It consists in having expiration mechanically assisted by compression of the thorax; about ten minutes every day a muscular person places the hands flatly upon the sides at the bottom of the thorax of the patient and compresses actively at the end of expiration. The breathing of rarefied air by means of Waldenburg's portable cabinet is said also in many cases to give great relief. It is even asserted that an improvement in the physical signs of emphysema can sometimes be demonstrated as the result of systematic pneumatic treatment.

ACUTE PNEUMONIA. LOBAR PNEUMONIA. FIBRINOUS PNEUMONIA. CROUPOUS PNEUMONIA.

DEFINITION.—An acute infectious disease pursuing a more or less typical course, caused by the invasion of the lung by a variety of bacteria, chiefly the diplococcus pneumoniæ, characterized anatomically by a fibrinous inflammation of the lung and clinically by continued fever, by dyspnœa, and by a variety of symptoms due in part to the absorption of toxins from the diseased organ.

ETIOLOGY.—Pneumonia occurs in all parts of the world, but with various frequency. In Norway, according to Holmsen, from four per cent. to eleven per cent. of the entire population are infected, and Bary stated that from three per cent. to four per cent. of the patients in a number of hospitals in St. Petersburg had pneumonia. In those regions in which it is prevalent about seven per cent. of all deaths are due to this disease,—for example, eight per cent. in Massachusetts and seven per cent. in Louisiana. It occurs more often in winter and spring than at other seasons, and is found in men four times as often as in women. It is especially frequent in young adults, and the pneumonia of children under two years of age, according to Southworth, is of a lobar character in one-third of the cases. The weak and debilitated, whether from lack of food, overwork, bad hygienic surroundings, disease, or old age, and those exposed to cold and wet, are oftenest attacked. It is common in malarial regions. Although gout is supposed by many English writers to be of etiological importance, the experience at St. Thomas's Hospital opposes this view. Injuries, such as falls and blows, are occasionally followed by pneumonia, the contusion apparently diminishing the power of the individual to resist the activity of the more immediate cause.

The infectious nature of pneumonia was inferred long before the discovery of the micro-organisms which have been found to be of etiological importance by the frequent typical course, by the independence of the general symptoms and pulmonary lesions, by the occurrence of epidemics and endemics, and by its outbreak in persons soon after their exposure to other cases. The observations of Eberth, Koch, Friedländer, Leyden, Günther, Fraenkel, and others have demonstrated that bacteria are always present in the exudation of pneumonia, and that in about eighty per cent. of all cases a definite variety, the diplococcus pneumoniæ, is to be found. This organism has been proved to be identical with one which had been isolated in the sputum by Sternberg and Pasteur. It has been found not only in the inflamed lung, but also in the blood of patients with pneumonia. It has been observed in the numerous complications of pneumonia, as pleurisy, pericarditis, endocarditis, meningitis, peritonitis, and arthritis, and in various suppurative inflammations occurring in this disease and independently of it. Many of these localized inflammations have been produced experimentally by the introduction into the tissues of this diplococcus. Pneumonia, therefore, is to be regarded merely as one of the results, although the most frequent, of the invasion of the individual by this organism, the inflammation of the lung being the local manifestation of its entrance into the body. Other bacteria have been found in pneumonia,—namely, Friedländer's bacillus, the influenza bacillus, the streptococcus pyogenes, the staphylococcus aureus, and the typhoid bacillus in the pneumonia of typhoid fever. The presence of any of these as the especial bacteriological characteristic of the disease is exceptional as compared with that of the diplococcus pneumoniæ.

This diplococcus, also called pneumococcus and micrococcus lanceolatus, usually appears in pairs, the individual cocci being rather ovoid than round, and their outer ends are somewhat pointed. The organism is stained readily by any of the aniline dyes and by Gram's stain, by means of which it is to be distinguished from the pneumobacillus of Friedländer. An important diagnostic feature is the presence of a capsule, which can be determined by appropriate staining.

MORBID ANATOMY.—The characteristic appearances are due to the accumulation in the alveoli and smaller bronchi, more rarely in the large tubes, of fibrin and cells, which in favorable cases undergo absorption. The progress is divided arbitrarily into three stages, namely, congestion or engorgement, hepatization, and resolution, although each of the three stages may be present simultaneously in the same lung. Ordinarily an entire lobe is diseased: hence the term *lobar pneumonia*. Sometimes two lobes are inflamed, and in about one-sixth of the cases both lungs are involved. In three-fourths of the cases the lower lobe is affected,—oftenest of the right lung,—and in one-half of the cases it is the only lobe diseased. The upper lobe is inflamed in two-fifths of the cases, being alone diseased in about one-fifth of them.

In the stage of *engorgement*, which usually lasts about twenty-four hours, although it may be prolonged for several days, the affected portion of the lung is distended, dark red, heavy, and dense. On section there escapes a somewhat viscid, bloody fluid, the result of combined congestion and œdema, and containing many small air-bubbles. Microscopical examination of the lung at this stage shows that the alveoli are filled with an albuminous fluid in which are desquamated swollen epithelium, red blood-corpuscles, and leukocytes. The capillaries are injected and tortuous.

The second stage usually begins on the second day of the disease, and is called *hepatization*, from the fancied resemblance of the diseased lung to the liver. The alveoli contain fibrin in addition to cells. The distended lung is increased in weight perhaps threefold. The heavy portions sink in water, and are non-crepitant and friable. Transverse depressions are to be seen upon the surface of the lung, caused by the pressure of the ribs, and a fibrinous membrane covers the pleura, which consequently is dull and opaque. On section the surface is either red or gray, according to the quantity of blood present, which is greater in the early and less in the late stage of the hepatization. The cut surface appears granular from the projection of the clotted fibrin in the alveoli. Ordinarily the granules are minute, but in an emphysematous lung they are large. The exudation may be dark red in case hemorrhage is a complication, and the lung is dotted with black spots when there is excessive carbonaceous pigmentation. The interstitial tissue is frequently swollen and opaque from the presence of the exudation in the course of the lymphatics. In old people and in those enfeebled by chronic disease the quantity of fibrin in the exudation is not as abundant, consequently the lung is less dense and more moist.

In *resolution* the lung is less solid and resistant, and the cut surface exudes on pressure an opaque puriform fluid, and appears smooth instead of granular. These changes are due to the fatty degeneration and softening of the exudation. The lung-tissue is easily crushed, the puriform fluid fills the gap, and thus the presence of an abscess is frequently simulated.

Although hepatization is followed normally by resolution, if for any reason the blood-supply of the inflamed portion of the lung is interfered with gangrene readily occurs from the passage of putrefactive bacteria in the bronchi into the diseased part. The gangrenous portions may be separated from the rest by an inflammatory line of demarcation, and an abscess result in which the necrotic tissue lies loose. In rare instances the hepatized lung does not undergo resolution, but forms a reddish-gray, homogeneous, airless mass. (See Chronic Pneumonia.)

The associated lesions are due to absorption of the bacterial toxin into other organs of the body, or depend upon the direct entrance of the bacteria into the circulation and their development in some other organ. The lymphatics at the bifurcation of the trachea are large, soft, and injected; the spleen also is large and soft and of a reddish-gray color from hyperplasia of the pulp. Granular degeneration of the heart, liver, and kidneys is usually present. Pericarditis is common when the left lung is attacked, endocarditis, either simple or ulcerative, may be present, and diplococci are usually to be demonstrated in the exudation or in the diseased valves. A rare complication, and then associated with endocarditis, is inflammation of the pia mater, in which case the diplococcus of pneumonia is found in the meningeal exudation.

SYMPTOMS.—For two or three days before the onset of pneumonia there is in a certain number of cases a nasal or pharyngeal catarrh or slight general discomfort. The first symptom of the affection of the lung is a chill, generally sudden, and mild or severe irrespective of the course which the disease is to take. The chill is soon followed by fever and thoracic pain unless the inflamed portion of the lung is deep-seated. The pain is usually referred to the nipple of the affected side, but may be located in the axillary region or in the back. Dyspnœa then takes place, is perhaps synchronous with the pain, and is due probably to the engorgement of the lung with frothy, bloody fluid, the presence of which is made evident by numerous coarse and fine, moist and dry râles. As the solidification of the lung progresses, the pain lessens, but the fever and dyspnœa persist. There is but little appetite, thirst is not extreme, constipation is the rule, and the occurrence of diarrhœa, especially late in the disease, is indicative of the serious nature of the attack. The face is flushed and the expression anxious; the respiration is superficial, labored, and often interrupted by a distressing short cough. The expectoration is scanty, tough, and viscid, and of a rusty color. The patient is restless, perhaps delirious, and suffers from headache, backache, and weakness. The pulse throughout is full and strong, and in the beginning there is marked pulsation of the carotids.

Resolution takes place commonly between the fifth and the eighth day, rarely earlier, often later. The temperature then falls several degrees, either suddenly, by *crisis*, or gradually, by *lysis;* the dyspnœa diminishes, the cough is less distressing, the expectoration is more abundant, and the general symptoms improve, although the signs of solidification continue, as a rule, for a number of days afterwards.

The temperature rises rapidly after the chill, and within twenty-four hours is generally 104° or 105° F. It usually remains thus elevated, with slight morning remissions and evening exacerbations, until the critical fall, which oftenest occurs on the seventh or eighth day, but may be delayed for several days. At times a *pseudo-crisis* appears on or about the third or fourth day, but the temperature rarely reaches the normal and quickly returns to the maximum. In a certain number of cases the temperature falls gradually, attaining the normal point in the course of a few days instead of in a few hours. Persistence of high temperature beyond the time when the critical fall is to be expected, or the appearance of wide variations in the daily range of temperature, should arouse suspicion of a complicating gangrene or suppuration.

The frequency of respiration in most cases is between thirty and forty per minute, the number of respirations being higher in children, in weak and in nervous patients, and in cases where large portions of the lung are involved.

The pulse is usually between 110 and 120, not increasing in proportion to the frequency of respiration. The rate may be considerably faster than this in children and nervous persons, and be below 100 in elderly people. A fall of forty or fifty beats at the time of the crisis is not unusual. In the earlier days of the pneumonia the pulse is full and bounding, but as resolution approaches it is soft, compressible, and perhaps dicrotic.

The cerebral symptoms are especially marked in children and in alcoholic persons. In children convulsions may be among the incipient symptoms of pneumonia, delirium is early among abusers of alcohol, and delirium tremens is likely to occur late in the disease. Mild delirium is frequent among elderly people and persons of a nervous temperament.

Physical Examination.—The patient is found lying on the back or the affected side according as the pain makes it necessary to limit the movements of the chest. The body is bent forward, the head is raised, and the nostrils are expanded with each inspiration. There is a bright-red flush on each cheek, while the lips and nose are dusky and the rest of the skin is pale. Rarely there is a slight degree of jaundice. As a rule, there is profuse sweating, frequently accompanied with sudamina; herpes of the lips or nose is present in about one-third of the cases.

The examination of the chest shows a limitation of movement on the affected side. Palpation gives an increase in the vocal fremitus unless there is also pleuritic effusion. In the early stages friction may often be felt. During the stage of congestion the resonance on percussion is likely to be increased, perhaps tympanitic, but with the development of hepatization is replaced by dulness, almost flatness, oftenest first recognized in the subscapular region and near the posterior axillary line. If aerated lung-tissue overlies the hepatized portion of the lung there is resonance on percussion. As resolution advances, the dulness gradually disappears.

On auscultation in the stage of congestion fine crepitant, so-called subcrepitant, râles are to be heard, caused by a viscid exudation in the alveoli, and frequently accompanied by coarse moist râles produced in the bronchi. As hepatization progresses, the râles disappear and the breathing becomes tubular, or ceases in case the larger bronchi are obstructed. When resolution takes place there is a return of the fine moist râles,—*crepitus redux,*—and coarser râles soon follow. In central pneumonia the transmission of the râles is interfered with by the overlying aerated lung. On auscultation of the heart the second pulmonic sound is accentuated, and on palpation and percussion in the region of the spleen and liver these organs are found frequently enlarged.

The sputum of pneumonia is usually characteristic of the disease. It is sometimes scanty, and may be wholly absent. At the outset it is streaked with blood and is so viscid as to adhere to the side of the inverted cup. During hepatization it is more abundant, gelatinous, of a reddish-yellow color from the presence of decomposed blood-pigment, hence called *rusty.* In severe cases, when there is abundant hemorrhage, the sputa are liquid and of a dark-brown color resembling prune-juice. During resolution they are thick and yellow, muco-purulent in character. In certain cases the sputum is green,—according to Von Jaksch, from the transformation of hæmoglobin into bilirubin. Fibrinous casts of the bronchioles and alveoli are often present, and may be recognized in the sputum diluted with water and spread upon a glass plate. Diplococci of pneumonia are to be found often in the sputum, but their presence is not necessarily of diagnostic value, since they may be seen when there is no pneumonia.

Examination of the blood shows almost invariably a leukocytosis, which, as a rule, becomes greater as the disease advances. According to Ewing, it is usually between twenty thousand and thirty thousand, and, in general, the higher the leukocytosis the more severe the disease; but, exceptionally, in very grave cases there may be little or no increase in the number of leucocytes. In the absence of all physical signs permitting the localization of a suspected pneumonia, the occurrence of a marked leukocytosis is in favor of such a condition with a central seat. Sudden increase of an existing leukocytosis occurs when the disease extends to a hitherto unaffected portion of the lung. The number of leukocytes rapidly returns to the normal after the crisis is reached.

The urine is scanty, high-colored, and concentrated. The chlorides become very much diminished early in the attack, but return after the crisis. Albumin is found in the urine in small amounts in about one-third of the cases, and is associated with the presence of hyaline casts. Both albumin and casts usually disappear in the course of a few days after convalescence. According to Von Jaksch, the appearance of peptone in the urine is indicative of the beginning of resolution.

VARIETIES.—All cases of pneumonia do not run the same typical course, and the genuine, frank, sthenic pneumonia has been long distinguished from the atypical, asthenic, or typhoid pneumonia. It seems not unlikely that the variation in the course may be due in part to differences in etiology, the more common typical variety being caused by the diplococcus of pneumonia, while those pursuing an atypical course, or occurring as complications of other diseases, as typhoid fever, malaria, or nephritis, may result from the bacilli of typhoid fever or of influenza, or from staphylococci or streptococci, either alone or in combination with the diplococcus. The typical course of genuine pneumonia also varies frequently in consequence of the age and the general condition of the patient, and the presence of complicating diseases. In children, in whom, as already stated, lobar pneumonia is much more common than has been generally supposed, there are rarely typical consolidation and physical signs. The initial chill is often absent, perhaps replaced by a convulsion, and the mortality is low. In elderly people, on the other hand, the chill and characteristic signs of hepatization may be absent, but the disease is very fatal. In alcoholic subjects the symptoms and signs referable to the chest may be so inconspicuous that the pneumonia is overlooked, delirium being the striking feature. In typhoid fever the course of the pneumonia is greatly modified by the toxic condition due to the typhoid bacillus. The term *typhoid pneumonia* indicates merely the association of low, muttering delirium, extreme prostration, a dusky, perhaps yellow skin, and diarrhœa, conditions which occur in varieties of pneumonia independent of typhoid fever and of the typhoid bacillus.

In malarial regions it is observed that consolidation of the lungs appears insidiously, the typical symptoms being absent, resolution is slow, termination in abscess is frequent, and the mortality is high. In rare instances the symptoms and signs of pneumonia disappear at the end of two days, and the patient is said to have had an *ephemeral pneumonia*, or an attack of congestion of the lungs. In other cases resolution takes place in the course of twenty-four hours, and recovery and convalescence begin from the third or fourth day. To this condition the term *abortive pneumonia* is applied. *Apical* and *central* pneumonias are discriminated. In the former the infiltration begins in the upper lobe, and frequently pursues a severe course, associated with severe cerebral symptoms,—hence *cerebral pneumonia*. In central pneumonia, although the symptoms indicate an inflammation of the lung, the physical signs, with the exception of localized, apparently deep-seated, bronchophony and bronchial breathing, are absent. In *wandering pneumonia*, which sometimes, though not necessarily, is preceded by facial erysipelas, one portion of the lung after another is continuously involved. The discovery of streptococci in the exudation in certain of these cases suggests that the peculiar course may be due to this variety of bacterial infection.

COMPLICATIONS.—The complications are due either to the direct extension of the inflammatory process to neighboring organs or surfaces, or to the lodgement and development of the diplococci in distant organs. Bronchitis is one of the most frequent accompaniments of pneumonia, and its presence is indicated by coarse moist râles. The presence of pleurisy is so constant that the term *pleuro-pneumonia* is often used as a synonyme of pneumonia. In pneumonia of the central portions of the lung, however, the pleura is free from alteration. The variety of pleurisy oftenest observed is the dry or fibrinous pleurisy, the fibrinous exudation forming a thick or thin layer on the surface. There is usually no considerable quantity of serous fluid in the pleural cavity, but in certain instances pus is present, and the case then runs the course of an empyema. The pericardium is often inflamed, especially in pneumonia of the left lung, and, although the pericardial exudation is generally fibrinous and sometimes very scanty, in rare instances there may be large quantities of fluid, which may be of a sero-purulent character. Endocarditis, especially of the aortic and mitral valves, is a frequent complication of pneumonia, and the characteristic diplococci have been found repeatedly in the vegetations. According to Osler, nearly twenty-five per cent. of the cases of malignant endocarditis occur in this disease. Arterial embolism may thus arise as a complication, and is perhaps explanatory of the reported occurrence of symmetrical gangrene in the sequence of pneumonia. As has been stated previously, meningitis with pneumococci in the exudation at times occurs independently of pneumonia, and inflammation of the lung is a frequent condition in cerebro-spinal meningitis. Peripheral neuritis has been observed as a complication of pneumonia, as have also parotitis, arthritis, and orchitis, and the diplococci of pneumonia have been found in the accompanying exudation. Nosebleed and intestinal hemorrhage at times occur late in the disease, and acute nephritis, with hæmaturia as a complication, occasionally is present. There may be profuse flowing when miscarriage or premature labor occurs.

DIAGNOSIS.—The onset of an attack of pneumonia is usually so typical, and the development of the physical signs so marked, that but little difficulty is encountered in the diagnosis of most cases. When, however, the localization is central, the physical signs are often so obscured by the overlying aerated lung that the diagnosis is to be inferred almost wholly from the symptoms and from the exclusion of other causes of thoracic disease. If the patient is seen first at a late stage, the diseased lung may be covered with a serous or a purulent exudation and the signs of consolidation be concealed until the removal of the pleuritic exudation permits them to be transmitted to the ear. Attention has been called already to the diagnostic importance of examination of the blood in such cases. The progress of the pneumonia may be so retarded that consolidation is delayed for several days, especially in children, in old people, and in those suffering from chronic disease. The sudden onset of cerebral symptoms in children and of delirium tremens in persons addicted to alcohol, when associated with fever and rapid respiration, necessitates always a physical examination of the chest.

Acute œdema of the lungs closely simulates the early stage of pneumonia. There are dyspnœa, frothy, bloody sputum, and fine moist râles. In œdema, however, fever is absent, and there is evidence usually of valvular endocarditis. The râles are present throughout the chest. and not limited to a lobe of the lung as in pneumonia. The solidification of the lung in acute pulmonary tuberculosis is occasionally mistaken for acute pneumonia, and the bacilli of tuberculosis may be absent from the sputum until pulmonary hemorrhage or softening arises. The febrile course of acute tuberculosis is less typical than that of pneumonia, resolution fails to take place at the usual time, and the characteristic bacilli appear eventually in the sputum. Acute pleurisy is often confounded with pneumonia, for its onset may be equally sudden, and announced by a chill, thoracic pain, and dyspnœa. In both diseases dulness on percussion, bronchial breathing, bronchophony, and crepitation occur. In pleurisy there is usually displacement of the heart or of the liver. and tactile and vocal fremitus are faint or absent. The abnormal respiratory sounds are confined for the most part to the upper part of the dull area. while in pneumonia they are more marked in the lower portion of the region of dulness. In case of continued doubt the chest should be punctured with the exploratory needle.

PROGNOSIS.—Although it has been estimated that between one-sixth and one-fourth of all cases of pneumonia prove fatal, the statistics aid but little in the prognosis of the individual case, owing to the importance of the peculiarities of the individual, especially as determined by age, habits, and complicating diseases. Children generally recover from fibrinous pneumonia. In persons above sixty the mortality is high. In alcoholic subjects the mortality may be even fifty per cent. The prognosis of pneumonia is serious when it is a complication of emphysema, heart disease, nephritis, diabetes, or advanced pregnancy. The more extensive the area of lung involved the graver the prognosis, it being especially severe in double pneumonia. The mortality in pneumonia of the upper lobe, so often associated with cerebral disease, is generally considered higher than in pneumonia of the base, although numerous exceptions exist. In asthenic or in bilious pneumonia with typhoidal symptoms the prognosis is far less favorable than in typical fibrinous pneumonia.

Death takes place usually during the stage of red hepatization, and shortly before the expected crisis, although now and then it occurs during the incipient stage of engorgement, or soon after crisis has taken place. It is generally the result of cardiac incompetency and asphyxia, due in part to the effect upon the nervous system of the toxin of the diplococcus of pneumonia, and in part to the solidification of the lung. The ominous symptoms are a persistent increase in the frequency of the pulse and respiration, tracheal râles, prune-juice sputum, stertorous breathing, cyanosis, and muscular tremor. The grave prognostic importance of extreme, slight, or absent leukocytosis has been previously mentioned.

TREATMENT.—In the treatment of pneumonia it is essential to recognize that, though the disease may be a unit from the pathological point of view, therapeutically it comprises essentially diverse diseases. A pneumonia whose physical signs cannot be made out in the beginning, but gradually creep up towards the chest-wall,—a pneumonia whose expectoration is in the beginning prune-juice, whose crepitant râle is never typical, whose physical signs are obscure until complete consolidation gives percussion dulness,—or a pneumonia occurring in the alcoholic, in the old, in the victim of renal disease, in the broken-down debauchee, in the worn-out city merchant or professional man,—is in its management essentially distinct from a pneumonia the result of exposure of a strong, healthy countryman to a Western blizzard or other cold. Hence the folly of statistical inquiries into the mortality of different methods of treatment of pneumonia,—statistics in which all the forms are lumped together as if they were one disease. In one form of pneumonia sedative treatment may kill the patient; in another form sedative treatment at the beginning of the attack may be necessary for the saving of the patient. When in the first twenty-four hours of a pneumonia there is violent constitutional reaction, with flushed face, rapid and noisy breathing, bloody sputa, intense headache and drowsiness, a hard bounding or a tense corded pulse, venesection may markedly lessen all the symptoms, and if combined with dry cupping over the whole chest may, we believe, lessen the amount of engorgement of the lung and the final area of consolidation.

During the bleeding the patient should sit up, and the blood should be taken rapidly from a large orifice until some impression is made upon the pulse, or until twenty-five or even thirty ounces have been abstracted.

Nevertheless, if it were possible in any way to obtain the immediate effects of the venesection without the after-exhaustion, such procedure would be preferable to blood-letting. Veratrum viride in full dose, by reducing the force of the heart, diminishes the *vis a tergo* which drives the blood to the lungs, and at the same time dilates the abdominal blood-vessels and so invites the blood into them. As these abdominal blood-vessels can, when fully relaxed, contain all the blood of the body, the action of veratrum viride is decisive. Its influence, however, lasts but a few hours, so that its withdrawal is rapidly followed by return of the circulation to the norm without exhaustion. The veratrum viride treatment of robust cases of pneumonia bleeds a man into his own blood-vessels, but allows the return of this blood to the circulation when the stage of consolidation is reached without persistent depression to the heart or the vessels themselves. In order to obtain these effects the veratrum viride should be given freely (three to five minims of the tincture, two to three minims of the fluid extract) every forty minutes until nausea is produced. Vomiting should be the signal for its immediate withdrawal. The veratrum viride treatment of pneumonia differs from the older method of Rasori, in which enormous doses of antimony were given, in producing only temporary depression: the antimony in the large doses used caused not only excessive discharge from the stomach, but also violent serous purging, with its consequent exhaustion.

In the great majority of cases of pneumonia as seen in our large cities, active depressing treatment even in the beginning is not useful; and in all cases after consolidation has set in, the efforts of the physician must be directed to maintaining the forces of the patient and mitigating the symptoms.

The usual causes of death in pneumonia are general exhaustion, failure of the right heart, and failure of the respiratory forces. The causes of exhaustion are the high temperature, the loss of material from the blood into the diseased lung, and the blood-poisoning from the secondary products in the inflamed tissue. Over two of these sources of exhaustion we have little or no control; the question how far we should attempt to reduce temperature is vital, but is in practice answered very differently by different practitioners. The fever in pneumonia lasts but a few days: hence a temperature of 103° F., which in a long-continuing pyrexia like that of typhoid fever is serious, is a matter of little importance in a pneumonia, and in most cases may safely be disregarded. If, however, the temperature rise to 104° F. or above, it is important that it be reduced, and we believe that its reduction by the external use of cold is much safer than by large doses of antipyretic drugs.

These drugs in large dose are sedatives to the circulation, but the small dose of antipyrin has no such effect, so that in pneumonia this drug and its allies may be used in small dose to moderate nervousness, to lessen the bodily temperature when it is above the norm, and to prolong the effect of the cool bath in severe fever. No more than five grains of antipyrin should be given at once, nor more than ten grains in the twenty-four hours. Among antipyretic remedies may be mentioned quinine. In order to obtain from it any effect upon the temperature it is necessary to give it in doses of thirty grains a day, which are so disturbing to the patient, and, after all, so ineffective, that their administration does not seem to us justifiable. If employed at all, quinine should be used rather as a stimulant, not over fifteen grains a day.

We believe that in cases of pneumonia much injury is frequently done by the retention of heat by poultices and other applications to the lung. (See Acute Bronchitis.) Cold wet compresses, or even the ice-bag, may be often applied with great advantage over the affected lung; in many cases tepid baths (85° to 90° F.) are of great service if properly used. No exertion on the part of the patient should be allowed: he should be lifted into the bath, or the cot-bath may be employed. (See Typhoid Fever.) The bath is usually followed by sleep, with lowered respiration and a sense of refreshment. In very weak people and in children the temperature of the bath may be a little above 90° F. When the fever is very high the bath temperature may be as low as 80° F. Usually it is better to cool the water while the patient is in the bath than to immerse him directly in cold water.

To prevent exhaustion by maintaining the forces of the patient is the great object of the nursing in a case of pneumonia. Absolute confinement to bed is to be enforced from the beginning of the attack, the patient, however, being allowed to sit up if more comfortable in that position. The sick-room should be kept as quiet as possible; the feeding should be at short intervals (two to three hours); the food should be simple, nutritious, and digestible,—milk, milk products, raw eggs, light meats, such as birds or sweetbreads; if the digestion be very good, a moderate amount of farinaceous food may be allowed, but in most cases it has a tendency to produce flatulence and is objectionable. Cold drinks should be allowed as freely as the stomach will bear.

In the beginning of sthenic pneumonia alcohol is injurious; in the advanced stages it may be used in small quantities with the food to aid digestion, or, if there are evidences of exhaustion or of cardiac failure, may be given freely. At this stage the disease from a therapeutic point of view is allied to an infectious fever, much of the constitutional disturbance being due to the absorption of poisonous products from the affected area: hence stimulants should be given as they would be employed in a similar condition occurring in a low fever. The alcohol is of value as a cardiac stimulant, but cannot take the place of digitalis, strychnine, and cocaine.

In the advanced stages of a severe pneumonia digitalis in doses of from five to fifteen minims of the tincture at intervals of from four to six hours often acts most happily as a heart stimulant and tonic, and seldom, if ever, interferes with digestion. Its effect upon the pulse should be the guide to its administration : whenever the pulse-rate falls to eighty-five, or even to ninety, the drug should be in part or altogether withdrawn, to be resumed when the effects wear off.

As high temperature makes the heart insensible to digitalis, large doses are often necessary, and some care should be exercised at the time of the crisis lest the sudden fall of temperature bring about an over-action of the drug.

Nitroglycerin and amyl nitrite have been strongly recommended by some practitioners as stimulants to the circulation in pneumonia. It should be remembered, however, that in any dose sufficient to produce perceptible effect these drugs always lower the arterial pressure by depressing directly the muscle-fibres in the blood-vessel walls, and that, although their first action upon the heart is that of a stimulant, the slightest overdose converts such action into that of a powerful depressant. Further, their effect lasts but a few moments. It is evident, therefore, that great caution is necessary in their use, and that they should not be employed when vaso-motor weakness is an already existing danger. In sudden cardiac failure they may at times, given in small doses, be of temporary value.

Ammonium carbonate is very largely used, in the adynamic form of pneumonia especially, partly as a stimulant and partly on account of its alleged expectorant properties. There is no reason for believing that it has any direct influence upon the consolidated lung, and its power as a stimulant is certainly inferior. We have seen many hundreds of doses of it given in the disease, and have never been able to detect any effect upon the pulse or the respiratory rate. On the other hand, its free use readily endangers digestion. If given at all it should be in small doses (three grains in emulsion) at intervals of half an hour to an hour. Spirit of ammonia is preferable when the heart gives out suddenly or when an immediate effect is desired. It is, however, also of inferior value.

Very important drugs in the treatment of pneumonia are the four alkaloids strychnine, cocaine, atropine, and caffeine, which act as stimulants both to the circulation and to the respiration. As a cardiac stimulant and tonic strychnine is inferior only to digitalis, and should always be used when there is exhaustion in pneumonia. Cocaine resembles strychnine in its cardiac action ; atropine as a heart stimulant is decidedly inferior, but exceeds both strychnine and cocaine in its influence upon the vaso-motor centres, and is, therefore, especially applicable to those cases in which collapse occurs or is threatened at a time of crisis, at which period its power of checking excessive sweating often gives it further advantage. Of these alkaloids atropine is probably the most active in increasing respiratory movements in the normal man, but it has less power in asserting itself in the face of opposition than has either strychnine or cocaine, and is therefore practically less available. Strychnine seems to be more active and efficient than cocaine, and does not, as does cocaine, produce cerebral excitement. Caffeine cannot be looked upon as a powerful respiratory stimulant, but affects decidedly the cerebral cortex, and therefore, if employed at all, must be used in small dose as an adjuvant, or in special cases when it is desired to overcome stupor.

Of all these remedies strychnine is the most generally available, but in severe cases the best results are to be obtained by the use of both strychnine and cocaine. The strychnine and cocaine should be given alternately, in a bad case, every four hours, so that every two hours one of the remedies will be taken; ordinarily they may be exhibited by the mouth, but when the symptoms are alarming they should be administered hypodermically. Each of them is a stimulant to the heart, and also to the vaso-motor system, so that they do more than simply aid in the maintenance of the respiratory forces. The dose should be increased according to the needs of the case, commencing with one-twenty-fourth grain of strychnine and one-sixth grain of cocaine, slowly increased to one-fourteenth grain of strychnine and one-half grain of cocaine. We have seen life apparently saved by even larger doses than these; but when such doses are employed it is essential to have a judicious trained nurse under orders to reduce or suspend the drug should any evidence of overaction appear. When large doses are given at shorter intervals it should always be hypodermically, so as to insure their immediate absorption.

The special indication for the free use of these respiratory stimulants is cyanosis, with hurried breathing and other evidences of respiratory distress. Under such circumstances the inhalation of oxygen gas sometimes affords temporary relief. Its influence is very fugacious, and its inhalation often more or less irksome to the patient, so that its use should be restricted to times when the respiratory symptoms are very threatening. Some authorities recommend the use of a mixture of oxygen and nitrous oxide. Nitrous oxide, however, is inert, is not decomposed in the system, and does not yield oxygen. It acts, therefore, only as a diluent, and if the oxygen needed diluting it would be better and cheaper to employ ordinary air for the purpose. It is hardly necessary to say that the preference should be given to pure oxygen.

In advanced pneumonia, when cyanosis is very great and is accompanied with distention of the right heart and with rapid respiration and great collateral engorgement of the lung, free venesection is recommended by some authorities. Momentary relief is certainly often obtained. The explanation of this is that the reduction in the amount of blood lessens the congestion of the lung and the work of the failing heart. It is very doubtful, however, whether the ultimate result is good. Osler states that of twelve cases which he bled under such circumstances eleven died, —a result which does not seem equal to that which could have been obtained by the free hypodermic use of digitalis, strychnine, and other cardiac stimulants.

In many cases of pneumonia there is much pain in the chest, with general distress, which are greatly relieved by minute doses of opium in the form of Dover's powder or morphine. Whilst no hesitation should be felt in using opiates in small quantities, care should be exercised not to carry their administration so far as to interfere with an already embarrassed respiration. Hypnotics should be administered without hesitation when there is insomnia; chloral is the most effective, but should never be given when the heart is oppressed; sulphonal is probably the least disturbing, but is uncertain; trional appears to be between the two in its action. Opiates sometimes act better than do any of the modern hypnotics. The combination of chloral (ten grains) with morphine (one-eighth of a grain) is sometimes excellent in its effect.

When in a case of pneumonia the stage of consolidation is reached, the question whether it is possible in any way to hasten the softening and removal of the exudate is of great importance. The ordinary expectorants are of no value. Even large doses of the alkaline expectorants (such as potassium citrate) do not sensibly increase the fluidity of the sputa. Pilocarpine, as suggested by Riess, has some clinical reports in its favor, but we have never used it.

The difficulty of determining in any individual case how far a drug which has been administered hastens resolution is very great; we are, however, inclined to believe that mercury and the iodides have some power. The infective nature of the pneumonic process on theoretic grounds led to the practical abandonment of mercury, but we know now that mercury is a valuable drug in the treatment of serious infectious disease,—witness diphtheria: hence theoretically there is no objection to its use in pneumonia, whilst its proved power in hastening the breaking down of exudations seems to indicate its employment. Certainly, however, if either mercury or the iodides be given, the doses should be so small that they can have no influence in deranging digestion or in increasing the general weakness.

There are often in cases of pneumonia troublesome symptoms which must be met: headache is to be relieved by cold applications to the head,—sometimes by caffeine, or even by antipyrin, used cautiously; if need be, hypodermic injections of morphine and atropine may be given. Such injections, also, are sometimes of advantage in relieving chest-pains. So far as the cough is concerned, the principles discussed in the article on Acute Bronchitis are applicable. Local applications to the chest are often useful in pneumonia. When venesection is not practised, or a little later in the pneumonia when there seems to be an excessive collateral congestion, cut cups are often very serviceable. They are especially useful when there is much pleurisy. Dry cups are often of value: like sinapisms and other rubefacient applications, they affect not the pneumonia itself, but the congestion of the lungs which surrounds the absolutely diseased part. Poultices are especially valuable in children. (See Catarrhal Pneumonia.) Blisters are of great service when there is pleurisy with the pneumonia, and sometimes may be advantageous in overcoming collateral effects of the disease.

In the treatment of adynamic pneumonia it is essential to begin the use of stimulants very early and to push these remedies steadily through the disease in the largest doses that can be borne. Alcohol, digitalis, strychnine, cocaine, and atropine are the drugs upon which reliance must be chiefly placed. Musk has a certain limited value, and we have seen it apparently save life. As stated many years ago by Trousseau, it is especially effective in the pneumonia of alcoholics. As most of the musk of the market is inert, the utmost care should be taken to get as pure an article as possible: if it does good in any individual case, it will quiet the delirious and nervous excitement and bring about sleep; if it does not produce distinct effects, there is no use in continuing its administration. We have seen, as an example, a drunkard wildly delirious from pneumonia receive a rectal injection of fifteen grains of musk and pass into a quiet sleep of five or six hours' duration, during which time food was taken regularly, then awake furiously maniacal, to be again subdued by another dose of musk, until, thus tided over the period of greatest danger, the patient finally convalesced.

In 1892, Klemperer instituted a series of experiments having for their object the treatment of pneumonia as an infectious disease by an antitoxin. It is affirmed that rabbits can be immunized against the pneumococcus, and in a number of cases human pneumonia has been treated by injection with the serum obtained from cases of pneumonia in man. The best result that has been claimed for the method is that it hastens very greatly the development of the crisis. In trials made in Philadelphia with the treatment the effects of the injections were distinctly evil, and at present the method is simply in the stage of preliminary experiment; its use is not justified except for the purposes of research in the hands of experts.

CHRONIC FIBROUS PNEUMONIA. CHRONIC INTERSTITIAL PNEUMONIA.

Definition.—An obliteration of the lung-tissue due to the presence of an increased quantity of fibrous tissue of inflammatory origin.

More or less confusion has resulted from the attempts to include under chronic interstitial pneumonia the terminal results of various pathological processes in which fibrous tissue is found in the lungs. It may grow from the alveolar wall or from the interstitial tissue, whether between the lobules, beneath the pleura, or along the bronchi and blood-vessels. It is present often in small quantity and limited to a part of the lung, and again may be abundantly distributed over a lobe or throughout both lungs. This fibrous tissue forms a scar, filling the gap due to gangrene, abscess, the necrosis of tuberculosis, or the destruction of a gumma, or causes the obliteration of an atelectatic portion of the lung. It encapsulates animal and vegetable parasites which have invaded the lungs, and becomes increased in the vicinity of the various tumors which develop in the lung. In chronic pleurisy it may extend from the inflamed pleura into the compressed lung and permanently prevent its expansion.

New-formed fibrous tissue also may be distributed in greater or less abundance along the bronchi in the chronic bronchitis resulting from the continuous inhalation of irritating particles of dust in certain trades, the *pneumonokoniosis* of Zenker. The varieties of dust principally concerned are coal-dust among workers in coal, causing *anthracosis*, particles of steel among scissor-grinders and file-makers, causing *siderosis*, and bits of sand among stone-cutters, producing *chalicosis*. The inhaled dust enters the interstitial tissue of the lung, where it in part remains, and from which it in part is carried by means of the lymphatics both to the pleural surfaces and to the bronchial lymph-glands. Abundant dust produces increased weight and density of the lung and peculiar modifications of color,—black from coal, reddish yellow from iron or steel, and grayish white from sand. In chalicosis gritty particles may be felt with the knife on section of the lung. In consequence of the long-continued inhalation of the dust, chronic bronchitis and interstitial pneumonia take place. The thickened fibrous tissue is either diffused or distributed in the form of nodules, the latter being due to the induration of lymph-follicles or to the transformation into fibrous tissue of the foci of broncho-pneumonia. Bronchiectasis and emphysema are also results. The patient suffers from chronic cough, with profuse expectoration and progressive emaciation. In anthracosis the sputum is especially characteristic, being of a constant dark-gray or black color from the presence of particles of carbon. In consequence of the chronic cough and persistent emaciation the cases have been grouped under phthisis, and designated coal-miner's phthisis, scissor-grinder's phthisis, or miller's phthisis, according to the trade especially concerned. Pneumonokoniosis is essentially a severe chronic bronchitis due to a particular cause, and ending in bronchiectasis, emphysema, and fibrous pneumonia. It includes the conditions which, when extensively distributed, Corrigan described as *cirrhosis of the lung.*

The term fibrous pneumonia, however, when used to characterize a disease, should be restricted to the rare termination of acute pneumonia in carnification instead of resolution, gangrene, or abscess. This condition was recognized by Laennec, but since his time has usually been overlooked.

ETIOLOGY.—The etiology of genuine fibrous pneumonia is presumably that of acute fibrinous pneumonia, of which it represents a terminal stage. Little or nothing is known of the cause or causes of such a termination. It may be that other bacteria than the diplococcus of pneumonia were concerned in the acute attack. Marchand suggests that previous disease of the lung, by causing induration and pleural adhesions, may interfere with the absorption of the fibrinous exudation of acute pneumonia and thus promote the formation of fibrous tissue. According to this observer, genuine fibrous pneumonia is to be found in persons addicted to alcohol, and in persons poorly nourished and in bad hygienic surroundings.

MORBID ANATOMY.—The affected portion of the lung is distended, dense and heavy, and exceedingly resistant to pressure. The pleura is thickened and opaque. The cut surface of the lung is of a pale reddish-gray color, translucent, smooth, or slightly granular. Later in the disease the color of the lung is still paler, and the cut surface shows numerous minute yellow specks, due to the fatty degeneration of the cells in the alveoli. The bronchi contain an opaque fluid, and there is a visible increase of the peribronchial fibrous tissue and that around the blood-vessels. The microscopic sections show a thickening of the alveolar wall and coherent, fibrillated casts resembling those of fibrinous hepatization, but composed of vascularized granulation-tissue. These are the alterations characteristic of *carnification*, and in genuine fibrous pneumonia may be found as early as three weeks after the onset of the acute pneumonic symptoms. But little is known of the nature of the permanent alterations of the lung which are found in cases of apparent recovery. It is probable, however, that interlacing bands of fibrous tissue, obliterated alveoli, dilated bronchi, and vesicular emphysema are results of the process.

We are indebted to Heller for our knowledge of a congenital fibrous pneumonia affecting symmetrically both lungs and due to syphilis. The fibrous tissue is diffused throughout the lungs, forming a coarse mesh-work by which the alveoli are narrowed. In consequence of this affection the child may die at or immediately after birth. If it survives, the right heart becomes hypertrophied. There is an especial liability to bronchitis and pleurisy, but the subject may reach adult life.

SYMPTOMS.—The symptoms at the outset of this variety of pneumonia in no way differ from those of typical pneumonia. The temperature even may fall at the usual time by crisis or lysis, but rapid breathing and cough persist, and the temperature soon rises one or two degrees, remaining elevated for weeks, although the appetite improves. The pulse is in the vicinity of 100, and there is a sero-purulent sputum. The physical examination of the chest shows that the dulness, bronchial breathing, and bronchophony caused by the hepatized lung continue, and, in addition, fine and coarse, moist and dry râles are to be heard over the affected region. These conditions persist with but little change for weeks and even months, when, in favorable cases, the fever slowly disappears, the cough diminishes, and the patient improves in strength. There is usually a progressive shrinkage of the chest, but the disappearance of dulness and the return of broncho-vesicular breathing indicate that the function of the affected portion of the lung is more or less completely restored. In other cases there are persistent cough, shortness of breath increased on exertion, and hypertrophy of the right side of the heart, perhaps eventually followed by failing compensation, as indicated by the occurrence of cyanosis and œdema. It is not unlikely that in this series of cases are to be included certain of those designated *fibroid phthisis.*

DIAGNOSIS.—Clinically the condition is one of acute pneumonia in which the symptoms persist. The most common cause of such a persistence is the tuberculous nature of the pneumonia, but the absence of bacilli in the sputum negatives this diagnosis. The physical signs frequently continue for a number of weeks in the delayed resolution of fibrinous pneumonia, but the symptoms early disappear and the patient progressively improves.

PROGNOSIS.—Death may occur during the early weeks of this disease, although recovery is the rule. Permanent disability, however, ensues. The patient is liable to frequent or persistent bronchitis, and may suffer from the symptoms of bronchiectasis. The greater the destruction of the lung the more considerable is the hypertrophy of the right side of the heart, which may eventually prove the cause of death from failing compensation.

TREATMENT.—In the early stages of chronic fibrous pneumonia the most earnest efforts should be made to maintain the nutrition of the patient by the careful use of regulated exercise, out-door life, high feeding, and tonics, at the same time every precaution being taken by warm underclothing to prevent suffering from cold. Cod-liver oil and whiskey given together may be of great service. Mild prolonged counter-irritation when judiciously used is of value, probably by relieving rather the accompanying catarrh than the condition of the lung. Croton oil is perhaps the most manageable agent. Creosote, the expectorant volatile oils, and terebene are often temporarily useful, especially when there is much secondary catarrh. Narcotics are sometimes necessary to control cough. (See Acute Bronchitis.) The long-continued use of small doses of arsenic (one drop of Fowler's solution three times a day) distinctly benefits many cases. Potassium iodide (five to ten grains a day) and corrosive sublimate (one-fortieth to one-seventieth of a grain three times a day) may be used continuously between courses of arsenic, to aid in the absorption of fibrinous exudate.

In the later stages of chronic fibrous pneumonia, however, the most satisfactory results are undoubtedly to be obtained by climatic treatment, associated with the institution of such measures as are best fitted to produce bodily vigor. We have seen solidification of the lung, believed to be due to fibrous pneumonia, disappear after six months' residence in a high and dry locality. The management of the case, and the precautions to be taken, are entirely similar to those which have been discussed in detail under the head of Phthisis Pulmonalis. Even if far distant from the medical adviser the patient should during this period use arsenous acid in some form, in such minute dose as to be incapable of irritating the gastro-intestinal mucous membrane whilst at the same time preserving the power of affecting the general nutrition.

BRONCHO-PNEUMONIA.

DEFINITION.—Circumscribed inflammation of the lung, usually multiple, secondary to a capillary bronchitis with which it is associated.

ETIOLOGY.—Broncho-pneumonia is chiefly due to the inhalation of irritating material, especially that containing bacteria. A bronchitis results, which is continued into the capillary bronchi and thence into the alveoli. Numerous bacteria are concerned, foremost among which is the bacillus of tuberculosis, but the bacillus of diphtheria, the influenza bacillus, Friedländer's bacillus of pneumonia, the diplococcus of pneumonia, streptococci, and staphylococci are also of etiological importance. The effect of the bacteria is intensified by the associated inhalation of coarser material, as the secretions from the throat in diphtheria, particles of food in feeding through the tube, the vomitus in exhausted or unconscious persons, blood in case of extensive hemorrhage into the respiratory tract, particles of dust in various trades, and irritating gases. The entrance of such irritating material into the smaller bronchi is promoted by causes interfering with the closure of the glottis, as ulceration, tumors, central or peripheral paralysis, by violent paroxysms of coughing, as in whooping-cough, and by the enfeebled or unconscious state of the patient in anæsthesia, intoxication, or severe disease. The inflammatory action of the bacteria in the lungs is furthermore increased by the persistent local congestion and œdema of frequent occurrence in typhoid fever and other acute and chronic diseases in which weakened circulation and prolonged confinement to bed are constant conditions. Broncho-pneumonia is of greater frequency at the extremes of life, and during the winter months, and not uncommonly follows directly exposure to cold.

MORBID ANATOMY.—The changes characteristic of broncho-pneumonia are disseminated in nodules or lobules, which may be agglomerated or diffused over a considerable portion of a pulmonary lobe. They consist of an exudation of serum, fibrin, leukocytes, occasional red blood-corpuscles, and desquamated epithelium, which fills the bronchioles and the adjacent alveoli. The neighboring interstitial tissue is largely infiltrated with leukocytes. In *nodular broncho-pneumonia* the individual foci vary in size, and may be as large as the tip of the little finger. Their presence is often indicated by a shot-like feel in the unopened lung. On section the affected portions of the lung are slightly elevated, dark red or reddish gray, resistant, and yield on pressure a reddish-gray or puriform viscid fluid, which usually escapes from the section of a central bronchus. Groups of nodules frequently are closely clustered in consequence of the limitation of the broncho-pneumonia to the branches of a particular bronchus. In *lobular broncho-pneumonia* the affected portions of the lung are wedge shaped, and the base of the wedge is often to be seen beneath the pleura as a slightly elevated, dark-red, polygonal patch, the surface of which is covered with a delicate, fibrinous membrane. When the nodules and lobules are numerous and closely agglomerated the intervening lung tissue is often injected, œdematous, and somewhat collapsed. In both nodular and lobular broncho-pneumonia the neighboring portions of the lung are frequently in a condition of collateral emphysema. The broncho-pneumonic nodules resulting from the inhalation of food rapidly become gangrenous, and then are soft, almost diffluent, and of a dark-green color. Abscesses also follow the inhalation of food, either as a primary effect or from the establishment of an inflammatory line of demarcation around a gangrenous centre. In tubercular broncho-pneumonia necrosis of the nodule results, and the familiar characteristics of cheesy degeneration are produced. All varieties of broncho-pneumonia may end in resolution or in permanent obliteration of the alveoli, manifested by a localized induration and pigmentation of the non-aerated lung.

SYMPTOMS.—The symptoms of broncho-pneumonia result from the mechanical obstruction to the air-passages and from the absorption of toxins from the diseased portions of the lung ; the severity of the symptoms is dependent largely upon the number of foci present. Since broncho-pneumonia is usually a secondary condition, following the course of a bronchitis, the symptoms at the outset are generally those of a bronchitis gradually or suddenly increasing in severity and extending into the capillary bronchi. The extension of the capillary bronchitis to the alveoli is indicated by exacerbation of the fever, dyspnœa, and cough. Pain is inconstant, since pleurisy accompanies only when the nodules are superficial. The temperature remains continuously elevated in the vicinity of 103° F., and is without typical curve. The higher and more prolonged the elevation of temperature the more probable are numerous foci of inflammation. The elevation of the pulse and respiration is in proportion to that of the temperature, and, particularly in children, the pulse may reach 150 and the respirations be upward of 60 per minute. The dyspnœa is marked, the accessory muscles of respiration being called prominently into play, and the breathing is short and quick, often irregular from fear of exciting cough and pain and wheezing from the presence of coarse râles in the larger tubes. Cough is frequent, distressing, and in paroxysms. In children the efforts at coughing are likely to cause vomiting, and the bronchial secretion which has been swallowed is to be found in the ejected contents of the stomach. The cough may be so violent that, in connection with the dyspnœa, rupture of the alveolar wall takes place, and interstitial emphysema of the lung follows, at times being extended from the root of the lung into the subcutaneous tissue of the neck and chest. The sputum is usually scanty, viscid, and streaked with blood, and the urine frequently contains a trace of albumin.

On physical examination of the chest the conspicuous signs at the outset are those of a bronchitis extending into the smaller tubes. Resonance on percussion may be somewhat increased, and numerous coarse and fine, dry and moist râles are to be heard. If the broncho-pneumonic nodules are large and superficial, which is especially likely to be the case when there are numerous nodules in the posterior portion of the lower lobes, sharply defined signs of solidification may be found, as dulness, bronchial breathing, and bronchophony.

The course is usually protracted, even in favorable cases extending over a fortnight, the temperature gradually falling to the normal as the dyspnœa and cough diminish. In unfavorable cases the skin becomes dusky, and the expression anxious, until sopor occurs, interrupted perhaps in children by convulsions, and ending, often suddenly, in death. In such cases the fatal termination may occur early, as in fibrinous pneumonia from acute congestion and œdema, or late in the course of the disease from complicating pulmonary gangrene or abscess. Convalescence from broncho-pneumonia, especially in children, is not infrequently protracted over a period of months, during which slight and irregular elevations of temperature are frequent, and at such times the respiration is quickened and moist râles are to be heard. It is in these cases in particular that infection with the bacilli of tuberculosis is to be feared and especially guarded against.

Diagnosis.—Since broncho-pneumonia is the result of a capillary bronchitis, and the pulmonary lesions are often so small or so situated as to give no physical evidence of their presence, the diagnosis of broncho-pneumonia is chiefly based upon the persistence of the symptoms and signs of a capillary bronchitis. Dyspnœa, frequent, short, dry cough, high fever, and abundant, fine moist râles in a resonant lung in the course of acute bronchitis are suggestive of a capillary bronchitis or bronchiolitis. The longer the symptoms and signs continue, the more probable is the presence of foci of broncho-pneumonia, which may form, in the course of two or three days, with a rapidity equal to that of the solidification of the lung in acute fibrinous pneumonia. This disease is to be excluded by the absence of a sudden onset and the well-marked physical signs. Central pneumonia, in which the physical signs of solidification may be obscured, lacks the numerous fine moist râles of the capillary bronchitis associated with broncho-pneumonia, and rusty sputa may be present. Sharply defined broncho-pneumonia, especially when occurring as a nodule of lobular pneumonia, is not infrequently due to the bacillus of tuberculosis, the presence of which is to be suspected if the symptoms and signs have persisted for several weeks. Even if characteristic bacilli are not found in the sputum, a chronic sharply defined patch of broncho-pneumonia is to be regarded with anxiety, since, if not tuberculous at the outset, it readily becomes so.

PROGNOSIS.—The general mortality from broncho-pneumonia is high, perhaps from one-third to one-half of the cases proving fatal. It varies considerably, however, under the conditions of its occurrence. In infants it is often associated with diphtheria, measles, or whooping-cough, and the death-rate is large. In old people also the mortality is high. When broncho-pneumonia is due to the inhalation of foreign bodies of any considerable size, and especially to the entrance of food, death is the usual result. In the broncho-pneumonia following measles in children otherwise healthy, or occurring as an epidemic in them or in adults, the disease runs a favorable course. A sharply defined broncho-pneumonia, even if the course is protracted and the origin tuberculous, not infrequently ends in recovery. In any particular case the prognosis depends upon the extent of the disease, the age and the previous health of the patient, and the exciting cause. The unfavorable symptoms are the persistence of high fever, irregular and superficial respiration, rapid, weak pulse, drowsiness, and delirium.

TREATMENT.—It is especially important to have skilful and watchful nursing, in order to keep the child at absolute rest, and to note and report at once the development of sudden serious symptoms, such as a rise of temperature or the development of dyspnœa or cyanosis. The food should consist chiefly of animal broths, which may often be thickened with nutritive material, or an egg may be stirred into the soup whilst still very hot. The white of egg dissolved in water and sweetened to the taste of the child is useful especially for diluting alcoholic stimulants, which are often required very early. The room should be kept at an even temperature of about 65° F., free from draughts, with a well-moistened air.

The indications which underlie the medical treatment of capillary bronchitis differ from those of ordinary bronchitis chiefly as required to meet the tendency which exists, especially in the advanced stages of the disease, to cardiac failure. Grave danger, therefore, even in the beginning of the disease, attends the use of depressing remedies. It is not justifiable to give to the child suffering from broncho-pneumonia such drugs as veratrum viride or tartar emetic, although in the beginning of the attack the potassium citrate cough mixture with ipecacuanha, or, in the robust child, with apomorphine, should usually be exhibited. If, as is frequently the case, the temperature is high, its reduction is most urgently demanded on account of its weakening influence upon the right heart. It is plain that aconite must be employed, if at all, with the greatest caution, and even phenacetin and its allies are to be looked upon with disfavor, except in small doses, on account of their occasional cardiac effects. The tepid bath, 90° F., usually affords the safest method of keeping down the fever, especially if it be aided by the application of cold compresses to the chest.

In Germany much reliance is placed upon the use of tepid packs. The naked child is wrapped in a sheet which has been wrung out of water having a temperature of about 75° F., and then in a dry woollen blanket. When the heart is weak, very cold baths are dangerous. (See page 142.)

Stimulant expectorants are usually indicated more early in capillary than in ordinary bronchitis. Ammonium chloride is especially valuable, to be preferred, we think, to ammonium carbonate, which is so much used by practitioners, but which in whole or in part undergoes decomposition in the stomach. The ammonium should be given every hour during the day and every two or three hours at night. A little later in the disease oil of eucalyptus and terebene are often of great service; but the employment of any expectorant should always be subordinate to its effects upon the stomach.

What has been said concerning the use of counter-irritation in acute bronchitis applies with equal force to broncho-pneumonia. In very young children moist applications, the jacket-poultice or cold compresses, as may be selected, are much more effective than in adults. It appears probable that the absorption of water may take place through the thin-walled chest, so as to exert a local soothing influence upon the internal organs. In children blisters must be employed with great caution, if at all. When the broncho-pneumonia is part of a general infectious process they are especially dangerous; their excessive local effects may become a serious complication. There are, however, cases in which they may be serviceable if used with due judgment.

It is believed by many practitioners that the free use of water has a pronounced tendency to lessen the viscidity of the inflammatory products in the bronchial tubes: hence the child should be encouraged to drink copiously of watered milk, plain or effervescent waters, or weak lemonade.

In young children capillary bronchitis frequently produces death by the accumulated secretion mechanically interrupting the lung-function. A slight insufficiency of respiration may bring about a slow accumulation of carbonic acid, which more and more depresses respiration. In many cases it is dangerous to allow a child to sleep longer than three hours without being thoroughly awakened, and we have seen cases in which it was necessary to arouse the child every twenty minutes, and even to produce a crying-fit, in order to clear off the excessive carbonic acid. It will be readily seen that opiates are, therefore, to be used for the relief of the pleuritic or other pain only with the greatest reserve, and that whenever there is a tendency to stupor, with lividity of the face and lips, or even a general cyanosis (suffocative catarrh), it is essential not only to support the respiratory centres by hypodermic injections of strychnine and cocaine, but to free the lungs from retained secretion by vomiting. Zinc sulphate or mustard should be selected as the emetic, even ipecacuanha and apomorphine being too depressant to be used with freedom, though they may be employed as adjuvants if necessary. Whenever there is cyanosis and the child is old enough to be docile, frequent inhalations of pure oxygen should be given.

In young children suffering from capillary bronchitis and broncho-pneumonia, after the entire failure of emetics to clear the lungs, when the narcosis was so deep that the power of swallowing was lost and death seemed inevitable, we have saved life by the following procedure. Three tubs having been provided, one empty, one full of water at about 110° F., one full of very cold ice-water, the child's body is to be held over the empty tub and a ladleful of the hot water dashed upon the chest, immediately followed by one of the ice-water, so as to produce a violent respiratory spasm, by which the air is drawn into the lungs. If after the douches have been repeated a number of times change of color of the surface of the body shows that the accumulated carbonic acid has been in part thrown off and that consciousness is returning, the whole body may be immersed in the water, which is so hot that the child screams, and thereby fills its lungs with air. The use of the ladleful of hot water in these cases is to intensify the shock and to prevent cooling of the body.

PULMONARY GANGRENE.

Etiology.—Gangrene of the lung, as of other parts, is due to the action of putrefactive bacteria upon the tissues whose nutrition has been impaired or arrested by an interference with the supply of blood. It is to be found, therefore, in inflammation as fibrinous pneumonia, broncho-pneumonia, especially when due to the inhalation of food, and in connection with cavities, whether of tubercular or of bronchiectatic nature. The disturbance of nutrition may be the result also of injury and of embolism. Putrefactive bacteria may be present in the affected portion of the lung before its nutrition is disturbed, or enter in the inhaled food or through a fistulous communication between the alimentary and respiratory tracts, or be introduced in an embolus from a gangrenous source.

Pulmonary gangrene is of more frequent occurrence in those enfeebled by age, bad habits, or disease than in vigorous persons of good habits in the prime of life.

Morbid Anatomy.—The anatomical appearances are oftenest present in the lower lobes, are usually circumscribed, single or multiple, but in rare instances are diffused over the greater part of a lobe. The multiple nodules and diffused gangrene are frequently due to the dissemination of the gangrenous products from the primary focus along various bronchi by inhalation. At the outset the diseased portion of the lung appears as a more or less sharply defined mass of various size, irregularly rounded, of extremely offensive odor, and of a dark-green color. It is friable, and soon becomes deliquescent. The fluid is easily washed away, leaving a cavity filled with shreds of tissue adherent to the wall. The surrounding lung-tissue is hepatized, and the necrotic and putrefactive changes are often continued in it until the cavity progressively increasing in size becomes as large as the fist. The pleura, if reached, is perforated, and an ichorous pneumothorax results. Abscess of the brain, presumably of embolic origin, is an occasional complication.

SYMPTOMS.—Fever, cough, fetid sputa, and foul breath are the prominent disturbances in pulmonary gangrene, and are of rapid or gradual onset according to its especial causation. The fever is constant, the range of temperature shows irregular variations, and the pulse is quick and feeble. There is no appetite, vomiting and diarrhœa are frequent, and there is rapid loss of flesh and strength. Cough occurs at irregular intervals, is frequently paroxysmal, and results in the raising of abundant liquid sputum resembling that of putrid bronchitis with bronchiectasis. After standing the upper portion is frothy, the middle layer thin and discolored, and the lower portion, of a dirty-brown color, contains fat-crystals, bacteria, and shreds of lung-tissue in which, during the advance of the gangrene, elastic fibres are to be found. Blood is present frequently in the sputum, usually in small quantity, and produces a green or brown coloration of the fluid and forms granular pigment in the sediment. Profuse and even fatal hemorrhage may occur from the sudden rupture of a large blood-vessel in the gangrenous tissue. The physical signs at the outset are those of consolidation, but soon yield to those of a cavity, and are often overlooked if the gangrenous disturbances are of small area or deep-seated in the lung.

Exacerbation of the temperature and the simultaneous development of a sharp stitch-like pain in the side indicate a complicating pleurisy which is likely to prove purulent. Sudden dyspnœa accompanied by a tearing sensation is evidence of a rupture of the pleura overlying the gangrenous cavity and of the passage of air into the thorax, in which case pneumothorax and ichorous pleurisy are soon followed by death. Frequently the progress of the gangrene is arrested by the formation of a granulation-tissue in the inflamed lung surrounding the affected portion, and then the disease assumes a chronic course, the symptoms being those of a putrid bronchitis, and eventually, perhaps, of a bronchiectasis. If the destruction of lung is of small area, these symptoms may cease in the course of months and the cavity be obliterated by the formation of a scar.

DIAGNOSIS.—The presence of pulmonary gangrene is to be inferred during the acute stage by the rapid production of the abundant offensive sputa containing elastic fibres in connection with the etiological factors above mentioned. In the chronic stage it is easily confounded with putrid bronchitis, especially as shreds of lung-tissue may no longer be found in the sputum. Putrid bronchitis, however, occurs in the course of long-continued chronic bronchitis, while gangrene of the lung is commonly of acute onset.

PROGNOSIS.—Although recovery from gangrene of the lung is possible when a small portion only of the lung is involved, the prognosis in general is unfavorable. Death is caused usually by acute or chronic septicæmia, and in rare cases is attributable to cerebral abscess. In other instances the course is that of pulmonary abscess.

TREATMENT.—It is not possible in any way by medicinal treatment to affect gangrenous lung-tissue. Creosote may be used internally as freely as it can be taken, and inhalations of carbonic acid or other disinfectant vapors or sprays steadily practised; but there is no reason for believing that these measures have any curative effect. The general treatment should, of course, be stimulating and supporting to the fullest extent. When a limited area of the lung is affected, and is so situated as to be reached surgically, pneumotomy may be performed, as successes have been achieved.

PULMONARY ABSCESS.

DEFINITION.—A circumscribed cavity in the lung with a wall of inflamed pulmonary tissue and with purulent contents.

ETIOLOGY.—The presence of pyogenic bacteria is the immediate cause of pulmonary abscess, and the conditions favoring their growth are promoted by the exciting causes,—namely, trauma, inflammation, and embolism. The bacteria may be dormant in the lung previous to the approach of the exciting cause, or may be admitted with it, for example, in a septic embolus, or in inhaled food, or be introduced from a suppurating process in the vicinity, especially in the pleural or the peritoneal cavity, or from abscess of the liver, mediastinum, or peribronchial lymph-glands.

MORBID ANATOMY.—The abscesses are solitary or multiple, the latter being due especially to embolism or food-inhalation. The solitary variety is found more frequently in the upper lobes, but multiple abscesses are seated oftener in the posterior portion of the lower lobes. Multiple abscesses are usually small, not larger than the finger-tip, rounded, deep-seated, or superficial. In the early stage they appear as sharply localized purulent infiltrations of the lung-tissue, and the pus may be squeezed from the cut surface as from a sponge. Later the cavity is sharply defined, its wall of an opaque, yellowish-gray granulation-tissue separated from the aerated lung-tissue by a hepatized zone. The largest, usually solitary, abscesses may be of the size of the fist, and have a relatively smooth wall surrounded by indurated lung-tissue. When the pleura forms a part of the wall of the abscess a fibrinous or suppurative pleurisy and perhaps pneumothorax are associated.

SYMPTOMS.—In the clinical consideration of pulmonary abscess an important distinction to be drawn is that between *acute* and *chronic abscess*. Acute abscesses when multiple are small, and are formed usually without any characteristic symptoms, since they occur as complications of other morbid processes. The presence of a large acute abscess may be made manifest towards the close of hepatization in acute pneumonia by the sudden evacuation of a considerable quantity of pus in which blood-crystals and elastic fibres are to be seen.

Chronic pulmonary abscess is indicated by persistent cough, by abundant expectoration, and by the physical signs of a cavity. The sputum may contain shreds of lung-tissue, also crystals of cholesterin.

The acute abscess is to be distinguished from gangrene by the greater quantity of pus in the sputum and the less offensive odor. In chronic abscess the freedom from characteristic bacilli serves to eliminate a tubercular cavity, and the absence of antecedent chronic bronchitis and the presence of elastic fibres in the sputum exclude bronchiectasis.

The prognosis of abscess of the lung is serious. A large acute abscess may cause death from suffocation if the pus suddenly enters a large bronchus and is inhaled into its branches. Rupture of the abscess into the pleural cavity usually produces a fatal pyopneumothorax. Pulmonary abscess may heal, however, and the sac become obliterated either spontaneously or after evacuation through the chest-wall.

TREATMENT.—There is no known medical method of successfully influencing pulmonary abscesses. The general treatment must be supporting and palliative. If the abscess be superficial, pneumotomy should be performed, with thorough after-drainage.

TUMORS OF THE LUNG.

Tumors of the lung occasionally occur, but usually are not sufficiently large or numerous to give rise to symptoms. Primary and secondary growths are to be found. The former include fibroma, which is generally small, often multiple, and then in origin on the border line between inflammation and new formation, and lipoma, chondroma, and osteoma, which are rarities. These tumors are benignant, although they may produce disturbance of respiration by pressure upon the lung or the larger air-passages. The malignant tumors are malignant lymphoma, sarcoma, and cancer. From their essentially similar clinical characteristics they are conveniently grouped under Cancer of the Lung.

CANCER OF THE LUNG.

ETIOLOGY.—Malignant tumors of the lung, though often found as secondary growths, sometimes originate in this organ, and cancer is present as a primary tumor more often than the other varieties. Sarcoma and malignant lymphoma are usually secondary either to disease, especially of the lymph-glands, in the vicinity, or to a primary growth in remote parts of the body. Secondary cancer of the lung is more common in women than in men, perhaps from the frequency of extension to the lung through the intervening pleura of cancer of the mammary gland, so common in women. The frequent occurrence of malignant lymphoma in the cobalt-mines of the Tyrol, as described by Wagner, is used in support of a local, perhaps infectious, cause of malignant neoplasms.

Morbid Anatomy.—The malignant disease of the lung is to be found as nodules or as a diffuse infiltration; when primary it is limited to a single lobe or to one lung, but when secondary is usually bilateral. The nodules are solitary or multiple, and vary in size from a pin's head to a mass as large as the fist. Large solitary nodules are likely to be sharply defined, whereas large multiple nodules are often gradually continued into the lung-tissue. The nodules are irregularly distributed throughout the lungs, and are usually more abundant in the lower lobe. Infiltrating cancer follows the course of the bronchi, and may so grow within the smaller bronchi and alveoli as completely to close them, although on microscopical examination the elastic fibres in the alveolar wall remain in normal grouping. Cancer of the lung is usually soft, medullary, and of a grayish color. An opaque juice is to be squeezed from the cut section, and is composed largely of epithelioid cells often fattily degenerated. The growth sometimes projects into the larger bronchi as nodules, rounded or flattened, and occasionally with a villous surface. When the tumors grow from the pleura, they are not infrequently associated with hydrothorax or pleurisy, the latter at times of hemorrhagic character. The lymph-glands at the root of the lung, in the neck, and even at remote parts of the body are often simultaneously diseased.

Symptoms.—When the tumors of the lung are of sufficient size or so situated as to produce symptoms, dyspnœa is usually the first and most frequent disturbance. Perhaps at the outset it is manifested only on exertion, but it is at times paroxysmal, and occasionally stridulous, especially when the root of the lung is involved. Cough is frequent, often dry, but sometimes accompanied by a viscid sputum compared from its color to the juice of black currants. There may be no pain, or stitch-like pains are present when there is inflammation of the pleural surface, which is of occasional occurrence only, having been present, according to Bennett, in six out of thirty-nine cases. Fever is usually absent unless there is a complicating broncho-pneumonia. Digestive disturbances are infrequent, and loss of flesh and strength is often inconspicuous. On physical examination the face is either pale or livid: in the latter case pressure upon the veins at the base of the neck from the extension of the disease to the mediastinal lymph-glands is probable, and œdema as well as cyanosis of the face and of the upper half of the body is likely to be present. Persistent dulness on percussion, bronchial breathing, crepitant or subcrepitant râles, and bronchophony are to be observed. The abnormal respiratory and voice sounds not infrequently disappear in consequence of the accumulation of fluid in the pleural cavity or from the filling of the bronchi and alveoli with the cancerous growth.

Diagnosis.—The existence of cancer of the lung is to be inferred from the persistent dyspnœa and the results of the physical examination of the chest, including the aspiration of a bloody fluid from the pleural cavity. The diagnosis is aided by the discovery of enlarged lymphatic glands at the base of the neck and by evidence of venous pressure in this region, and is strengthened if within a year or two a malignant tumor has existed elsewhere in the body. Pulmonary tuberculosis is to be eliminated by the absence of bacilli in the sputum, and broncho-pneumonia by the persistence of the symptoms in the absence of fever. Simple pleurisy is to be excluded by the rapid return of a serous or bloody fluid and by the failure of aspiration to give relief.

PROGNOSIS.—Cancer of the lung proves at times rapidly fatal, even within a few weeks after the symptoms have been complained of. Death occurs within a year from the onset of the disturbances.

TREATMENT.—Relief from symptoms is the only result to be expected from treatment. Frequent examinations of the chest are to be made, since distress due to the presence of fluid is often relieved by paracentesis, which is to be repeated as often as may be necessary.

CHAPTER III.

DISEASES OF THE PLEURA AND OF THE MEDIASTINUM.

DISEASES OF THE PLEURA.

PNEUMOTHORAX.

Definition.—The presence of air in the pleural cavity.

Etiology.—Air enters the pleural cavity in consequence of perforation of the parietal or of the visceral pleura. The parietal pleura is perforated by penetrating wounds of the thoracic wall from either accident or malice, and sometimes for therapeutic purposes, as in thoracentesis or thoracotomy. The spontaneous evacuation of pus through the thoracic wall in empyema is followed by pneumothorax. The air may enter when the diaphragm is perforated by subphrenic abscesses communicating with the alimentary canal and in the progress of ulcer or cancer of the stomach. Air may be admitted also through the parietal pleura in rupture, perforating ulcer, or cancer of the œsophagus. The pulmonary pleura may be perforated from the free surface in injury from a stab or from a broken rib, or in consequence of the evacuation into a bronchus of the pus in empyema. More frequently the perforation is due to destruction of the pleura from the pulmonary side, particularly when there is a cavity due to pulmonary tuberculosis, gangrene, or abscess. Rupture of the pleura from within may occur also from sudden and violent muscular effort, especially when the lung is emphysematous. In more than nine-tenths of all cases, according to Fraentzel, pneumothorax is due to pulmonary tuberculosis, and, according to Weil, it occurs in nearly ten per cent. of cases of phthisis.

Morbid Anatomy.—The immediate result of the admission of air into the pleural cavity is the retraction of the lung, in virtue of the shrinkage of its elastic tissue. The degree of contraction is dependent upon the presence or absence of solid material within the lung, or of adhesions between the pleural surfaces. If these are present, a circumscribed pneumothorax follows, but if there is neither solidification of the lung nor pleural adhesions the pneumothorax is diffused and the entire lung is collapsed and is withdrawn to the upper and posterior portion of the chest. The heart and mediastinal tissues are displaced towards the unaffected half of the thorax, the diaphragm is depressed, and the liver also, in case of pneumothorax of the right chest. The hole in the pleura may be readily recognized, or may be seen with difficulty, especially in emphysema and in pulmonary tuberculosis. Inflation of the lung placed under water often reveals the situation of the opening by the escape of bubbles of air. Weil discriminated between open pneumothorax, closed pneumothorax, and valvular pneumothorax. In open pneumothorax the air freely enters and leaves the pleural cavity. In closed pneumothorax the opening in the pleura becomes closed, and no more air enters. In valvular pneumothorax, which is the variety oftenest present, air is permitted to enter, but is prevented from escaping, and in consequence extreme degrees of deformity of the thorax and dislocation of its contents are occasioned.

The anatomical changes in pneumothorax depend also upon the simultaneous or subsequent admission of other material than air. Such material is usually infectious and causes pleurisy, which is serous, suppurative, or ichorous, and *hydropneumothorax* or *pyopneumothorax* results. In such cases, in addition to the collapse of the lung and the displacement of the organs, the pleura presents the characteristics of an acute or a chronic pleurisy, and the pleural cavity contains, in addition to air, a greater or less quantity of sero-fibrinous, purulent, perhaps hemorrhagic, exudation, which may have a putrid odor.

SYMPTOMS.—In diffused pneumothorax there are sudden pain, often of a tearing character, dyspnœa, which may be extreme and accompanied by cyanosis, anxiety, a feeble pulse, and even a condition of collapse. In circumscribed pneumothorax the adhesions may be so abundant, or the solidification of the lung so considerable, that few or no symptoms arise, and not infrequently the pleural cavity is found unexpectedly to contain air at the post-mortem examination of a case of extensive pulmonary tuberculosis. When pneumothorax is caused by the discharge through a bronchus of pus from a pleural cavity, there is, in addition to the above-mentioned symptoms, a sudden paroxysm of cough, associated with a greater or less quantity of purulent expectoration.

On physical examination, the more extreme the degree of pneumothorax the more distended and the less movable is the affected half of the chest, the wider are its intercostal spaces, and the greater is the displacement of the heart and, in case of right-sided pneumothorax, of the liver, the lower edge of which may be found near the navel. There may be no dislocation of these organs, and in open pneumothorax the walls of the chest move on inspiration. On palpation vocal fremitus is absent. On percussion the resonance has a metallic character, and the pitch is higher when the mouth is open. It is usually tympanitic, perhaps amphoric, though at times it is dull, presumably in consequence of the extreme tension of the contained air. On auscultation the vocal resonance has a faint metallic sound, but may be absent, especially over the lower portion of the chest. The respiratory murmur often has also a metallic character. It is best heard at the upper part of the chest and in the back, and is faint and bronchial when there is marked compression of the lung. In open pneumothorax the breathing may be distinct and amphoric. An important characteristic of pneumothorax is the peculiarly modified metallic sound to be heard at the back of the affected half of the chest when the edge of a coin is sharply struck against the flat surface of another in close apposition to the chest-wall in front. When fluid also is present it moves about with great freedom, and the resulting dull area on percussion quickly changes with the change of position. A splashing sound is to be heard often at a distance from the patient when he shakes his body, or when it is shaken. A metallic tinkling is frequently to be heard, especially when a long breath is drawn, and is attributed to the falling of material into the pleuritic exudation.

Diagnosis.—Diffused pneumothorax is easily recognized on physical examination of the patient. The accession of fluid is made evident by splashing on succussion and by the ready change in the outline of dulness on change of position. Circumscribed pneumothorax is at times with difficulty differentiated from a large pulmonary cavity, but in pneumothorax the vocal fremitus and the respiratory murmur are diminished or absent, while they are present and perhaps exaggerated in the examination of a cavity. Circumscribed pyopneumothorax is distinguished also with difficulty from subphrenic pyopneumothorax. In the latter the previous history is of disease of the abdominal organs and not of the lungs. There is but little distention of the chest. Vocal fremitus and vocal resonance are distinct, perhaps loud, in the upper part of the chest, and are to be recognized somewhat lower on deep inspiration.

Prognosis.—In unilateral pneumothorax from a wound or in consequence of extreme muscular strain, and when independent of acute or chronic inflammation of the lung, the prognosis is favorable, since the air is usually rapidly absorbed. In pneumothorax from gangrene or abscess of the lung the resulting pleurisy is of a septic character, and the prognosis is doubtful until the results of treatment are apparent. In pneumothorax in pulmonary tuberculosis the ultimate prognosis, as a rule, is unfavorable, in consequence of the frequent extensive disease of the lung. If the pulmonary process is sharply defined, the patient may recover both from the pneumothorax and from the disease of the lung. In the open pneumothorax following the surgical treatment of empyema the patient may live for years in active employment, though eventually likely to die from amyloid disease in consequence of the long-continued suppuration.

Treatment.—The occurrence of air in the pleural cavity does not greatly modify the treatment of a coexisting effusion. If the latter be purulent, free incision and permanent drainage should be practised, as in simple empyema. If the exudation be serous, it may be let alone or may be withdrawn by aspiration, according to the amount which is present. Not rarely, when pneumothorax arises in phthisis, the symptoms are so slight that no immediate local treatment is required. Increase of the fluid under such circumstances is to be met by aspiration. When there are such immediate distress and dyspnœa as to make it probable that there is high intra-thoracic pressure, a thin needle may be introduced into the portion of the pleura which is full of air, and the air be allowed to escape or even aspirated. If this fail to remove the distress, hypodermic injections of morphine become necessary.

HYDROTHORAX.

DEFINITION.—The accumulation in the pleural cavity of a transuded fluid of non-inflammatory origin.

ETIOLOGY.—The immediate causes of hydrothorax are obstruction to the outflow of subpleural venous blood and lymph and such disturbance of the nutrition of the walls of the blood-vessels and the lymphatics as occasions an increased porosity. According as the action of these causes is general or local, the hydrothorax is part of a general dropsy or it exists alone. When part of a general dropsy, the mechanical obstruction is the result of interference with the passage of blood through the heart and lungs. The hydrothorax occurring in nephritis and in the later stages of fibrous hepatitis is partly of mechanical origin and partly cachectic. It is probable, also, that the cachectic hydrothorax present in diseases with extreme disturbance of nutrition, as general amyloid degeneration and cancer, is in part of mechanical and in part of cachectic origin. Hydrothorax independent of general dropsy is the result of local pressure upon the large veins as they enter the thorax and upon the thoracic duct. Such pressure is occasioned by intra-thoracic tumors, especially of the mediastinum, and more rarely of the lungs.

MORBID ANATOMY.—The appearances vary according as fibrous adhesions between the pleuræ are present or absent and according to the quantity and quality of fluid. Hydrothorax is usually bilateral, but not infrequently one pleural cavity, especially the right, contains more fluid than the other. If the adhesions are distensible they become œdematous, and the lung is separated from the thoracic wall by a gelatinous mass, perhaps two inches in thickness, from which abundant serous fluid is to be squeezed as from a sponge. If dense adhesions are present, obliterating a part of the pleural cavity, encapsulated hydrothorax results. When an entire pleural cavity is obliterated, the hydrothorax is limited to the other half of the chest, although the cause may be general. Unilateral hydrothorax, when both pleural cavities are free from disease, usually depends upon the limitation of the immediate cause to the affected half of the chest.

The larger the quantity of fluid, which is sometimes several quarts, the greater the degree of retraction of the lung and the more considerable the atelectasis. The pleural surfaces are generally unaltered, at the most slightly opaque and of diminished lustre. The fluid is usually of a watery consistency, the specific gravity being below 1015, and the quantity of albumin less than three per cent. (See Ascites, p. 968.) It contains occasional leukocytes, red blood-corpuscles, and desquamated endothelium, which may be fattily degenerated, and does not coagulate spontaneously unless the hydrothorax is of long duration or complicated with pleurisy. The fluid sometimes contains chyle, *chylothorax*, presumably from obstruction of the thoracic duct or from rupture of one of its intra-thoracic branches. The fluid then resembles milk in color, though it is sometimes pink from the presence of blood. In the course of several hours after its removal a creamy layer forms on the surface.

SYMPTOMS.—Hydrothorax produces but little disturbance unless the quantity of fluid is large. The circulation and respiration are then disturbed, and a sense of substernal constriction, a quick, weak pulse, cyanosis, and dyspnœa result. These symptoms may arise when a small quantity of fluid only is present, especially if there is cardiac incompetency.

DIAGNOSIS.—The presence of free fluid in the pleural cavity is readily recognized from the localized dulness and absence of respiratory sounds, the physical signs quickly changing with the alteration of the position of the patient. The dropsical nature of the fluid is indicated by the evidence of general dropsy and the absence of fever and of pleuritic pain, and is confirmed by the characteristics of the aspirated fluid. In encapsulated or in unilateral hydrothorax, especially when of long standing, the diagnosis may be difficult, since chronic pleurisy with liquid exudation often pursues a latent course and the characteristics of the fluid may not differ from those of the fluid of chronic hydrothorax.

PROGNOSIS.—Since hydrothorax is a symptom merely, the prognosis depends upon that of the disease which occasions it. It is, therefore, often favorable when due to remediable disturbances of nutrition, as in various anæmic conditions and in acute nephritis. When due to obstructive disease in the heart or lung or to intra-thoracic tumors, the prognosis is unfavorable, since the hydrothorax then usually represents the terminal stage of these affections. According to Bargebuhr, who has collected twenty-two cases of chylothorax, its prognosis is unfavorable, seventeen of the patients having died.

TREATMENT.—Immediate aspiration should be performed in an extensive hydrothorax. If there be a tendency to reaccumulation of fluid, the patient should be freely purged with salines or elaterium, and hydragogue diuretics or diaphoretics should be exhibited. The disease or condition which produces the hydrothorax must be carefully treated.

In chylothorax repeated tapping is undesirable, in consequence of the removal of a highly nutritious fluid, unless the symptoms from pressure are marked.

HÆMOTHORAX.

DEFINITION.—The presence of blood in the pleural cavity.

Bleeding takes place into the pleural cavity in consequence of laceration of the intra-thoracic blood-vessels from wounds of the thoracic wall or of the lung, or from rupture of an aneurism of the aorta or of an intercostal artery. Hæmothorax occurs also when the pleura is ruptured in case of hemorrhage into a gangrenous or tubercular cavity.

The resulting symptoms are those of a sudden anæmia, and vary according to the quantity of blood which escapes. The initial pain, of obvious or concealed origin, is immediately followed by vertigo and weakness and by rapid, perhaps difficult, breathing. The skin is pale, the pulse is quick and soft, and in severe hemorrhage the face becomes pinched, the skin cool and moist, and the respiration long-drawn. The blood, when limited in quantity, may be quickly absorbed. If considerable in amount, and especially if clotted, absorption may be prolonged over a period of weeks, the clots often becoming encapsulated at the bottom of the pleural cavity. When infection of the pleura is associated with hæmothorax, as in wounds or in ruptured pulmonary cavities, pleurisy, usually suppurative or ichorous, results. The signs from auscultation and percussion are the same as in hydrothorax, and the diagnosis is based upon the symptoms of a rapidly progressing anæmia, the physical examination of the chest, and the aspiration from it of blood.

The prognosis depends upon the cause, and may be favorable in case of wounds, but is necessarily fatal in ruptured aortic aneurism. In fatal cases death may take place quickly in consequence of the extent of the hemorrhage, and symptoms of collapse are then likely to be followed by sopor or convulsions. When severe pleurisy accompanies the hemorrhage the prognosis and treatment are essentially those of the pleurisy, and vary according to its etiology and nature.

PLEURITIS. PLEURISY.

Etiology.—Inflammation of the pleura is of great frequency, evidences of the occurrence of this disease at some time in life being found in the large majority of post-mortem examinations. It occurs at all ages, in men more often than in women, and particularly during the winter and early spring. A distinction is usually made between primary and secondary pleurisy. The former is rare, and is attributed to exposure to cold or to injury. Most cases of pleurisy, however, are secondary, —that is, occur in the course of various diseases with which they are more or less intimately connected and of which they represent a complication, although the disturbances from the pleurisy may surpass those of the original disease. These diseases are usually of structures covered by the pleura, especially the lungs: hence pleurisy is frequent in pulmonary tuberculosis, pneumonia, broncho-pneumonia, pulmonary embolism, gangrene, and abscess. In consequence of the propinquity of the pleura it is inflamed frequently in the course of pericarditis, in tuberculous affections of the spine, ribs, sternum, and the subpleural lymphatic glands, and in cancer of the œsophagus. It occurs often when the peritoneum covering the diaphragm is inflamed, in general peritonitis, and in the course of abscess of the liver, of appendicitis, and of ulcer and cancer of the stomach. Pleurisy is one of the complications of acute infectious diseases, especially those in which the respiratory tract is sooner or later affected, as measles, diphtheria, influenza, typhoid fever, and septicopyæmia. It is of occasional occurrence in acute articular rheumatism, also in nephritis, gout, and syphilis. The diseases in which pleurisy is oftenest found are pulmonary tuberculosis, pneumonia, and bronchopneumonia.

It is the prevalent opinion that micro-organisms, either alone or in combination, their products, and, perhaps, chemical irritants of other origin are the immediate causes of the inflammation of the pleura, and are conveyed to this membrane by means of the blood-vessels or lymphatics. The ease of their direct passage from parts covered by the pleura is readily understood, and their transfer by means of the circulation from remote parts, as in erysipelas, phlegmonous inflammation, suppurative osteomyelitis, or gonorrhœa, is feasible. Various bacteria have been found in the pleuritic exudation, especially when it is purulent. These are the diplococcus of pneumonia, the streptococcus pyogenes, the staphylococcus pyogenes, and the bacillus of tuberculosis. The diplococcus of pneumonia is present particularly in the pleurisy associated with pneumonia, *metapneumonic pleurisy*, and is the variety usually found in the empyema of children,—according to Levy, in two-thirds of the cases. The streptococcus is found more often in the empyema of adults than in that of children. Other bacteria are more rarely seen, as Friedländer's bacillus, the typhoid bacillus, the colon bacillus, the gonococcus, the proteus vulgaris, and various saprophytic bacteria. The assertion often made that tuberculosis is the chief cause of pleurisy is based rather upon clinical and anatomical than upon bacteriological evidence. Pleurisy often occurs in persons suffering from tuberculosis or in whom tuberculosis subsequently develops, and evidences of pleurisy are usually associated with those of pulmonary tuberculosis. The bacilli of tuberculosis, however, are rarely found in the exudation, and then especially when the fluid is purulent. The assumption that nearly three-fourths of all cases of serous or fibrino-serous pleurisy are due to tuberculosis is best justified by the observations of Eichhorst, who found that guinea-pigs became tuberculous when inoculated with the serum from fifteen out of twenty-three cases, sixty-five and two-tenths per cent., of acute pleurisy occurring suddenly without obvious cause during health. It is furthermore supported by the estimate that from one-third to one-half of the patients with simple pleurisy sooner or later become tuberculous. The sero-fibrinous exudation of pleurisy, as a rule, contains no bacteria, although in it, according to the observations of Netter, Prudden, and others, the diplococcus of pneumonia, the streptococcus, and the staphylococcus may be found. The purulent exudation usually contains bacteria, the diplococcus of pneumonia being present generally as the only microorganism,—according to Netter, in nearly one-half of the cases,—and pneumonia, as a rule, is associated. The diplococcus of pneumonia may be present, however, in empyema when there is no lobar pneumonia. The streptococcus is the variety oftenest found in suppurative pleurisy independent of pneumonia, and the staphylococcus is only occasionally found. Although the typhoid bacillus has been isolated in pleurisy occurring in typhoid fever, usually other bacteria are present, especially, according to Weintraud, the staphylococcus pyogenes. While bacteria may be the immediate cause of pleurisy, it is presumable that exposure to cold and injury, and the various diseases in which pleurisy is a complication, promote the growth of the bacteria by affording suitable conditions for their development.

MORBID ANATOMY.—The inflamed pleura presents various appearances, according to the stage and severity of the inflammation. They are circumscribed or diffused, and when limited to the vicinity of the diaphragm characterize *diaphragmatic pleurisy.* At the outset the pleura is injected and without lustre, but soon it becomes thickened, opaque, and covered with a thin layer of fibrin, which causes a roughening of the surface. At this stage of the disease the alterations are designated *dry* pleurisy or *fibrinous* pleurisy.

In the further progress of the inflammation the swollen pleura becomes opaque from cellular infiltration, minute hemorrhages are frequent, and the subpleural fibrous tissue also is swollen from cellular and serous infiltration. In the pleural cavity a more or less abundant exudation of serum and fibrin is accumulated. This exudation varies in different cases in the relative proportion of serum and fibrin, and accordingly the pleurisy is designated *serous*, *sero-fibrinous*, or *fibrino-serous.* The fibrin may appear in the thin, pale yellow fluid as flocculi, or form masses soaked with serum, or be present as adhesions between the opposite pleural surfaces. The quantity of serum may be as high as four quarts. It often coagulates after exposure to the air, and contains leukocytes and occasional endothelial cells. These fibrinous adhesions not infrequently enclose spaces in which the serum is retained, *encysted pleurisy*, and when the enclosed space is between the lobules the condition is known as *interlobular pleurisy.* In other cases the exudation is purulent, *empyema.* This variety is rare at the outset, except in the metapneumonic pleurisy of children, but frequently follows an earlier serous or sero-fibrinous exudation. The pus is thin or thick, yellow or greenish yellow, usually odorless, and the quantity of fibrin is generally moderate. In *ichorous pleurisy* the pus is thin, of a grayish color, and exceedingly offensive in consequence of the admission of putrefactive organisms from gangrene of the lung or from a fistulous communication with the alimentary canal, or, more rarely, from the use in treatment of unclean instruments. The liquid exudation at times contains more or less blood, the condition then being known as *hemorrhagic pleurisy.* This variety is associated usually with tubercles or cancer of the pleura, with nephritis, with fibrous hepatitis, and with the hemorrhagic diatheses. The exudation also may contain blood if the vessels are lacerated when the chest is tapped, and, in rare instances, independently of the above-mentioned conditions.

In pleurisy with abundant liquid exudation the lung is retracted, and in extreme cases is compressed into a flat mass along the spine at the upper and posterior portion of the chest. The compressed lung is dense, non-crepitant, and of a pale gray color, from diminution in the quantity of blood. The heart and the mediastinal tissues also are displaced towards the side of the chest which is free from disease, and the affected half of the diaphragm, with the subjacent spleen and stomach or liver, is pushed downward.

If the exudation is absorbed, the pleura subsequently may appear normal or may present the characteristics of a *chronic pleurisy.* These are manifested by fibrous plates of various thickness, frequently associated with deformity of the lung, and there may be few or many fibrous adhesions of greater or less density, sometimes obliterating the pleural cavity and making it necessary in the removal of the lung to tear away the costal pleura. Thickening of the pleura and the formation of fibrous adhesions always result when the process of absorption extends over a long time. The delayed absorption is more likely to occur when there is abundant fibrin or pus, and the inflammatory product then frequently becomes inspissated, encapsulated, and calcified. The earthy salts often are deposited also in the adhesions and in the thickened pleura. In consequence of the shrinkage of this new-formed fibrous tissue the lung is prevented from expanding, the wall of the affected half of the chest collapses, the intercostal spaces are obliterated, the ribs overlap, and lateral curvature of the spine takes place. The other half of the chest becomes dilated from compensatory emphysema of the contained lung. At times a growth of fibrous tissue extends from the pleura into the lung, producing a chronic interstitial pneumonia, usually associated with bronchiectasis and emphysema, the effects of which are described in the article on fibrous pneumonia.

In suppurative pleurisy or empyema, when unrelieved by appropriate treatment, destruction of the pleura is likely to occur, and the pus is discharged either through the lung or through the thoracic wall. Perforation may take place into a large bronchus, when the pus escapes through the mouth, or into numerous alveoli, in which case the lung becomes infiltrated with pus. When the costal pleura is destroyed the intercostal muscles are perforated, and the pus appears beneath the skin, usually near the lower part of the sternum, as an abscess, which often discharges spontaneously, sometimes in the vicinity of the navel, *empyema necessitatis.* Perforation of the pericardium, of the diaphragm, and of the œsophagus also may occur. When the diaphragm is perforated the pus may enter the peritoneal cavity or the stomach, or extend along the retroperitoneal tissues and point in the groin or perineum, and may be discharged even into the bladder. When the fistulæ have existed for a long time, the deformity of the lung and thorax previously mentioned is constant, and more or less extensive amyloid disease of the abdominal organs is likely to be present.

SYMPTOMS.—The method of onset of pleurisy varies extremely. In certain cases there is an initial chill, followed by a sharp knife-like pain in the chest, aggravated on inspiration, and compelling the patient to take a short breath. In other cases there is a gradually increasing sense of weakness, with diminution of appetite, and shortness of breath on exertion is the first symptom that calls attention to the existence of the disease. In such cases of *latent pleurisy* the chest may contain a large quantity of fluid and the patient be occupied with his customary pursuits. As a rule, however, thoracic pain, stabbing or stitch-like and referred to the region of the nipple or axilla, is present in the early stage of pleurisy. In diaphragmatic pleurisy the pain follows the line of the costal insertions of the diaphragm, and sometimes extends into the shoulder and neck; and in this variety, according to De Mussy, the pain is most severe one or two finger-breadths from the median line and on a level with the tenth rib. There is a frequent dry cough, usually suppressed through fear of increasing the pain, and hiccough is frequent in diaphragmatic pleurisy, although the presence of this symptom in pleurisy is not to be regarded as indicating an especial localization of the inflammation in the pleural covering of the diaphragm. The temperature is elevated two or three degrees, and the frequency of the respiration and pulse is increased. In dry pleurisy there may be little or no fever; in serous pleurisy it is moderate in degree, but in suppurative pleurisy the temperature is frequently as high as 103° or 104° F., and wide variations between the morning and the evening temperature occur. Intercurrent chills are also present. Sweating is frequent and often profuse in suppurative pleurisy. In the course of a week or two as the exudation increases in quantity the stitch-like pain disappears in consequence of the separation of the inflamed pleural surfaces, and is replaced by a sense of distention and weight at the lower part of the thorax, but the dyspnœa becomes more considerable. The greater the quantity of the liquid exudation the more likely is cyanosis to be present. There is also a sense of substernal constriction, especially when the heart is displaced, and there is pain in the lower part of the chest from the pressure of the exudation.

The patient, if confined to the bed, prefers to sit upright, or with the back supported; if in the lateral position, he usually lies upon the diseased side. In diaphragmatic pleurisy the body is bent forward, the hands are frequently applied to the sides to restrain the movements of the chest, and the expression is one of great suffering.

Physical Examination.—On inspection, when there is considerable serous exudation, distention of the thorax is perceptible. The intercostal spaces are prominent, and absent or diminished motion of the distended chest is apparent. The apex-beat of the heart may be outside of the left nipple in extensive pleurisy of the right chest, and may be invisible or be found at the right of the sternum in pleurisy of the left half of the chest. On palpation there is recognized by the hand a rubbing sensation in dry pleurisy and impaired mobility of the chest in the sero-fibrinous variety. Especially important as indicative of liquid exudation is the absence of vocal fremitus. In case of displacement of the spleen or the liver, these organs are to be felt through the abdominal wall. Percussion gives evidence of the presence of the exudation when more than six ounces are accumulated, since extreme dulness or flatness and a sense of resistance to the percussing finger are caused. The limits of the upper border of flatness vary in accordance with the quantity of fluid present. In left-sided pleurisy the dulness is to be recognized first in Traube's semilunar space, where the stomach is overlain by the complementary space of the pleural cavity. When the quantity of fluid is moderate, not extending above the inferior angle of the scapula, the upper border of dulness is usually transverse. With considerable degrees of exudation the line of dulness forms a curve the highest point of which is in the axilla. Damoiseau asserted that the upper border of dulness formed a parabola with the highest point in the axillary line. Ellis also found the highest point of dulness in the axillary line, from which the upper border of dulness inclined slightly downward towards the sternum. He discovered that the curved line of dulness between the spine and the axilla was shaped like the letter S. This observation was corroborated by Garland by means of experiments on animals. When the chest is filled with fluid the letter S outline disappears and the curve rapidly rises to its highest point at the top of the shoulder, resonance being found only at the upper part of the chest between the scapula and the spine and beneath the clavicle, where it has a tympanitic, and at times on strong percussion a cracked-pot, character, best to be heard by listening at the open mouth of the patient. The tympanitic resonance is at times higher in pitch when the mouth is open than when shut. The outline of the displaced stomach or liver is to be determined also by percussion. On auscultation in the vicinity of the seat of pain in the early stage of pleurisy there is to be heard on inspiration and expiration a continuous sound, often compared to the creaking of leather, the pleuritic friction-sound, due to the rubbing of the apposed pleuræ roughened from adherent fibrin. As the liquid exudation accumulates this sound disappears, and the respiratory and vocal sounds are feeble or absent. When the liquid exudation is excessive and produces compression of the lung the breathing is bronchial, being loudest at the upper part of the chest, and especially in the back, and may be apparent as a distant sound in the region of dulness, but in children may be so loud as to suggest consolidation of the lung. The voice-sound also is feebly transmitted, except in the region of bronchial breathing, in which place there is bronchophony, and not infrequently in extensive serous exudation the transmitted voice-sound has a peculiar nasal character compared to the bleating of a goat, and designated ægophony. According to Baccelli, the whispered voice is transmitted through a serous but not through a purulent exudation.

In the course of two or three weeks after the onset of fibrinous pleurisy, or when there is but little serous exudation, the fever subsides, the exudation is absorbed, the friction-sound is again to be heard, though eventually disappearing, and recovery takes place by resolution. If the fever persists into the third or the fourth week and the physical signs of exudation are present, the disease, if untreated, may become chronic, lasting for months, in which case its tuberculous nature is especially to be suspected. The serous fluid then may be gradually absorbed, and a permanent thickening of the pleura with possible incomplete expansion of the lung and deformity of the chest be the result. If the liquid exudation is extreme, sudden death may occur from pulmonary embolism, from œdema of the unaffected lung, from weakened action of the heart in consequence of its displacement, or, according to Bartels, in left-sided pleurisy from obstruction of the passage of blood through the inferior vena cava in consequence of its compression by the exudation.

Suppurative pleurisy occurs at all ages, and when metapneumonic is usually limited to the region of the affected lobe, not becoming manifest until at least a week after the pneumonia has begun. The constitutional disturbances in empyema are apt to be considerable, but the affection of respiration is often slight. There is frequently a marked leukocytosis, and peptonuria has been observed. When the wall of the chest is perforated and the pus appears beneath the skin, so-called empyema necessitatis, œdema of the skin over the lower portion of the chest usually precedes the appearance of the pus, which is indicated by the appearance of one or more fluctuating subcutaneous tumors, generally in the vicinity of the fifth rib near the sternum.

When the empyema is in the left half of the chest, and in rare instances in empyema of the right half, the pulsations of the heart are transmitted by the exudation, and are to be seen or felt as a more or less forcible heaving of the chest-wall, oftenest in the second and third intercostal spaces, and synchronous with the beat of the heart, intrapleural *pulsating empyema.* The pulsations may be visible also in the superficial abscesses occurring in empyema necessitatis, and have been observed not only in the front of the chest, but also in the back and in the left lumbar region. Osler and Wilson have recently called especial attention to the occurrence of pulsating pleurisy, and in the collection of sixty-six cases, mostly in males, made by Wilson, the pleurisy was on the left side in sixty-one cases, and the exudation, almost invariably purulent, was intrapleural in twenty-eight instances, and was manifested as an empyema necessitatis in thirty-seven cases. Important in the production of pulsating pleurisy are a weakness of the intercostal muscles or a perforation of the parietal pleura and strong cardiac action. Toulmin has shown that an increased intra-pleural pressure is not essential, since the aspiration of several ounces of fluid was not followed by cessation of the pulsation.

Pneumothorax is a frequent complication of empyema. (See Pneumothorax, page 770.) Perforation into the bronchi, so often recovered from, is sometimes a cause of immediate death by the flooding of the lungs with pus.

Diagnosis.—The suspicion of a pleurisy is excited by the occurrence of thoracic pain, a short dry cough, rapid respiration, and elevated temperature. The diagnosis is based essentially upon the physical signs. Since many of the symptoms, and such of the signs as dulness on percussion, bronchial breathing, and bronchophony, at times occur in both pleurisy and pneumonia, these diseases are not infrequently confounded. In typical cases with abundant exudation there is but little difficulty in diagnosis, since the onset of pneumonia is sudden and usually announced by a chill, which is immediately followed by a marked elevation of temperature, pursuing a relatively typical course. There is a characteristic rusty sputum, and the chlorides of the urine are markedly diminished. Abundant, fine, moist, subcrepitant râles are followed by bronchial breathing and bronchophony, to be replaced by the return of the râles. In pleurisy, on the contrary, the onset is neither so sudden nor so violent, the fever is lower, the discomfort is less, and rusty sputa are absent. At the outset a continuous rub is to be heard, instead of subcrepitant râles at the end of inspiration. Where dulness is present the chest is distended, the vocal and respiratory sounds are distant or absent, not bronchial, the vocal fremitus is diminished, and the physical signs are not limited to the lobar structure of the lung. When the bronchi, however, are obstructed in pneumonia, bronchial breathing is absent and the sounds of the voice are not transmitted, and in moderate pleuritic exudation, especially when circumscribed between the lobes, there may be no characteristic dulness. Localized dulness and feeble respiratory and vocal sounds may be produced also by pleural thickening and by thoracic tumors. The absence of fever, the slow progress of the disease, and the freedom from symptoms of pressure upon the larger bronchi and blood-vessels may aid in the diagnosis of the tumor, but are of little avail in differentiating pleural thickening from pleuritic exudation. In pulsating pleurisy the presence of aneurism is not infrequently suggested, and, although in general the absence of murmurs and the seat of the dulness and pulsation are sufficient in diagnosis, the possibility of doubt is not always thus to be excluded. The pulsating tumor of empyema necessitatis also may be so seated as to suggest aneurism, but its tension is influenced by respiration, and pressure may empty the sac.

Sooner or later an exploratory puncture of the chest is likely to be demanded for therapeutic, if not for diagnostic, purposes. It is practically without risk if the ordinary precautions against septic infection are employed, even when several punctures are made, which are sometimes necessary, especially when the fluid is encapsulated. The withdrawal of fluid from the pleural cavity at once eliminates pneumonia, pleuritic thickening, and pleural tumors, unless combined with liquid exudation. If the fluid is free from blood, aneurism is excluded. The inflammatory instead of the dropsical nature of the serous fluid is indicated by its specific gravity, upward of 1015, its high percentage of albumin, and its frequent spontaneous coagulation. A bloody fluid, in the absence of the signs of aneurism, is suggestive of tuberculosis or cancer of the pleuræ, but may be found in pleurisy independently of these affections, as has been stated in the section on morbid anatomy. The presence of a thick pus is suggestive of a metapneumonic pleurisy, and the recognition of the diplococcus of pneumonia would confirm this view. The pus is thin in empyema from streptococcus infection, and both thin and offensive in putrid empyema. Circumscribed dulness and protrusion of the intercostal spaces, associated with an irregular range of temperature, due to the presence of pus in the subpleural tissue, *peripleuritis*, sometimes occur apparently spontaneously, and are with difficulty to be differentiated from an encapsulated empyema. Even when empyema is manifested by the exploratory puncture it may be necessary to know whether the exudation is above or below the diaphragm. Empyema or pyopneumothorax is to be diffentiated from subphrenic abscess or subphrenic pyopneumothorax first by the early symptoms, which in empyema relate to respiration and not to digestion. The signs of displacement of the lung, and perhaps of displacement of the heart, are present in suppurating pleurisy to a much greater extent than in subphrenic peritonitis. Especially important, according to Litten, in the differential diagnosis is the relation of the phrenic phenomenon to dulness. When the broad, moving shadow indicative of the entrance of the lower border of the lung into the complementary space is seen above the level of dulness, the cause of the latter is below the diaphragm. The upward displacement of the diaphragm by a large abscess of the liver produces the effect upon the lung of pleuritic exudation, but the outline of dulness is convex upward. Circumscribed pleurisy in the left half of the chest may with difficulty be discriminated from pericarditis, in the fibrinous stage of which a continuous friction-sound is to be heard; but its persistence when the breath is held excludes its pleural origin. When the pleuritic exudation is liquid and abundant the heart is dislocated, but the dyspnœa and discomfort are not so great as would be the case in pericarditic exudation with an equally extensive area of dulness.

Prognosis.—The prognosis of pleurisy varies mainly in accordance with the anatomical variety, which is largely dependent upon the immediate cause. In general it has been estimated that death occurs from pleurisy alone in about five per cent. of the cases. Dry or fibrinous pleurisy usually terminates favorably in the course of a few weeks. In serous or sero-fibrinous pleurisy absorption of the fluid may take place and recovery be established in the course of a month, but, as previously stated, death may occur suddenly when the quantity of the liquid exudation is excessive. This variety, however, not infrequently pursues a chronic course, perhaps manifested by frequent recurrences, and extends over a period of months. In such cases the prognosis is to be guarded, especially since nearly one-half of the sufferers sooner or later are afflicted with tuberculosis. Pulmonary phthisis was present in one-third of Bowditch's cases observed in the course of thirty years. In hemorrhagic pleurisy the prognosis is grave, though not necessarily hopeless, from the frequency with which extensive tuberculosis or malignant disease of the pleura is associated. Suppurative pleurisy has a doubtful prognosis. Frequent recoveries occurred before the days of its surgical treatment from the spontaneous evacuation of pus either into the lungs or through the walls of the chest in empyema necessitatis, but the mortality of empyema varies largely with reference to the cause and to the condition of the wall of the chest at the time of treatment. Metapneumonic empyema, the variety associated with pneumonia and due to the diplococcus of this disease, has a mortality ranging between two per cent. and ten per cent. In children it is frequently recovered from after a single aspiration of the pus. Empyema due to staphylococcus infection also may be recovered from after simple aspiration. If the pus contains streptococci the prognosis is more serious, since septic infection of the body is likely to exist, but, on the whole, is favorable, provided that its nature is early discovered, that its surgical treatment soon follows, and that there are no grave complications. The prognosis of ichorous pleurisy is also serious, but not necessarily fatal, if early surgical treatment is instituted. Empyema of tubercular origin is usually sooner or later fatal, either from amyloid disease following the long-continued drainage from the chest or from tuberculosis elsewhere. When there is extensive deformity of the chest in consequence of chronic pleurisy and inability of the lung to expand from fibrous pneumonia, the prognosis depends essentially upon the condition of the heart, the right side of which becomes dilated and hypertrophied. In the course of years insufficiency of the heart is likely to arise, and the prognosis becomes that of an incompetent heart.

Treatment.—In acute sthenic pleurisy with fibrinous exudation immediate relief to the pain and often distinct modification of the disease can be obtained by local blood-letting, by means either of leeches or of wet cups, to the extent of from three to seven ounces, according to the strength of the patient. Dry cups should never be used, as in thin-walled chests their irritating influence may extend through to the pleura. If the pain be not relieved, strapping the affected half of the chest in the manner practised for fracture of the ribs will often, by arresting respiratory movement, afford great comfort to the patient, and lessen the irritating influences upon the inflamed area of excessive movement. In some cases an ice-bag is agreeable to the patient, and may be employed. More usually warm moist applications, as poultices, are preferred. Internally calomel should be given in small doses at regular intervals, partly for the purpose of thoroughly emptying the alimentary canal, but largely for the effect which it has in lessening the amount of fibrinous exudation and also the inflammatory changes in serous membranes. Potassium iodide is in no way capable of replacing it, and even in chronic pleurisy with serous exudation the power of the iodide is doubtful.

When serous effusion has taken place, mustard plasters and similar rubefacients are of no value, and it is doubtful whether the local application of iodine, though much practised, has any effect. If the iodine be used it should be in the form of the saturated solution in oil, which should be well rubbed into the side twice a day. There can be no doubt as to the great value of large blisters during the forming stage of serous effusion. If the effusion does not increase, they should be repeated frequently at short intervals. The so-called "dry" treatment has for its object the lessening of the fluids of the body by cutting off the supply of water: its effectiveness is, however, doubtful. The liquid allowed in the twenty-four hours is reduced to eight or ten fluidounces, meat, dry bread, eggs, and other foods containing very little water being selected according to the needs of the case. During the period of dieting salines are freely to be given, once, twice, or three times in the forty-eight hours, according to the strength of the patient,—from one to two ounces of Rochelle salt, or from half an ounce to an ounce of Epsom salt, in a little water. The old-fashioned diuretic pill, one grain each of calomel, digitalis, and squill, is a very effective diuretic combination which may be employed—one every six to every eight hours—during the dry treatment. When the effusion rapidly fills almost the whole chest, or when in spite of the application of blisters it continues to increase, aspiration should be practised. The operation properly performed is without danger in a suitable case.

The rule formulated forty years ago by Henry I. Bowditch, of Boston, that "in any case of even moderate effusion lasting more than a few weeks, and in which there should seem to be a tendency to resist ordinary mode of treatment," aspiration should be practised, is correct; and when the pleural cavity is found full of fluid there should be no waiting to test the possibility of getting rid of the fluid by medicinal treatment. In the performance of paracentesis thoracis the antisepsis should be absolute. The skin should be thoroughly washed with soap and water, then treated for ten to fifteen minutes with a solution of corrosive sublimate 1 to 500, and, after the use of ice and salt for the purpose of producing local anæsthesia, should again be well washed with eighty per cent. alcohol immediately before the needle is introduced. The point of election is in the seventh interspace, below the centre of the axilla, or in the eighth interspace, at the outer angle of the scapula. The interspaces may be widened by raising the point of the elbow outward and upward. The needle should be thrust through close to the upper margin of the rib, so as to avoid the intercostal artery. The fluid should be taken away not too rapidly, from one to four pints, according to the amount of the exudate. Any symptoms of syncope should be the signal for the withdrawal of the aspirator, since sudden death is said to have occurred during pleural aspiration.

The condition of great distress, with albuminous expectoration and dyspnœa, spoken of by some writers, we have never seen. It is not rare for severe coughing to come on after some of the fluid has been taken out: if it be excessive, the aspirating needle should be withdrawn; and if the cough still continue, a hypodermic injection of morphine should be given.

After the aspiration the treatment directed for the removal of serous effusion in the preceding paragraphs may be often advantageously practised for the purpose of delaying the reaccumulation of fluid.

The treatment of empyema is that of abscess, but in children a single aspiration sometimes cures an empyema; if it fail, and in adults whatever the symptoms may be, however desperate the patient's condition may appear, the pleura should be freely opened and thoroughly drained. If the empyema be an old one, or if the discharge be in any degree fetid, the whole cavity should be well washed out with warm sterilized water. Resection of the rib and insertion of a drainage-tube are often necessary.

The general treatment should be supporting and symptomatic; advantage is often found in allowing the patients, as they grow stronger, to use several times a day an apparatus made by so uniting two large Wolfe bottles, or two half-gallon jars with rubber corks doubly perforated, that the patient can blow backward and forward from one to the other a half-gallon of water. The expansion of the lungs, which this apparatus is supposed to aid, may often be assisted with advantage by the cautious use of the pneumatic cabinet or of pulmonic gymnastics, and after convalescence by living at high elevations.

Chronic pleurisy is to be treated by aspiration when the fluid is in excess, by repeated blistering followed by the local use of a saturated solution of iodine in oil, and by building up the general condition of the patient. In many cases deep breathing, the pneumatic cabinet, and other forms of respiratory gymnastics are of service. When it is possible, in an obstinate case the patient should live in a dry, equable climate.

TUMORS OF THE PLEURA.

Primary and secondary pleural tumors are to be found. The former are fibroma, lipoma, osteoma, sarcoma, and endothelioma, and arise from the pleura or the subpleural tissue. The secondary tumors are lymphoma, sarcoma, and cancer, and invade the pleura from neighboring or remote parts by means of the blood-vessels or lymphatics. In such instances the primary seat of the disease is to be found in the mediastinal lymph-glands, the ribs, the mammary gland, the œsophagus, the stomach, or elsewhere. Those of especial clinical interest are the malignant tumors, among which are lymphoma, sarcoma, endothelioma, and cancer.

The morbid growth is manifested as a diffuse thickening of the pleura, or circumscribed nodules, often multiple and sometimes minute, are present. The new formation is hard or soft, often very vascular, and is usually present on the parietal and visceral layers. Hydrothorax or pleurisy is frequently associated, and considerable quantities of liquid may be the result. As a rule, the symptoms are those of a chronic progressive pleurisy associated with emaciation, pallor, and debility. Occasional stitches and slight disturbance of respiration, especially on exertion, may exist for some time before the evidence of fluid is present. In other cases the growth of the tumors is rapid, and is associated with fever and abundant exudation without especial disturbance of nutrition. The diagnosis is based ordinarily upon the presence of symptoms and signs suggestive of a pleurisy or hydrothorax, and is substantiated by the withdrawal from the chest of a bloody fluid in which, at times, the structural characteristics of a malignant new formation are to be found. The prognosis of malignant growths of the pleura is necessarily fatal, death occurring usually within a few months after dyspnœa becomes prominent or aspiration necessary. The treatment consists in the relief of symptoms as they arise, especially in the frequent withdrawal of fluid when it is a cause of dyspnœa, and in the relief of pain by opiates.

DISEASES OF THE MEDIASTINUM.

MEDIASTINITIS.

Acute and chronic inflammation of the fibrous tissue of the mediastinum occur: both are due to extension of inflammatory processes from neighboring parts. Acute mediastinitis follows injury or deep-seated inflammation of the fibrous tissue of the neck, whether proceeding from the vicinity of the submaxillary glands or originating as a retropharyngeal process. Acute mediastinitis at times is caused by infection of a wound in tracheotomy or in cut-throat. It is likely to result from perforation or rupture of the œsophagus, or from abscesses of the mediastinal lymph-glands, and to be continued from a pleurisy or a pericarditis.

Chronic mediastinitis may be the outcome of an acute mediastinitis, but is especially likely to accompany chronic inflammatory processes of the thymus or of the mediastinal lymph-glands or of the spine and the sternum. Especial attention has been directed of late years to the concurrence of mediastinitis and pericarditis, a condition designated *indurative mediastinitis* or *mediastino-pericarditis.*

Acute mediastinitis occurs either as a phlegmonous inflammation characterized by a gelatinous infiltration of the fibrous tissues with serum and cells, or as a substernal or periœsophageal abscess. In indurative or chronic fibrous mediastinitis the fibrous tissue of the mediastinum is thickened and dense, and fibrous obliteration of the pericardial cavity is frequently associated.

The symptoms of acute mediastinitis vary in accordance with the seat of the inflammation in the anterior or in the posterior mediastinum, and are dependent also upon the quantity of the exudation. Chills and fever, sweats and prostration, delirium or stupor, and a rapid and weak pulse, which are present to a greater or less extent, are attributable to the primary inflammation. Evidence of the localization of the inflammation in the mediastinum is afforded by substernal pain, especially near the ensiform cartilage, and sometimes very severe, and dyspnœa or difficulty in swallowing when the abscess is of sufficient size to produce mechanical interference with breathing and deglutition. The presence of a large abscess in the anterior mediastinum not only causes dyspnœa and weakening of the action of the heart, but also may be manifested above or at the side of the sternum by an elastic, perhaps fluctuating, swelling, which may transmit pulsation from the neighboring arteries. The pulsation, however, is not expansile, there is no double murmur, and the heart-sounds are feebly transmitted. The pus may be discharged into the œsophagus or into the trachea. It may enter the pleural cavity or be discharged externally through an intercostal space in the vicinity of the sternum, or it may escape through the abdominal wall. Acute mediastinitis is a severe affection, and not infrequently proves the cause of death soon after its occurrence.

Chronic mediastinitis is sometimes manifested by an abscess, in which case its origin from a tubercular process is probable. The pus may become inspissated, or, as in the case mentioned by Da Costa, be discharged by the mouth after a year of symptoms resembling those due to aortic aneurism. In chronic fibrous mediastinitis associated with an obliterated pericardium and hypertrophy and dilatation of the heart the symptoms are shortness of breath, cyanosis, and dropsy, not infrequently associated with increasing weakness of the pulse on inspiration,—the paradoxical pulse of Kussmaul.

TREATMENT.—A mediastinal abscess should be aspirated, great care being taken to prevent the admission of air during or after the operation.

TUMORS OF THE MEDIASTINUM.

Tumors of the mediastinum are of occasional occurrence, and proceed from the thymus, the mediastinal lymph-glands, and the fibrous tissue or the thyroid gland, especially a supernumerary or an aberrant thyroid. The primary tumors are lymphoma of the thymus or of the lymph-glands, sarcoma of the fibrous tissue, and, rarest of all, dermoid cyst. The secondary tumors proceed from the lymph-glands, which are often cancerous and sometimes sarcomatous in consequence of malignant disease elsewhere in the body. The mediastinal tumor oftenest found is lymphoma, usually multiple, and occurring independently as malignant lymphoma or in connection with similar tumors of the lymph-glands elsewhere in the body in leukæmia and in pseudo-leukæmia.

Symptoms.—The disturbances caused by tumors of the mediastinum are due to their pressure upon adjacent parts. One of the earliest as well as one of the most persistent symptoms is dyspnœa, which may result from pressure upon the trachea or a bronchus or upon the lung itself, and may be caused also by compression of the pneumogastric nerve, in which case paroxysms of dyspnœa of an asthmatic character result. The dyspnœa often is added to by fluid in the pleural cavity. Orthopnœa eventually may occur and persist until death takes place. Usually, next in sequence are the manifestations of pressure upon the veins entering the thorax, especially of the superior vena cava or of an innominate vein. If the superior vena cava is obstructed, there is venous congestion of the head, arms, and upper half of the body, manifested by headache, vertigo, ringing in the ears, and cyanosis and œdema of the skin. The obstruction may be so considerable that there is conspicuous dilatation of the anastomoses between the cutaneous veins of the chest and of the abdomen. If but one innominate vein is compressed, the cyanosis and œdema are limited to the corresponding half of the face, neck, chest, and arm. If the tumor is so situated as to press upon the inferior vena cava, there is œdema of the lower half of the body. The effect of pressure upon the arteries proceeding to the arm may be manifested by differences in the strength of the radial pulses. Pressure upon the œsophagus causes difficulty of swallowing. This symptom also results from pressure on the pneumogastric nerve, and, in addition, the frequency of the pulse is diminished if the nerve is irritated, and quickened and irregular when the nerve is paralyzed. Pressure on the phrenic nerve causes hiccough, and quickened respiration also, from interference with the movement of the diaphragm. There are hoarseness or aphonia from paralysis of the vocal cords if the laryngeal nerves are compressed, and irregularity of the pupils from pressure upon the sympathetic nerves. Numbness or pain in the chest and arms at times occurs, in consequence of pressure upon the nerves proceeding to these parts. There is a frequent dry cough, which is sometimes stridulous, and blood is occasionally mixed with the sputum. There may be but little disturbance of nutrition, or the appetite may fail and emaciation rapidly take place. The patient is often distressed from loss of sleep.

The tumor may be so large as to cause circumscribed distention of the chest and impairment of its motion, and at times is to be seen or felt in the sternal notch, in which case it may transmit the pulsations of the larger arteries. In malignant lymphoma enlargement of the supraclavicular or axillary glands on one or both sides may be felt. In rare instances the tumor may perforate the wall of the chest and appear as a subcutaneous nodule. The heart is at times displaced downward and outward. On percussion there is an irregular area of dulness, usually in the region of the upper half of the sternum. On auscultation murmurs may be heard from pressure upon the large blood-vessels, and in the region of dulness the voice-sounds and the respiratory murmur are enfeebled. If a large bronchus is compressed, a localized musical murmur is likely to be heard, the respiratory sounds being feeble and the expiration prolonged in that part of the lung which is reached by the obstructed bronchus. If the tumor overlies the heart, the valvular sounds are somewhat indistinct.

DIAGNOSIS.—The symptoms and signs generally are those indicative of an intra-thoracic tumor, and are often suggestive of an aneurism of the aorta. An expansile pulsation, however, and a characteristic double murmur are lacking. The malignant nature of the tumor is directly to be inferred by the presence of enlarged glands in the neck or axilla. Pleurisy is to be excluded by the absence of fever, the irregular area of dulness, and the usual limitation of the symptoms of pressure to the upper part of the chest. The use of the exploring needle may be necessary in the differential diagnosis of pleuritic exudation and mediastinal abscess. When hydrothorax or pleurisy complicates a mediastinal tumor, the aspirated fluid may be bloody ; its removal is not followed by relief of the symptoms of pressure, and the fluid quickly returns. The dulness due to pericardial effusion may suggest that from a mediastinal tumor, but pericarditis is preceded by inflammatory symptoms, and the apex-beat is to be recognized between the sternum and the outer border of dulness, and not at this point, as in tumor.

PROGNOSIS.—Mediastinal tumors, except in case of the rare dermoid cyst or hydatid, are beyond the reach of the surgeon, and the prognosis, therefore, is in general to be regarded as hopeless. Death usually occurs in the course of weeks or a few months after the onset of the symptoms, and is due to exhaustion or gradual asphyxia, the intelligence of the patient often being preserved up to the time of death.

TREATMENT.—Mediastinal tumors, so far as their therapeusis is concerned, are chiefly surgical disorders, but a trial should be made of arsenic in malignant lymphomata, and Lugol's solution should be exhibited when the tumors are of thyroid origin. Considerable accumulations of fluid in the pleural cavity are to be removed by aspiration.

SECTION V.

DISEASES OF THE DIGESTIVE APPARATUS AND OF THE PERITONEUM.

CHAPTER I.

DISEASES OF THE MOUTH, TONGUE, SALIVARY GLANDS, PHARYNX, AND ŒSOPHAGUS.

STOMATITIS.

DEFINITION.—Inflammation of the mucous membrane of the mouth.

The mucous membrane lining the mouth is frequently the seat of inflammation, which is often diffused, though sometimes circumscribed to the tongue, gums, palate, or cheeks.

ETIOLOGY.—Of first importance in the causation of stomatitis are local irritants, among which are included excessively hot or acrid articles of food, tobacco, chewed or smoked, caustics, the eruption of teeth in infants, carious teeth or ill-fitting artificial teeth, the putrefactive changes in particles of food retained in consequence of insufficient care of the teeth, the direct inoculation of pathogenic bacteria or fungi, or their transfer, as in erysipelas, from the neighboring surface of the body through the lymphatics. Stomatitis also results from the extension of inflammatory processes into the mouth from the nose or the pharynx. It takes place in the course of infectious diseases, especially in measles, scarlet fever, small-pox, and syphilis, also in scurvy, and is a not infrequent complication of diphtheria. In this connection the repeated occurrence of stomatitis as an epidemic may be mentioned. Certain poisons, notably mercury, arsenic, bismuth, lead, and potassium iodide, act, after their absorption, as causes of stomatitis.

VARIETIES.—The varieties of stomatitis usually differentiated are the catarrhal, aphthous, ulcerative, gangrenous, and parasitic. In *catarrhal stomatitis* the mucous membrane, especially of the gums and cheeks, is reddened and swollen, and there is an increased secretion of mucus and saliva. Blisters, usually small, often form, break, and leave behind a raw surface. The catarrhal variety of stomatitis may become purulent in severe cases, as from gonococcal infection.

Aphthous stomatitis is a variety of catarrhal stomatitis in which somewhat painful, small, round, gray spots with a red margin appear at the edge of the tongue or upon the cheek. These spots, popularly spoken of as canker, may become confluent, and are due apparently to a thickening of papillæ from cellular or fibrinous exudation.

In *parasitic stomatitis*, or thrush, the catarrhal stomatitis is accompanied with soft, curd-like, slightly elevated, opaque white patches, to be scraped from the surface, which is slightly reddened. These patches are composed of the mycelial threads and buds of a fungus, the *oïdium albicans*, which grows between the epidermic layers of the surface. Thrush occurs particularly among feeble infants improperly fed, especially from unclean bottles, but is to be found also in adults enfeebled by disease or want. The fungous patches may extend from the gums and cheeks to the pharynx and the œsophagus.

Ulcerative stomatitis represents a more severe variety than those preceding, and is especially found among children crowded together under unsatisfactory hygienic surroundings; it also occurs in mercurial poisoning, and as a result of scurvy, in which case the exudation is hemorrhagic as well as purulent. The swelling of the gums is more conspicuous than in simple stomatitis, and ulcers are to be seen especially in the vicinity of the teeth. The inflammation may extend to the alveoli and to the periosteum, causing loss of teeth and necrosis of bone.

Gangrenous stomatitis is a rare affection, probably of bacterial origin, occurring especially among children, although sometimes found in adults. The disease is considered non-contagious, although in repeated instances numerous persons, especially in asylums, have been simultaneously affected. It is likely to occur among persons reduced by hardship or by severe diseases, although it sometimes is found in those otherwise in good health. This variety of stomatitis is also known as *noma* or *cancrum oris*, and is essentially a rapidly spreading moist gangrene. It usually begins at the corner of the mouth, where a black discoloration of the mucous membrane is surrounded by an inflammatory swelling. As the central necrosis is extended the peripheral inflammation advances.

SYMPTOMS.—In catarrhal stomatitis the complaint is of a sore mouth, disagreeable taste, offensive odor of the breath, and unwillingness to take food, partly from the discomfort of swallowing, partly from a lack of appetite. The unwillingness to swallow explains the frequency of drooling, and the consequent excoriation of the skin from maceration of the epidermis. There is little or no elevation of temperature. In aphthous stomatitis the inflamed spots are discomforting, but not especially painful. Salivation and difficulty in swallowing are inconsiderable. In thrush the local disturbances are still less, the mucous membrane being only slightly sensitive when the fungous patches are detached, but the possibility of œsophageal obstruction from the growth of the fungus in the gullet is to be remembered. These varieties of stomatitis are relatively mild, of short duration, and easily relieved by treatment.

In ulcerative stomatitis similar symptoms occur, but are severe. The flow of saliva is more profuse, pus and blood are present in the secretions from the mouth, and the odor of the breath is extremely offensive. The neighboring lymph-glands are swollen and tender, there is distinct febrile disturbance, and the disease is likely to have a protracted course.

In gangrenous stomatitis the swelling of the lymph-glands and the fever are even more extreme than in ulcerative stomatitis, and hemorrhages are abundant. Sopor or delirium may be present. Bronchopneumonia and pulmonary gangrene result from the inhalation of putrid material from the mouth, and the disease usually terminates fatally in a few weeks, the course being that of a septicæmia.

TREATMENT.—The treatment of aphthous stomatitis and that of ulcerative stomatitis are practically the same. Any derangement of the health, and especially of the digestive organs, must be carefully corrected. The individual ulcers may be touched with a point of solid silver nitrate, care being exercised to avoid the sound mucous membranes. Borax is often a useful application, but potassium chlorate is much more effective. Its influence is probably altogether local, but, as it is freely eliminated with the saliva, it is better to give it internally, to maintain a steady application. From three to five grains of the dry powder, with a little sugar, may be put upon the diseased mucous membrane every two to four hours in the case of a child; from five to ten grains for an adult. Thymol mouth-washes are sometimes comforting.

Parasitic stomatitis, or thrush, is to be chiefly overcome by the careful treatment of the condition upon which its existence depends. Frequently it remains incurable so long as the hygienic surroundings of the child are not of the best character; in bad cases it may be necessary to give the child breast-milk. If artificial feeding be maintained, it is essential that the mouth of the child, the nipples, and the bottles be kept as clean as possible. Solution of sodium sulphite (a drachm to the ounce), of boric acid (saturated), or of potassium permanganate (five grains to the ounce), may be applied locally after each feeding.

Gangrenous stomatitis is especially a disease of extreme prostration and exhaustion, so that stimulants and very careful feeding form the basis of its treatment. The sloughing portions are to be destroyed by one of the strong acids, or, what is probably better, by the cautious application of Paquelin's or of an electro-thermic cautery; subsequently antiseptics, especially thymol and carbolic acid, should be freely used. The child should be kept in the open air day and night, as far as practicable.

GLOSSITIS.

DEFINITION.—Inflammation of the tongue.

Inflammation may affect independently the surface and the parenchyma of the tongue. Superficial glossitis usually occurs in the various forms of acute stomatitis, but a series of changes are at times to be found which are usually regarded as evidences of chronic inflammation. Among these is the loss of epithelium in spots and patches, leading to more or less extensive erosion of the surface of the tongue, which becomes irregularly outlined, the lines upon a map being suggested, whence the term *geographic tongue.* This variety of glossitis is found especially among infants and young children, and has been regarded as evidence of a disturbance of innervation as well as a manifestation of inflammation. In adults the occurrence of a somewhat similar affection has been described by Möller, and is accompanied by a sensation of heat and digestive disturbances. The *dissected tongue* is a congenital alteration characterized by deep furrows upon the surface, in consequence of which deformity of the tongue results.

Buccal leukoplakia is also to be regarded as a variety of superficial glossitis, and is characterized by the presence upon the back and edges of the tongue of slightly elevated, more or less rounded, somewhat translucent patches of an opaque gray color. They are due to enlargement of the papillæ and to the excessive formation and accumulation of epidermis. Other terms which have been applied to this condition are *ichthyosis, psoriasis,* and *keratosis* of the tongue. Excessive smoking is usually considered to be the commonest cause, and many observers have regarded it as a manifestation of syphilis. The latter view is opposed by the frequent lack of other evidence of syphilis and by the failure of antisyphilitic treatment to afford relief. It is possible, however, that the syphilitic patient may be more prone to this affection in consequence of an increased vulnerability of the mucous membrane, either from the frequent localization of syphilitic lesions in the mouth or from the effects of mercurial treatment. The white patches last for months or years and obstinately resist treatment. In repeated instances cancer has developed from the diseased patch. The treatment consists in the careful removal of any source of local irritation, in the thorough treatment of digestive disturbances, and in the local application of mild stimulating solutions. The best of these is probably that of chromic acid, three to five grains to the ounce.

PARENCHYMATOUS GLOSSITIS.

Parenchymatous glossitis is a relatively rare affection, characterized by enlargement of the whole, more rarely of one-half, of the tongue, which may protrude from the mouth or cause it to be kept constantly open. Slight degrees of acute parenchymatous glossitis occur in stomatitis. Extreme degrees, however, represent a local infection of the tongue, as in foot-and-mouth disease or in consequence of injury or of poisoning from the stings of insects. The enlargement of the tongue is due to the presence of an increased quantity of lymph or of pus, according to the severity of the process, in the interstitial tissue. There are prostration, fever, and difficulty of talking, swallowing, and breathing. The tongue is painful, saliva flows from the mouth, and the neighboring lymph-glands are swollen. Acute parenchymatous glossitis ends in resolution or in the formation of an abscess.

An acute *œdematous enlargement of the tongue*, accompanied with a burning sensation, sometimes occurs both in children and in adults in consequence of disturbance of gastric digestion. This is closely allied to giant urticaria, and is easily to be discriminated from acute inflammatory enlargement of the tongue by the rapidity of its onset, its brief duration, and the absence of constitutional disturbance.

Chronic parenchymatous glossitis results from repeated attacks of acute inflammation, and may lead to considerable enlargement of the tongue, associated with excessive salivation, and causing difficulty of speech and swallowing. Pressure of the enlarged tongue against the teeth may lead to ulceration, and has required extraction of the teeth. Myxœdematous enlargement is a rare variety of chronic glossitis, and its nature is to be recognized by the evidences elsewhere of myxœdema. A congenital enlargement of the tongue,—*macroglossia*,—essentially a cavernous lymphangioma, is to be discriminated from chronic glossitis by its occurrence at birth.

TREATMENT.—In the treatment of acute glossitis, ice should be continually applied to the inflamed tongue. If the swelling be very severe, longitudinal scarification sometimes gives relief. If pus forms, it should be at once freely evacuated. In rare cases tracheotomy is necessitated by the interference with the respiration.

RANULA.

This term is applied to the deformity produced by a cystic tumor in the floor of the mouth containing a slimy fluid and giving an appearance which suggests the mouth of the frog. The cyst, sometimes of considerable size, lies below and at the side of the tongue, which is carried towards the roof of the mouth : the effect upon speech, swallowing, and respiration is essentially that resulting from enlargement of the tongue. Several explanations have been offered for the occurrence of the cyst. According to some authorities, obstruction of the ducts from the sublingual or submaxillary glands is the cause. Von Recklinghausen considers closure of the ducts of Blandin's glands, which lie beneath the tip of the tongue, to be the essential feature in etiology. It has been suggested also that the cyst may arise from the bursa mucosa found by Fleischmann at the side of the frænum. It is thus probable that several distinct conditions are included under ranula. In general, the deformity is persistent, but we have seen it occur in connection with stomatitis, last a few days, and then quickly subside.

TREATMENT.—The only treatment of chronic ranula is surgical. Three methods are in vogue : first, partial excision of the sac ; second, the introduction of the seton ; third, the injection of irritant fluids. Of these the first is to be preferred, though a second operation is often required. The seton and injection methods are apt to be accompanied by closure of the opening and danger of septic infection. When the ranula is an acute retention cyst, the treatment is that of catarrhal stomatitis. It disappears as the swelling of the duct subsides.

INFLAMMATION OF THE SALIVARY GLANDS. PAROTITIS.

Inflammation of the parotid gland, as a rule, arises from the advance of an inflammatory, probably bacterial, irritant from the mouth along the duct of Stensen. In most cases the irritant is the agent which causes the infectious and contagious disease mumps. The excitant of the parotid inflammation may be admitted to the gland also by means of the blood-vessels. Parotitis occurs as a secondary condition in a number of infectious diseases, especially in typhoid fever, typhus fever, measles, scarlatina, and pneumonia, and in the traumatic infections, including puerperal sepsis.

The enlarged gland varies in appearance according to the severity of the disease. The lobules are red, reddish gray, or yellow, according as the presence of blood or that of pus is the more conspicuous. The interstitial tissue is swollen, and either gray and translucent or opaque yellow, according as the contained exudation is serous or purulent. Abscesses may eventually be formed, and appear on the surface of the gland as opaque yellow spots, which may prove the source of a purulent infiltration of the surrounding tissue. This may extend upward into the auditory meatus, or into the cranial cavity through the glenoid fossa, or inward into the mouth or the pharynx, or outward to the skin.

Secondary inflammation of the parotid is manifested by a tender, painful swelling in the region of the parotid gland. The pain rapidly becomes severe, and the swelling produces a marked deformity of the region affected. There are high fever and decided prostration. Talking and eating are painful, and the pain often extends to the ear.

When the inflammation ends in suppuration, the abscess may break through the skin or the pus escape from the auditory canal or from the mouth. Extension of the suppuration to the cranial cavity causes headache, delirium, perhaps convulsions, and sopor suggestive of meningitis.

Secondary parotitis has a grave prognosis, and, even if recovery results in consequence of appropriate surgical treatment or of spontaneous evacuation of the pus, permanent deafness may follow from the complicating otitis, and facial paralysis occur from the extension of the inflammation to the facial nerve in its course through the gland.

INFLAMMATION OF THE SUBMAXILLARY AND SUBLINGUAL GLANDS.

As a rare condition in mumps the inflammation of the salivary glands may be limited to the submaxillary or sublingual glands. Usually the inflammation of these glands is extended from the mouth in the severer variety of stomatitis, although it may be localized in the glands without any primary buccal disturbance. Its possible occurrence as a complication in infectious diseases, as diphtheria and scarlet fever, should be remembered.

A painful, tender swelling makes its appearance in the posterior submaxillary region, and is associated with fever and difficulty of speaking and eating, in part in consequence of the elevation of the tongue and in part from the pain on motion of the muscles. The inflammation may end in resolution in the course of a fortnight, more or less, or extend to the tissues around the gland, in which case a deep-seated lymphangitis follows, terminating in suppuration or gangrene. This phlegmonous lymphangitis or cervical cellulitis has been designated *angina Ludovici*, Ludwig's angina, from the name of the physician who first called conspicuous attention to it. It is of rare occurrence, occasionally as an epidemic, and has been regarded by some writers as a disease *sui generis.*

It is characterized by a dense swelling which makes its appearance beneath the jaw and in the upper part of the neck and may cause œdema of the larynx or pressure upon the trachea. The fever becomes more extreme and assumes a typhoidal course indicative of a severe septicæmia. The lymphangitis may extend downward into the mediastinum and pleurisy or pericarditis follow, or, if suppuration is present, the pus may reach the surface and the abscess open through the skin or into the mouth or the pharynx. The prognosis of the severer forms is extremely grave.

TREATMENT.—In acute inflammation of the parotid or other salivary glands, not due to mumps, an attempt may be made, by the application of leeches followed by ice, to arrest inflammation ; if, however, suppuration occurs, the affected parts should be freely opened and afterwards receive the treatment of a septic inflammation.

From a therapeutic point of view, at least, Ludwig's angina is a surgical disease, the principles of whose treatment are the use of early and ample incision, with rigid antisepsis, and general support by feeding and medication. As has been especially insisted upon by Gerster, it is probably best to make a careful dissection of the submaxillary region, with a final incision through the mylo-hyoid muscle, followed by repeated thorough irrigation with a saturated solution of boric acid or with solution of corrosive sublimate one to one thousand, or with a hydrogen peroxide solution.

INFLAMMATION OF THE PHARYNX AND THE TONSILS.

Inflammation of the pharyngeal mucous membrane, *angina*, although usually associated with that of the tonsils, the superficial lymph-glands of the pharynx, may occur without any involvement of the latter : hence a clinical distinction is usually made between pharyngitis and tonsillitis, according to the more conspicuous localization of the inflammatory process, although there is often no difference in etiology.

ACUTE PHARYNGITIS.

ETIOLOGY.—A primary pharyngitis is to be distinguished from the secondary inflammation of the pharynx occurring in acute infectious diseases, as measles, scarlet fever, variola, influenza, and diphtheria, or in chronic infections, as syphilis. The primary affection is especially frequent among children, is usually attributed to exposure to cold, and is often associated with a nasal or a laryngeal catarrh; but it is probable that infection also plays an important part in the etiology. Bacteria are always present in the pharynx, and it is not unlikely that exposure to cold may offer favoring opportunities for their admission to the tissues. The frequent occurrence of epidemics of pharyngitis and tonsillitis also points towards an infectious origin, and the occasional succession of cases within a short time and in the same family is suggestive of its contagious nature. Local irritants are of importance, and the influence of faulty drainage in the development of pharyngitis and tonsillitis is often apparent. Some writers assert that gout and rheumatism are productive of inflammation of the pharyngeal mucous membrane, and that in acute articular rheumatism, in particular, inflammation of the tonsils is a frequent complication. Pharyngitis and tonsillitis, however, are affections of extraordinary frequency, and their occasional occurrence in rheumatism and in gout is to be expected. In our experience they are not so common in these affections as to seem dependent upon them. Inasmuch as acute articular rheumatism is probably an acute infectious disease, a localization of the infectious cause might be expected as well in the pharynx as in other parts of the body, and with as much reason as in typhoid fever or scarlet fever. Such localization, however, is decidedly infrequent.

SYMPTOMS.—Pharyngitis is acute or chronic, superficial or deep-seated. Superficial pharyngitis is a catarrhal inflammation, the deep-seated variety is phlegmonous.

Acute catarrhal pharyngitis (simple sore throat) may occur without any especial warning, and is not infrequently preceded by the symptoms of a mild nasal or laryngeal catarrh. It is often announced by chilliness followed by slight elevation of temperature. As a rule, there is but little constitutional disturbance; at the most, slight headache, backache, and loss of appetite. The soreness of the throat is early made apparent by discomfort in swallowing and by a sense of dryness. The soft palate and the uvula are reddened and moderately swollen, and the posterior wall of the pharynx is congested, perhaps covered with a layer of opaque gray mucus. The severer manifestations of acute catarrhal pharyngitis are associated with conspicuous alteration of the tonsils, and will be described in connection with tonsillitis.

Acute pharyngeal catarrh is usually a trivial affection, running a mild course, lasting for a day or two, and not necessarily interfering with the daily vocation of the patient. It is important only as probably representing the mildest stage of what may prove a severe disease, and it is always to be remembered that during the first twenty-four hours there may be no means, other than bacteriological, by which a simple catarrhal sore throat can be absolutely distinguished from a diphtherial sore throat.

In phlegmonous pharyngitis the irritant invades the submucous tissue and causes an acute œdema or a purulent infiltration which rapidly extends throughout the pharynx. There are great elevation of temperature, quick pulse, rapid respiration, pain in swallowing, hoarseness of the voice, and dyspnœa. The uvula, the soft palate, the palatine arches, the mucous membrane of the posterior pharyngeal wall, and perhaps the tonsils, are greatly swollen and livid. The neck is enlarged, painful on motion, and the submaxillary lymphatic glands are increased in size and tender. Death may occur suddenly from œdema of the glottis, or the inflammation may extend downward towards the mediastinum, as in inflammation of the submaxillary glands, and a complicating pleurisy or pericarditis result, or the pus escape by the mouth or through the skin.

In young children, especially, phlegmonous pharyngitis assumes the characteristics of an acute *retropharyngeal abscess.* The difficulty of swallowing, the rigidity of the neck, the altered voice, and the labored breathing are associated with a fluctuating swelling of the posterior pharyngeal wall. The chronic retropharyngeal abscess also causes a fluctuating tumor in this region, but it is usually the result of caries of the cervical vertebræ, and the severe symptoms of acute pharyngitis are lacking.

TREATMENT.—In the mildest cases of acute pharyngitis it is only necessary to swab the throat out twice a day with glycerite of tannin, or to apply carefully a solution of silver nitrate twenty to forty grains to the ounce, or to use a gargle of rhus glabra (formula 21) every three to four hours. In severer cases, with fever, a quarter of a grain of calomel should be given every two hours until it purges freely ; three to five drops of belladonna should be administered four times a day, unless dryness of the mouth supervene, when the dose should be reduced ; and the glycerite of tannin or the gargle should be used every two hours. A local application of a strong solution—forty grains to the ounce—of silver nitrate may be useful. The external application of the ice-collar and the free, continuous use of cracked ice is often very serviceable. Five to ten grains of quinine may also be given daily.

The diet should be light but nutritious. Milk and milk foods, broths, and farinaceous foods should be chiefly taken. Ice-cream is especially grateful. Meat should be allowed very sparingly, if at all.

A retropharyngeal abscess should be surgically evacuated as soon as the diagnosis is clear. In most cases it is probably better to make the incision from without rather than through the wall of the pharynx.

Violent hemorrhage from an erosion of a large blood-vessel is almost always fatal, though life has been saved by an immediate tying of the common carotid.

CHRONIC CATARRHAL PHARYNGITIS.

Chronic inflammation of the mucous membrane is the frequent result of repeated attacks of acute pharyngeal catarrh, but is due also to prolonged local irritation of the throat, either from tobacco, alcohol, and highly seasoned food, or from excessive use of the voice,—for example, by clergymen. Chronic passive congestion of the mucous membrane in obstructive cardiac and pulmonary disease is likewise a cause.

The throat is usually dry, but there is often a tickling sensation from the elongated uvula or a feeling as if secretion were dropping from the naso-pharynx. The patient awakes at night with a choking sensation, and on rising makes repeated efforts to clear the throat from a tenacious secretion which frequently forms a crust upon the posterior pharyngeal wall. Hawking is frequent during the day for the same purpose, and the voice is usually husky. The mucous membrane either is congested, redundant, and flabby, with visible enlargement of the veins and a coarsely granular appearance of the surface from hypertrophy of the lymph-follicles, or is pale, thin, tense, and shining from atrophy,—*pharyngitis sicca.* Chronic pharyngitis is an obstinate affection, subject to exacerbations and periods of temporary relief, but in the adult is extremely resistant to all attempts at permanent cure. The extension of the inflammation to the nostrils, Eustachian tube, and larynx is frequent, and headache, deafness, chronic cough, and permanent alteration of the voice often result.

In children, chronic catarrhal pharyngitis is especially characterized by the enlargement of the lymph-follicles in the mucous membrane of the roof and posterior wall of the naso-pharynx, in which they form reddish-gray, fleshy masses either nodular or papilliform, perhaps pedunculate, frequently filling the pharyngeal vault and interfering with nasal respiration. The secretion from the inflamed surface is abundant, and is usually swallowed. These "adenoids," or enlarged pharyngeal tonsil, are a frequent cause of mouth-breathing, and in children so affected the mouth is constantly open, the lips are thickened and everted, and the expression is one of stupidity. There are snoring, a thick voice, and frequent mental and physical sluggishness. With the persistence of this follicular pharyngitis the roof of the mouth becomes narrowed and raised. Nasal and auditory catarrh are often associated, and headache, and impairment of the senses of smell, taste, and hearing, are likely to follow. Deformity of the thorax may result, the upper part of the chest being distended, the lateral regions corresponding to the insertion of the diaphragm being depressed. Children with adenoids are liable to attacks of nightmare, and may suffer from asthma.

TREATMENT.—In the treatment of chronic pharyngitis it is essential, if possible, to remove the cause. Thus, in many cases cure cannot be obtained until the subject ceases from smoking or the use of tobacco, or from the taking of strong alcoholic drinks or highly spiced food, or from an excessive and improper use of the voice. Again, it may be necessary, by general treatment, by hygienic management, or by travel, to build up the general health. Locally, markedly hypertrophied parts may be touched by the galvano-cautery, whilst solutions of tannic acid or silver nitrate or chromic acid, and of the various other local remedies, are regularly applied.

Adenoid growths occurring in the pharynx—the so-called *pharyngeal tonsil*—should always be removed at once, as, if left, they often seriously affect the general health of the child, and as other than radical measures are of no avail. The child should be anæsthetized, and the adenoid tissue scraped off with the finger-nail or a curette. If, after removal of the cause, the habit of mouth-breathing which has been formed persist during sleep, the jaw should be kept up by a chin-strap. In most cases general hygienic treatment of the child is necessary.

TONSILLITIS.

As already stated, tonsillitis is to be considered as a form of pharyngitis in which the superficial lymph-glands of the pharynx—namely, the tonsils—are especially altered, and the infectious varieties of pharyngitis are those especially concerned in the production of acute inflammation of the tonsils. Acute tonsillitis is to be distinguished from chronic tonsillitis, and is the variety to which the terms follicular and lacunar tonsillitis have been applied. It is generally known, especially in consequence of the observations of Stöhr, that the tonsils are covered with mucous membrane which extends into and lines the recesses, crypts, or lacunæ, the walls of which are studded with lymph-follicles, and in which a few mucous glands are to be found. It is in virtue of the relation of the inflammatory product to the lacunæ that the term lacunar tonsillitis is applied.

ACUTE TONSILLITIS.

Acute tonsillitis usually begins with a chill, sometimes violent, followed by fever, the temperature rising rapidly to 103° F. and upward, and associated with headache, backache, pains in the bones, and muscular weakness. There is loss of appetite, with mental and physical prostration, and wakefulness is often present. Pain in swallowing is soon manifested, and rapidly increases. At the outset the mouth is dry and the tongue coated, but later there is an increased flow of saliva from the mouth, largely in consequence of difficulty in swallowing. On examination of the throat, in addition to the general swelling and congestion of the pharynx, a conspicuous enlargement of one or of both tonsils is apparent. The enlarged and reddened tonsils may almost wholly fill the opening of the pharynx, and soon become covered with a thin, in part translucent, in part opaque gray, film which apparently exudes from the crypts and becomes confluent. At the opening of the crypts are to be seen opaque white spots, which are composed of leukocytes, bacteria, and particles of food. The lymph-glands at the angle of the jaw are swollen and tender on one or both sides, according as one or both tonsils are inflamed. Pain extending into one or both ears and impairment of hearing are not infrequently associated. Albuminuria is frequently found.

In mild cases of tonsillitis convalescence usually begins in three or four days, and the swelling of the tonsils and difficulty of swallowing disappear in the course of a week. In the severest cases of tonsillitis, to which the term *quinsy* is especially applied, an abscess forms in the inflamed tonsil. On the third or fourth day of the tonsillitis there is no diminution in the severity of the symptoms, but the enlarged and resistant tonsil becomes soft and fluctuant. The abscess may break suddenly, usually into the mouth or the pharynx, when the local symptoms often at once disappear and rapid relief to the constitutional disturbance follows. In rare instances the pus has entered the larynx, causing suffocation, and still more rarely the suppurative inflammation has extended to the internal carotid artery, which has been perforated.

TREATMENT.—The treatment of acute catarrhal tonsillitis is the same as that already given for acute pharyngitis. It is believed by many practitioners that both this form of tonsillitis and suppurative tonsillitis are often of rheumatic origin, to be benefited by the use of salol or the salicylates.

In quinsy, to avoid the formation of pus all that can be done is to cleanse the throat with a very dilute solution of hydrogen peroxide, or a thymol mouth-wash, often preferably used by spray, and to soothe the irritation by the free internal and external use of ice. Temporary relief is sometimes afforded by scarification of the tonsils, and certainly any pus should be evacuated as soon as it has formed. If the abscess is in the soft palate, a little above and on the outside of the margin of the tonsil, the incision should be through the soft palate, just outside of and parallel to the anterior pillar, and in the neighborhood of the line of the upper margin of the tonsil. If the tendency is for the pus to escape through the crypt of the tonsil, the incision should be into the tonsil, as near as possible to the natural outlet of the pus. When the pus burrows downward it is often most difficult to reach, and in some cases when there is much swelling even an external incision may be necessary.

The diet should be as nutritious as possible. Ice-cold foods can often be swallowed when even ordinary warm liquids are rejected ; hence ice-cream is often very grateful to the patient.

For the relief of pain opiates should be used, and if there be sleeplessness at night, sulphonal, trional, or other of the minor somnifacients.

CHRONIC TONSILLITIS.

Chronic inflammation of the tonsils is of especial frequency in feeble children, although not limited to them, and its results are particularly apparent at or about the age of puberty. The vulnerability of the tonsils may be inherited, and chronic tonsillitis is often seen in scrofulous children, in whom enlargement of the lymph-glands elsewhere is likely to be associated. Its usual exciting cause is a recurrence of attacks of acute tonsillitis or pharyngitis, whether of primary or of secondary origin. The inflammation affects the surface or the substance of one or both tonsils.

The symptoms of superficial chronic tonsillitis are those of chronic pharyngitis. The affection is characterized by the presence of opaque white plugs encircled by congested mucous membrane and projecting slightly from the surface of the tonsil. These form casts of the crypts in which they lie, are often of the size of grape-seed, and have a smooth, rounded surface and frequently a blunt-pointed end. They are extremely offensive when crushed, and are composed of degenerated cells, fat-crystals, bacteria, fat-drops, and perhaps starch-granules. They give rise to a tickling or pricking sensation, and eventually either are swallowed or are expelled by vigorous hawking or coughing. A gap—the distended crypt—remains, which soon becomes narrow. More rarely these cheesy plugs are long retained, become infiltrated with lime salts, and form the tonsillar calculus.

When the entire tonsil is inflamed, enlargement results, due to hyperplasia of the cells and increase of the fibrous tissue, the consistency of the tonsil being modified by the predominant increase of the one or the other element. The tonsil varies in size from that of a walnut to that of a pigeon's egg, is rounded and elongated, either pedunculate or with a broad base, and the surface is usually smooth, though sometimes irregular. Enlargement of the tonsil causes obstruction of the fauces, which becomes almost complete when both tonsils are enlarged. The speech is thick, the voice is nasal. There is but little disturbance in swallowing, and the breathing is only slightly affected unless the lymph-follicles of the pharynx are also enlarged. The latter condition, however, is frequently combined with enlargement of the tonsils, and the symptoms of chronic follicular pharyngitis are then associated with those due to chronic tonsillitis. Children with enlargement of the tonsils are subject to frequently recurring acute attacks of tonsillitis and pharnygitis, and, when exposed to diphtheria, become infected more readily than children with normal tonsils.

After the age of puberty both the enlarged tonsils and the hyperplastic pharyngeal follicles usually become diminished in size and cease to be productive of disturbance. The popular idea that the removal of the tonsil is a cause of atrophy of the testicle is erroneous.

TREATMENT.—In the treatment of chronic tonsillitis the indication is especially to reduce the size of the tonsil. The attempt at this by the application of powdered alum, by tincture of iodine, and by other local remedies is very rarely successful. The tonsil may, however, be slowly destroyed by caustic or by the application of the electro-cautery; though there is no reason for believing that better results are obtained by this slow and painful method than by the excision of the tonsil, an operation which, properly performed, is free from danger.

DISEASES OF THE ŒSOPHAGUS.

OBSTRUCTION.

ETIOLOGY.—Obstruction of the œsophagus is the result of congenital malformation, stricture, tumors of the wall, foreign bodies, compression from without, and muscular spasm. Congenital stenosis occurs more frequently at the upper end of the œsophagus than near the stomach; in the former situation it is attributed usually to arrest of development, while the cause of its occurrence in the latter region is unknown. There may be no union of the upper end of the primitive intestine with the lower end of the pharynx, in which case atresia of the œsophagus results; the communication between the œsophagus and the trachea sometimes existing at birth is due, probably, to a like irregularity of development.

Strictures of the œsophagus are of either inflammatory or malignant origin. The former result from the swallowing of corrosive fluids, injury from foreign bodies, the evacuation of periœsophageal abscesses or softened cheesy glands, or syphilis. Malignant disease, especially cancer, produces stricture by so infiltrating the wall as to prevent its distention, by the contraction of the fibrous tissue of the cancer, or by projecting into the canal. Obstruction of the œsophageal canal may result from the presence of pedunculate polypi and various impacted foreign bodies, including the rare accumulation of the *oïdium albicans* in thrush. Obstruction of the œsophageal canal also results from the pressure upon the œsophagus from without of tumors, distended œsophageal diverticula, periœsophageal abscesses, and aneurism of the aorta. In obstruction from muscular spasm the symptoms are not associated with any organic lesion.

SYMPTOMS.—The essential symptom of obstruction of the œsophagus is difficulty of swallowing, which is greater for solids than for fluids. One or several mouthfuls may enter the œsophagus before regurgitation takes place. This may be easy, or the efforts at expulsion in case of impacted solids may produce asphyxia, emphysema of the subcutaneous tissues, and even rupture of the œsophagus. The progress of stenosis of the œsophagus depends upon the nature and extent of the obstruction and the degree of compensatory hypertrophy.

DIAGNOSIS.—The diagnosis is based upon the history of gradually increased dysphagia, especially as indicated by the delay in the passage of food from the mouth to the stomach. This is manifested on physical examination by the retarded œsophageal gurgle, which, according to Meltzer, is normally to be heard at the left of the ensiform cartilage, or of the spine near the tenth rib, about six seconds after food or liquid is swallowed. The seat of the obstruction is to be determined by the use of the œsophageal sound or tube. The former consists of a long, flexible handle of whalebone or steel, to which an olive-shaped tip is securely fastened, or resembles in composition a magnified urethral bougie. The tube is essentially the same as that employed for the purpose of removing the contents of the stomach, is about as large round as the little finger, and has a rounded or blunt-pointed end, which is either solid or hollow. Before passing the sound or tube it is important to eliminate the possibility of an aortic aneurism as the cause of obstruction, and the existence of varicose veins from fibrous hepatitis, since immediately fatal or profuse hemorrhage may follow rupture of the aneurism or laceration of the veins. The attempt at passing the sound or tube may give rise to asphyxia or collapse in a person with cardiac disease, and be productive of extreme discomfort in a nervous person. In passing the sound the patient should sit upright, with the head slightly thrown backward, that the cavities of the mouth, pharynx, and œsophagus may lie as nearly in a straight line as possible. As the tip of the sound or tube touches the posterior pharyngeal wall, the patient should make an attempt to swallow and should be encouraged to breathe. Under ordinary circumstances the tube or sound readily passes downward in the œsophagus until the seat of the obstruction is reached. If spasm of the faucial and pharyngeal muscles resists the entrance of the tip of the sound, the application of cocaine will relieve the sensitiveness of the mucous membrane. The distance from the incisor teeth to the stomach is about seventeen inches, and the seat of the obstruction is to be determined, after the advance of the tube is stopped, by fixing the point at which it is in contact with the incisor teeth, and measuring the distance from this point to the tip of the tube after its withdrawal. The use of the œsophagoscope in the exploration of the œsophagus gives but little information additional to that which may be obtained by the tube or sound.

The nature of the cause of the obstruction is to be determined by the history of the case, by the associated symptoms, and perhaps by the removal of portions of a tumor by means of the sound or by their escape during vomiting. The Röntgen rays may aid in detecting and locating a foreign body in the œsophagus.

PROGNOSIS.—The prognosis depends upon the cause of the obstruction. Infants born with atresia of the œsophagus die in the course of a week, either from starvation or from pneumonia following the inhalation of food into the air-passages. Cicatricial strictures are grave in proportion to their density and the length of the wall involved. but are frequently capable of being so dilated that the patient can live in comfort.

The prognosis of malignant strictures of the œsophagus is especially considered on page 814. Obstruction from foreign bodies usually offers a favorable prognosis even when there is impaction, since repeated operations of late years have shown that impacted foreign bodies resisting attempts at removal through the mouth may be reached and removed through either an incision in the neck or an opening in the wall of the stomach. Stricture of the œsophagus from spasm, although often obstinate, is usually not directly injurious to life or health.

TREATMENT.—Organic stricture of the œsophagus is a surgical disorder, to be treated by mechanical means; if the case be not malignant, by methodical and gradual dilatation by means of bougies. A flexible bougie should always be employed, finished at the end with an olive-shaped ivory tip. It may be that at first only the catgut bougie can be passed through the narrowed channel; but even in such a case persistent very gradual and gentle dilatation will often finally bring about a brilliant result. For the method of using the sound, see Diagnosis. In many cases the best results are obtained by teaching the patient to pass the bougies himself. When there is ulceration or inflammatory softening of the œsophagus there is always danger of making a false passage, with serious and, it may be, rapidly fatal results.

The question of feeding is always dominant. If even the smallest tube can be got through the stricture, various liquid foods should be given at regular intervals. If it be impossible to nourish the patient in this way, nutritive enemata should be used. Œsophagotomy or gastrostomy may be performed for the purpose of sustaining life.

Spasmodic stricture of the œsophagus commonly occurs in hysterical subjects, and is chiefly to be met by the treatment of the underlying constitutional neurotic condition. Antipyrin, bromides, and similar remedies are used for temporary relief. When, as is frequently the case, there is great psychical impressionability, a cure may be wrought by psychical impressions, so that even a single passage of the œsophageal bougie may suffice for permanent relief.

In cancerous disease of the œsophagus mechanical treatment of the stricture is sometimes of benefit, but should be so practised as to avoid giving pain. Surgical interference has thus far not yielded good results, death being apparently inevitable.

DILATATION.

Dilatation of the œsophagus is either diffuse, *ectasis*, or circumscribed, *diverticulum*.

Ectasis is primary or secondary, the former being a rare condition due apparently to muscular weakness of unknown origin, and occurs both in infants and in adults. This variety of dilatation generally involves a considerable extent of the œsophagus, the tube often being increased in length and somewhat tortuous. The wall usually is thickened, but sometimes is abnormally thin. Secondary ectasis is the result of stenosis, and arises after the compensatory hypertrophy of the wall is replaced by a relaxed condition. The diffuse dilatation of the œsophagus is associated with retention and subsequent regurgitation of food, and the breath is offensive from the decomposition of the retained food. The passage of the sound meets with no obstruction, unless the canal is very tortuous, and the tip moves about with unusual freedom before reaching the stomach.

Diverticulum.—According to Zenker and Von Ziemssen, two varieties of diverticulum, the pulsion and the traction diverticulum, are to be found in the œsophagus. The *pulsion diverticulum* is regarded by these authors as a hernia of the mucous membrane through a weakened portion of the muscular coat, caused either by injury, ulcer, or scar, and promoted by the act of swallowing. In opposition to this view it has been said that the pulsion diverticulum has never been observed in diphtherial paralysis, in which there is extreme muscular weakness. It has been suggested that the pulsion diverticulum is of possibly congenital origin and represents the remains of a branchial cleft, and Fitz has offered evidence that it may represent a misplaced vitelline duct. In opposition to the theory of a congenital origin is the fact that this variety of diverticulum has never been found in the infant, and usually becomes apparent during middle life.

The pulsion diverticulum is single, and arises from the posterior wall of the œsophagus, near the level of the cricoid cartilage. It is either globular or cylindrical, and after long continuance may be pear-shaped, several inches in length, and of large capacity. The opening into the œsophagus is narrow or wide, and sometimes the canal of the diverticulum appears as the direct continuation of the œsophageal canal, the lower end of the gullet apparently starting from the side of the diverticulum. The wall of the diverticulum contains a few muscular fibres near its origin from the œsophagus, but for the most part is composed of mucous membrane and fibrous tissue. The lining membrane is often granular or warty in consequence of the irritation of retained and decomposed food, but ulceration is rare.

As the pulsion diverticulum becomes sufficiently enlarged from the retention of increasing quantities of food, a sense of local discomfort, perhaps associated with choking, and resulting in paroxysms of coughing, is occasioned by food first swallowed, but is relieved after more food is taken. Regurgitation eventually occurs, sometimes of food which has been retained for weeks. The contents of the diverticulum may be repeatedly regurgitated and returned before finally reaching the stomach. If the diverticulum is large, it may appear as a deep-seated tumor in the neck, the size of which varies from time to time and may be diminished by pressure. The passage of the œsophageal sound is often obstructed by its entrance into the sac, but if the opening into the latter is closed by one or more sounds, the passage of a sound or tube into the stomach readily takes place.

The disturbances produced by the diverticulum, as a rule, are persistent, and slowly increase in severity, the affection having proved a cause of death in about one-half of the recorded cases.

The *traction diverticulum* is caused for the most part by contraction of chronic inflammatory adhesions between inflamed lymph-glands and the œsophagus. The immediate causes of the lymphadenitis are tuberculosis, pleurisy, and caries of the rib or sternum. These diverticula may be multiple, short, funnel-shaped, and are to be found in that part of the œsophagus which is in the immediate vicinity of the tracheal bifurcation. Perforation of the œsophageal wall is likely to follow the retention and decomposition of food which lodges in the diverticulum.

Traction diverticulum causes little or no disturbance, unless sufficiently large and so shaped that food may be caught, retained, and decomposed, in which case ulceration and necrosis may follow, resulting in perforation of a bronchus with subsequent broncho-pneumonia and gangrene from inhalation of food. The periœsophageal inflammation is likely to extend to the pleura or the mediastinum, and a septic pleurisy or mediastinitis then results.

In diffuse dilatation of the œsophageal walls, as well as in the localized form of dilatation in which there are circumscribed pouches, no medical treatment is of any value. Feeding with the stomach-tube is necessary when the patient is not sufficiently nourished.

PERFORATION.

The œsophagus may be perforated from the mucous surface or from the outside of the wall. Perforation from within may be due to foreign bodies, which during their passage tear the œsophagus or become impacted and produce ulceration and necrosis of the wall. Caustic fluids also may produce a necrosis of the wall resulting in perforation, and ulcerating cancer may so progress as to perforate the œsophagus. Perforation from the outside of the œsophagus oftenest results from the rupture of an abscess in the vicinity, as suppurating lymph-glands or an acute or chronic retropharyngeal abscess. Aneurism of the aorta at times perforates the œsophageal wall, and, rarely, a communication has been established between the œsophagus and a cavity in the lung.

The results of perforation of the œsophagus vary according to the nature and seat of the lesion and the presence or absence of communication between the œsophagus and the lungs, pleura, pericardium, and mediastinum. In eighty-five cases collected by Zenker and Von Ziemssen the perforated œsophagus opened into the bronchi in twenty-six cases, into the lungs in twenty, into the trachea in twenty-one, into the pleural cavity in eleven, and into the pericardium in seven.

There may be no symptoms calling attention to perforation of the œsophagus, or a pricking or tearing sensation with coughing or swallowing may occur. Eventually the symptoms become those of septicæmia, the physical signs indicating a localization of the inflammation in the lung, pleura, or mediastinum, the pus, when present, being coughed up or vomited. If perforation of the trachea or a bronchus has occurred, food may be raised from the larynx. Perforation of an aneurism is shown by the sudden hemorrhage, which is usually considerable and immediately fatal, though sometimes it may be slight and protracted from the presence of a thrombus in the aneurism.

RUPTURE.

In a few instances the œsophagus has been ruptured by severe muscular efforts made in the attempt to dislodge a foreign body which had become impacted in the gullet. Mackenzie considered that the sudden pressure against the œsophageal wall induced by the attempt at expelling a large quantity of material from the stomach might cause rupture. Previous softening of the œsophageal wall, if present, would undoubtedly act as a favoring cause. The evidence which has been presented of a primary softening of the œsophagus is in the main unsatisfactory and largely based upon the confounding of post-mortem softening with an ante-mortem lesion. The rent is longitudinal, perhaps two inches long, and probably results from the pressure of the contents of the stomach against an impacted body of such a nature as completely to close the œsophagus. Emphysema of the tissues is likely to occur, probably from rupture of the alveolar wall during the violent efforts at expelling the impacted body. In consequence of the rupture, which usually takes place into the posterior mediastinum, food enters the tissues, gangrene results, and, if the patient lives sufficiently long, an ichorous pleurisy is likely to follow and cause death from a septicæmia.

TREATMENT.—Perforation or rupture of the œsophagus is so entirely without possibility of relief by medical means that, even with the total absence of guiding experience and statistics, surgical interference is justifiable.

ŒSOPHAGITIS.

Inflammation of the œsophagus is due to the admission of irritating material, whether foreign bodies, excessively hot liquids or solids, corrosive fluids, or frequent concentrated alcoholic drinks. Decomposition of retained food above a stricture or in a diverticulum also serves as a cause of inflammation. Œsophagitis may occur in the course of typhoid fever, scarlatina, variola, and tuberculosis or syphilis. Chronic passive congestion from valvular disease of the heart has been considered of etiological importance. In rare instances inflammation has been extended into the œsophagus from the pharynx or the stomach.

Morbid Anatomy.—Since the lining membrane of the œsophagus resembles the skin in structure, the appearances of œsophagitis are analogous to those of a dermatitis, and in variolous œsophagitis typical vesicles and pustules are to be found. The usual anatomical varieties of acute œsophagitis are the desquamative, catarrhal, follicular, fibrinous, diphtheritic, and phlegmonous inflammations. Desquamative or exfoliative œsophagitis, *œsophagitis dissecans superficialis*, is characterized by the detachment of more or less of the superficial portion of the membrane of the œsophagus as a cast of the tube. The detached portion is almost wholly composed of pavement epithelium. There are but few cases of this affection on record. We have seen it follow a dose of chloral given to an infant.

In *catarrhal œsophagitis* there is also a detachment of epidermis rather in the form of curd-like masses than as flakes. In chronic catarrhal œsophagitis the mucous membrane is thickened, and warts may project or polypi hang from the wall.

In *follicular œsophagitis* the sparse acinous glands are swollen, partly in consequence of retained secretion in the ducts and partly from the round-cell infiltration of the tissue in the vicinity. The *fibrinous* variety is characterized by the presence of a loosely adherent fibrinous membrane, and the *diphtheritic* variety by a necrosis of the superficial portion of the wall. Both of these varieties may occur in diphtheria, although it is rare for this disease to extend into the œsophagus.

In *phlegmonous œsophagitis* the submucous fibrous tissue becomes infected either from within or from without the œsophagus, and a diffuse suppurative inflammation takes place which may result in the more or less complete detachment of the mucous membrane. The pus is usually discharged into the œsophagus through one or more openings, but sometimes may find its way into the larynx or the trachea.

Most important, in consequence of its frequency and severity, is *corrosive œsophagitis*, under which term are included the alterations due to the action of caustic acids and alkalies. In the milder cases only the superficial portion of the mucous membrane is affected, and the epidermis is shrivelled. If concentrated solutions of alkalies have been swallowed in large quantities, the mucous membrane is softened and gelatinous; sulphuric acid causes it to become dry and black, and nitric acid makes it yellow. The action of the corrosive fluids may cause a complete destruction of the mucous membrane, and the exposed muscular coat is then shrivelled and traversed by a net-work of carbonized blood-vessels. If the patient live, the necrotic portions become surrounded by an inflammatory line of demarcation, and an injected zone infiltrated with leukocytes separates the necrotic from the normal tissue. At a later stage the necrotic portions are detached, and ulcers remain which lead to perforation or end in stricture.

Pain in swallowing is the first discomfort of an œsophagitis, but may be absent in the exfoliative variety. It may be so severe as to cause regurgitation or lead to the avoidance of food. In the milder variety of inflammation the pain lasts usually but a few days. In severe œsophagitis the pain is more intense, the patient is unable to swallow, and motion of the neck is often so painful that the spine is kept rigid. There is fever, perhaps associated with chills, and the patient becomes exhausted. The region of the cervical portion of the œsophagus may be tender to the touch, and in case of periœsophageal suppuration the swelling may appear at the base of the neck on a level with the last cervical vertebra. The abscess may cause displacement of the larynx or trachea, and dyspnœa and hoarseness be associated. The pus may be evacuated through the œsophagus or escape into the air-passages: in the latter case there is danger of suffocation or of broncho-pneumonia. In consequence of a communication between the œsophagus and the abscess, food may enter the latter and become decomposed, and gangrene of the tissues around the abscess result.

Chronic œsophagitis is characterized by a sense of persistent aching or constriction in the region of the œsophagus, and by the frequent regurgitation of a viscid glairy fluid of an alkaline reaction, and sometimes frothy, to which the term *water-brash* is applied, and which is to be distinguished from the acid fluid regurgitated from the stomach in pyrosis. The act of swallowing may demand distinct muscular effort.

Diagnosis.—Continued pain in swallowing, in connection with its method of origin, is the characteristic symptom of œsophagitis, the mild or severe nature of which is apparent from the associated symptoms. Exfoliative œsophagitis is diagnosticated by the ejection of detached portions of the epidermic layer of the lining membrane.

Prognosis.—Recovery from the milder varieties of acute œsophagitis readily and rapidly takes place. In the severer varieties of inflammation of the œsophagus, which are usually of corrosive or infectious origin, the prognosis is grave from the severity of the lesions and the tendency towards perforation, abscess, and gangrene. If recovery from acute symptoms takes place, the prognosis is that of fibrous stricture. Chronic catarrhal œsophagitis is resistant to treatment largely because of the persistence of its causes, and is liable to exacerbations and remissions.

Treatment.—There is no general medical treatment for œsophagitis. The food should be broths, milk and raw eggs, or other nutritious liquids, and in some cases must be given through the stomach-tube. Opium affords the only method of controlling extreme pain. Local remedies, such as bismuth subnitrate or solution of silver nitrate, may by swallowing be brought in contact with the œsophageal mucous membrane, but care must be exercised that no harm be done to the stomach by these remedies. When pus forms, surgical interference is justifiable, as in retropharyngeal abscess.

TUMORS OF THE ŒSOPHAGUS.

Fibroma, lipoma, myoma, sarcoma, and cancer are the varieties of tumors to be found in the œsophagus. The fibroma in rare instances attains a large size, becomes polypoid, and causes obstruction. It arises from the anterior wall of the œsophagus in the vicinity of the cricoid cartilage, and has been found only in elderly people. Retention cysts have been noticed repeatedly in chronic œsophagitis, and cysts sometimes occur near the œsophagus, their origin being attributed either to a persistent branchial fissure or to a detached, unusually high vitelline duct. Weigert has reported the occurrence of a polypoid adenoma which produced no symptoms.

CANCER OF THE ŒSOPHAGUS.

Cancer is the most common form of tumor of the gullet, and is the most frequent cause of œsophageal disturbance. Zenker and Von Ziemssen state that it was found in one-quarter of one per cent. of some five thousand autopsies.

Etiology.—Three-fourths of the cases occur in men, and four-fifths in persons between forty and sixty years of age, two-thirds of the patients being between the ages of fifty and sixty years. A certain etiological importance is to be attached to heredity and to local lesions, whether due to irregularity of development or to pathological processes.

Morbid Anatomy.—Primary cancer is the variety generally found, although the disease sometimes extends from the stomach, pharynx, or thyroid gland. It is of the epidermoid variety, and is usually found at the narrowest portions of the œsophagus, either near the cricoid cartilage, in the vicinity of the bifurcation of the trachea, or at the lower end of the œsophagus. Authorities differ as to the greater frequency of the disease at the upper or at the lower third of the œsophagus. According to Carmalt, the cancer begins in the deeper layers of the lining membrane, from which it extends in all directions. At first an elevated, rounded, and flattened nodule appears, gradually increasing in length and breadth, sometimes by the formation and fusion of accessory nodules, and eventually may encircle the œsophagus. Its extension in depth leads to the infiltration of the muscular coat, and the œsophagus is thereby transformed into an unyielding tube for a distance of one or two inches, sometimes throughout the length of the œsophagus. The growth also extends into the neighboring organs and tissues, especially into the trachea, bronchi, spinal canal, pleura, pericardium, and peritoneum. The bronchial glands are frequently infiltrated, and the disease may extend into the large arteries in the vicinity. The cancerous growth may compress or displace the recurrent laryngeal, especially the left, and the pneumogastric nerves. Secondary nodules at times are found in the brain, lungs, pancreas, liver, kidney, and adrenal glands.

As the growth extends towards the surface of the œsophagus it projects in the form of granules, papillary excrescences, or nodules. The central older portions become necrotic, are detached, and leave an irregular ulcer, which extends superficially and in depth, its edges often being everted. Particles of food frequently lodge in the crevices at the bottom of the ulcer, putrefy, and thus act as favoring causes in the extension of the ulceration, which eventually may result in perforation of the œsophagus and in the formation of a fistula between the trachea and a bronchus, usually the left. Since the tendency of the disease is towards the production of a stricture, that portion of the œsophagus which is above the cancer is usually dilated, the muscular coat hypertrophied, and the lining membrane in a condition of chronic catarrhal inflammation.

SYMPTOMS.—Cancer of the œsophagus usually pursues a latent course for a long time, and the first symptom of this disease may be a sudden and fatal hemorrhage from perforation of a large blood-vessel. As a rule, it is announced by difficulty in swallowing solid food, which may increase so rapidly as to necessitate within a short time a liquid diet. The greater the difficulty in swallowing the more likely is the regurgitation of materials swallowed, either unaltered in appearance or mixed with bloody slime or frothy fluid. The distress may be so great as to compel the patient to refrain from eating, and to lead to the symptoms of starvation. Periods of temporary relief to the dysphagia occasionally arise in consequence of the necrosis and detachment of projecting portions of the cancer producing the obstruction. The seat of the discomfort and obstruction is usually referred to the back of the neck, the interscapular, substernal, or epigastric region. Progressive loss of flesh and strength accompanies the dysphagia.

In the further progress of the disease, other symptoms arise in virtue of its extension to neighboring parts and the influence of putrefactive conditions in the cancerous ulcer. Pain is often but little complained of, although it is severe, constant, or paroxysmal when the intercostal nerves, the pericardium, or the pleuræ are involved. Cough is frequent from the extension of the disease to the larynx, or from perforation of the larynx, trachea, or bronchi. It is usually distressing, is either paroxysmal or constant, and is sometimes accompanied with profuse secretion. If food enters the lung, broncho-pneumonia rapidly follows. If the recurrent laryngeal nerve is involved in the growth, aphonia is the result, and a rapid, irregular action of the heart follows irritation of the pneumogastric nerve. When the growth enters the spinal canal, paralysis may result from pressure upon the spinal cord. Bleeding from the mouth follows injury to the smaller blood-vessels, while immediately fatal hemorrhage is the result of perforation of the aorta or of its larger branches. The symptoms of septicæmia or of septico-pyæmia complicate and accelerate the progress of cancer of the œsophagus when broncho-pneumonia, pleurisy, pericarditis, or peritonitis occurs. The broncho-pneumonia becomes gangrenous, and perforation into the pleural cavity leads to an ichorous pleurisy. The complicating pericarditis and peritonitis are likely to be rather serous or fibrino-serous, perhaps hemorrhagic, than ichorous or putrid. Death usually results gradually from starvation, rapidly from one of the causes producing septicæmia, or suddenly from the erosion of a large blood-vessel.

DIAGNOSIS.—The recognition of the obstruction of the œsophagus is easy from the dysphagia, the delay in the production of the second œsophageal murmur, and the use of the sound. The cancerous nature of the obstruction is to be inferred from the age and sex of the patient and from the appreciation of the fact that ninety per cent. of the cases of œsophageal obstruction after middle life are due to cancer. This diagnosis is supported by the recognition of a palpable tumor in the neck, from the limitation of the disease to the cervical portion of the œsophagus, or from an infiltration of the cervical glands. It is rendered certain by the microscopical examination of portions of the cancer which have been regurgitated or removed with the œsophageal tube. The possibility that syphilis may be a cause of the obstruction is to be remembered, and should be eliminated by active antisyphilitic treatment if necessary, unless a positive diagnosis of cancer can be made by the examination of portions of the growth ejected.

PROGNOSIS.—Cancer of the œsophagus proves fatal usually within ten to fifteen months after the beginning of the symptoms. The prognosis in the individual case varies within wide limits, owing to the inability to anticipate the occurrence of hemorrhage or of perforation into the respiratory tract or into the serous cavities. In these events death is likely to occur either immediately or in the course of a few days or weeks.

TREATMENT.—In the treatment of cancer of the œsophagus the indications are to relieve pain and nourish the patient. When solid food can no longer be swallowed, milk, eggs, gruels, broths, purées, may be taken in large quantities at long intervals, or in small quantities at short intervals, as seems best to suit the individual. When the power of swallowing is almost gone, the œsophageal tube may be used. The œsophagus above the stricture should be occasionally washed out by the swallowing and regurgitation of a weak solution of boric acid. Just before the œsophageal tube is to be passed the subject should swallow a small piece of frozen solution of cocaine, which may readily be made by means of the ethyl chloride spray. Rapid dilatation of the stricture by graduated sounds or laminaria tents does not seem to us to be a good practice, because it involves the danger of rupture. Authorities recommend carrying by means of the sound canulas (funnel-shaped above) into the stricture and leaving them there. Surgical interference when the stricture is low down is of very doubtful utility, the results of œsophagostomy or gastrostomy so far not having been good. When the cancer is in the upper part of the œsophagus, extirpation is practicable.

SPASM OF THE ŒSOPHAGUS. ŒSOPHAGISMUS.

A localized spasm of the muscular coat of the œsophagus occasionally takes place, either at the upper or at the lower end of the tube. This condition is called œsophagismus, or, from the cause and result, spastic stricture or stenosis. The condition is closely allied to the globus hystericus of the pharynx, and is seen for the most part in nervous women between the ages of twenty and forty years, although sometimes observed in old age and in childhood. Those suffering from overwork, mental shock, or chronic disease, especially of the pelvic organs, are more likely to be affected, and English writers consider that gout is a predisposing cause. Its occurrence has been observed during the period of gestation. It may be a symptom of organic disease of the œsophagus, as inflammation, ulcer, or cancer, and also of the central nervous system, especially in the vicinity of the medulla oblongata, as in rabies and meningitis. It is sometimes present in chorea, tetanus, and epilepsy.

The spasm is either slight or severe, temporary or prolonged, occasional or constant. Its frequency and severity usually increase in the course of time and become constant, although attacks of longer or shorter duration may occur with intervals of freedom perhaps for years. The efforts of the patient at swallowing cause distress, and the food may be violently regurgitated. The spasm is at times accompanied by hoarseness of the voice, difficulty of breathing, and hiccough. Hyperæsthesia of the pharynx is not infrequently associated, and may so act upon the œsophagus that, as in rabies, the mere presence of saliva induces the spasm, and even the thought of swallowing may bring on a paroxysm. Pain varying in character, and a feeling of constriction, usually referred to the sternum, the spine, or the shoulders, not infrequently accompany the spasm, and a sense of œsophageal fatigue referred to the same regions at times follows the more severe spasms.

The spastic nature of the obstruction is to be inferred from the intermitting, irregular course of the affection in women of nervous temperament, and an organic cause for the obstruction is excluded by the passage of the sound. Despite the obvious discomfort, the nutrition of the patient is, as a rule, well maintained. If the spasms are continued for a period of years, dilatation of the œsophagus may result, and chronic œsophagitis, perhaps ending in a fibrous stricture, occur.

The treatment of œsophagismus is that for spasmodic stricture of the œsophagus (page 806).

PARALYSIS OF THE ŒSOPHAGUS.

The œsophagus may become paralyzed in consequence of lesions of the central and peripheral nervous system, and in rare instances from hysteria. A central origin for the paralysis is to be found in intra-cranial tumors, hemorrhage, softening, or sclerosis, especially near the medulla and the pons. Central causes are to be found also in multiple sclerosis, tabes, chronic poliomyelitis, and general paralysis. Affections of the peripheral nerves are causes for the paralysis in diphtheria and in poisoning from lead or alcohol. Œsophageal paralysis may result from compression of the pneumogastric nerve by tuberculous or syphilitic disease of the adjoining lymph-glands and the vertebræ.

The paralysis is manifested by difficulty in swallowing, of sudden occurrence or of slowly increasing severity. Solid food in large pieces is more readily swallowed than liquid food or small fragments. If the passage downward of the first mouthful is prevented, the swallowing of more food may overcome the stoppage, and if liquids are taken to overcome the obstruction regurgitation is likely to follow. The paralysis causes a delay in the production of the second œsophageal murmur, and a sound or tube passes freely, but the tip does not move about as readily as is the case when the œsophagus is dilated, which latter condition is somewhat simulated by paralysis of the œsophagus. Since paralysis of the œsophagus is the result of a variety of lesions of greater or less severity, the prognosis depends upon that of the exciting cause. In toxic paralysis, whether from diphtheria, lead, or alcohol, the prognosis is favorable, as is the case also in hysterical paralysis, whereas this symptom is of grave importance in disease of the central nervous system.

Treatment.—The treatment of paralysis of the œsophagus is the treatment of its cause. If the patient is unable to swallow, feeding by the stomach-tube will sustain life.

CHAPTER II.

DISEASES OF THE STOMACH.

METHODS OF PHYSICAL EXAMINATION.

In the determination of the nature of the various diseases of the stomach it is of importance to know the position and size of this organ, the nature of its contents, the degree of its motility, and its power of absorption. These attributes are to be recognized by means of inspection, palpation, percussion, auscultation, the chemical and microscopical examination of the contents, and the use of certain drugs.

Inspection of the epigastrium may disclose the size, shape, and position of the stomach, especially when inflated with air or gas, preferably by the former, since its supply is under more immediate control. For the purpose of inflation the stomach-tube or an effervescing powder is to be introduced. The directions to be followed in the use of the tube and the precautions necessary are given in the article on the œsophagus, page 805. When the tip of the tube is in the stomach, air is readily forced through it by means of a bulb syringe connected with the end of the tube. In giving the effervescing powder one teaspoonful of tartaric acid is dissolved in a half-tumblerful of water and swallowed. Immediately afterwards one teaspoonful of sodium bicarbonate also dissolved in a half-tumblerful of water is to be taken, and the patient cautioned not to permit the gas which is formed to escape from the mouth. The progressing distention of the stomach is usually visible to the eye as a circumscribed bulging of the abdominal wall, either in the epigastrium or in the umbilical region or in both, according to the position of the stomach. An hour-glass shape has also thus been determined. In rare instances, in consequence of pyloric insufficiency, the air or gas passes from the stomach into the intestine, as may be observed from the resulting change of shape of the abdominal wall. Tumors of the stomach also are to be recognized at times on inspection, especially when near the pylorus, and, although moving but little with the descent of the diaphragm, are frequently displaced with the pylorus to distant parts of the abdomen. Inspection also reveals peristalsis, especially when exaggerated, in consequence of neurosis or from hypertrophy and dilatation of the stomach due to stricture of the pylorus. The wave usually extends from the left to the right, but sometimes in the reverse direction, and peristalsis is often to be excited by tapping upon the epigastrium or by inflation of the stomach.

Rosenheim has successfully inspected the interior of the stomach by means of the gastroscope, which is essentially an enlarged œsophagoscope. Its use, however, is inconvenient, requires experience, and adds but little to the information to be obtained by other means of investigation. Mention may be made also of the inspection of the illuminated stomach, *gastrodiaphany*. It is stated that by this method of examination the lower border of the normal empty stomach is to be found at the level of the navel considerably below the point at which its presence is to be determined by means of percussion.

Palpation of the region of the stomach is best performed when the patient lies on the back, with the head low and the knees raised. The pressure should be gentle, and made with the finger-tips and the ulnar edge of the nearly flattened hands. The object of the palpation is to obtain evidence of the size and position of the stomach, to recognize points of tenderness and localized resistance. Under normal circumstances only a small part of the stomach near the pylorus and in the vicinity of the greater curvature is perceived by the fingers, and then only when distended. The pylorus is sometimes to be felt as a circumscribed movable resistance at the right of the median line and on a level with the anterior end of the eighth costal cartilage. The lower border of the stomach is at times to be differentiated by the touch from the transverse colon, in part from the variation in the tension and in part from the resistance of the omentum. Tenderness is frequent in disease of the stomach, and is often the result of a circumscribed peritonitis, whether acute or chronic. This perigastritis is diffused or circumscribed, the latter being due often to the presence of ulcer of the stomach.

The possibility of recognizing the resistance of a normal pylorus has been already mentioned. The hypertrophied pylorus is more readily appreciated, and tumors in this part of the stomach frequently are felt. Important evidence is often to be obtained by palpation of the stomach under various conditions of distention, either by air, gas, or food.

Percussion is a most important means of determining the position and size of the stomach, especially by making evident the situation of the lower edge, and it may aid also in defining the position of a tumor of the wall. It is to be done when the stomach is empty, and when distended with air, gas, or other contents. A comparison is to be made also between the results to be obtained in the upright and those to be obtained in the supine position of the patient. Although in general the contrast between the stomach and the transverse colon is appreciable on slight percussion in consequence of difference in pitch, in cases of especial importance this difference may be emphasized by the distention of the one viscus with air and of the other with water before the outlines are percussed. According to Pacanowsky, the normal upper border of the stomach is near the fifth intercostal space in front and at the level of the seventh or eighth rib in the axillary region. The lower border may be found three centimetres above the navel. The vertical diameter is from ten to fourteen centimetres, and is longer in men than in women. By marking on the skin the limits of resonance the position of the vertical dilated or prolapsed stomach may be graphically shown.

Percussion may aid in determining the position of a tumor, the dulness from which will disappear on inflation of the stomach if the growth is from the posterior wall.

Auscultation is of chief importance in the recognition of splashing due to the presence of a mixture of air or gas and fluid, and produced by quickly tapping upon or by shaking the epigastric region. The splashing is often caused by a voluntary contraction of the abdominal muscles by the patient. This sound may occur normally after eating, but if present immediately before meals is evidence of retention of the contents of the stomach either from atony of the wall or from stricture of the pylorus. If the splashing is heard over an unusually wide area it offers evidence of dilatation, although it may be heard below the navel when there is simply prolapse of the stomach.

A gurgle originating in the stomach is due to the motion of air or gas alone, and is of no pathological significance. It is stated that a sound resembling that from a freshly opened bottle of liquid charged with carbonic acid gas may proceed from a dilated stomach in consequence of fermentation of its contents. The heart-sounds are to be heard distinctly on listening over a stomach distended with air or gas, and present a clear metallic character.

Examination of the Contents of the Stomach. — Important evidence of the condition of the stomach, especially with reference to modifications of its digestive power and its motor activity, is to be obtained by the examination of the contents. More accurate and complete knowledge is to be obtained when the contents are siphoned or expressed from the stomach through the tube at a definite time after a test meal has been taken, than from an examination of the vomitus. The meal recommended by Ewald and Boas consists of three or four hundred grammes of weak tea or water and thirty to forty grammes of white bread, practically a glass of the liquid and a roll of bread. The contents of the stomach are to be removed about an hour after this meal has been taken. That recommended by Leube and Riegel requires a longer time for digestion, and is to be removed four or five hours after being eaten. It consists of four hundred grammes of soup, one hundred and fifty to two hundred grammes of beefsteak, and fifty grammes of white bread or of mashed potato, essentially a plate of soup, a piece of steak, and a roll of bread.

The contents of the stomach may be altered in quantity and quality. If more is removed than is introduced it is evident that there is retention from pyloric obstruction or enfeebled motor power of the stomach, and if several pints are expressed or siphoned out the stomach must be dilated.

Variations in quality comprise abnormal odor, color, consistency, and chemical and microscopical characteristics. When the odor is suggestive of the presence of an acid, fermentation has probably taken place, as in the enfeebled or dilated stomach. If the odor resembles that of vinegar, it is due to acetic acid; if that of rancid butter, the presence of butyric or volatile fat acids is indicated. To distinguish between the two by chemical means is of no practical importance. A fæcal odor is present when there are an incompetent pylorus and a complete and prolonged obstruction of the bowels.

The expressed contents of the stomach are usually colorless, but may be green from the presence of bile, or red or black from that of blood. The consistency is homogeneous, or particles of undigested food, indicative of retarded digestion, are present. Abundant mucus occurs in catarrhal gastritis, and a frothy scum is due to fermentation, which exists only when there is retention of the contents.

The chemical examination of the siphoned or expressed contents after a test meal relates to the presence of acidity, which is caused by free hydrochloric acid, fixed hydrochloric acid, and acid salts or organic acids, to the presence of pepsin and other digestive ferments, and to the products of digestion, especially peptones. The chemical examination alone of the contents of the stomach is insufficient for an exact diagnosis. It represents merely one source of evidence, and its results are of value rather as affording indications for treatment than as characterizing the nature of the disturbance. Of greatest practical importance is the recognition of free hydrochloric acid and of lactic acid, and the abundant presence of the former excludes any considerable degree of abnormal fermentation, which is the source of the latter.

The reagent commonly employed for the recognition of free acid is Congo paper, which is prepared by soaking absorbent paper in a watery solution of Congo red, one of the coal-tar colors. The red paper when dipped in the gastric contents becomes blue from the presence of free acids, usually free hydrochloric acid, since lactic acid, according to Riegel, is unlikely to be found in sufficient quantity to cause the change in color. The test for free hydrochloric acid is that of Günzburg, and consists of two parts of phloroglucin, one part of vanillin, and thirty parts of absolute alcohol. The mixture is to be freshly prepared from time to time and kept in a dark-colored bottle. A few drops of the reagent are to be added to the same amount of filtered gastric contents spread on a porcelain dish, and the mixture is to be heated slowly over a small flame. As the fluid evaporates, a red color is formed if free hydrochloric acid is present, and the experienced eye is able usually to detect an increase or a diminution from the shade and amount of color. The absence of free hydrochloric acid is indicative of a deficiency of the digestive power of the gastric juice; its persistent absence is the rule in cancer of the stomach and in atrophic or degenerative conditions of the mucous membrane. The quantitative estimation of the total gastric acidity is made by dropping from a burette a decinormal solution of soda into ten cubic centimetres of filtered gastric contents to which three or four drops of a one per cent. alcoholic solution of phenolphthalein have been added. When the mixture, which is to be stirred constantly, is rendered alkaline, it assumes a homogeneous red color. The degree of total acidity is measured by the quantity of the soda solution required to render the gastric contents alkaline. Each cubic centimetre of the sodic hydrate necessary for this purpose corresponds to 0.003646 gramme of free hydrochloric acid. Normally from 4 to 6.5 cubic centimetres are used, representing a total acidity of 0.145 to 0.236, or for 100 cubic centimetres a percentage of 0.145 to 0.236. It is of no practical importance, however, to determine quantitatively the total acidity, since the digestive power of the gastric juice is dependent upon the presence of free hydrochloric acid.

The recognition of lactic acid is important, since this substance normally does not occur in appreciable quantities in the stomach unless it is a constituent of the food. When present it is the result of fermentation in the contents of the stomach, retained either from mechanical obstruction or from diminished motor activity. To avoid the introduction of lactic acid in the food, Boas recommends that before applying the test the stomach should be washed by means of the tube. A gruel made by the addition of a teaspoonful of oatmeal to a quart of water and flavored with salt is then to be taken, and an hour later the contents of the stomach are to be removed and tested for lactic acid. According to Rosenheim, however, there is not sufficient lactic acid in Ewald's test breakfast to form a source of error by giving a positive reaction by the test ordinarily used. The presence of lactic acid is to be determined by Uffelmann's test, which, although rough, is sufficient for practical purposes. Ten cubic centimetres of a four per cent. solution of carbolic acid are to be diluted with twenty cubic centimetres of distilled water, and one or two drops of the official liquor ferri chloridi are to be added, when a clear blue fluid is formed. The production of a greenish-yellow color when this is mixed with the filtered gastric contents indicates the presence of lactic acid. Riegel states that a like result follows the use of the fluid prepared by the addition of distilled water to a few drops of the ferric chloride solution until the latter is nearly colorless. The addition of a few drops of a two per cent. to four per cent. solution of carbolic acid will produce a blue-colored reagent, which is to be freshly prepared whenever used. The frequent presence of lactic acid in cancer of the stomach even before the appearance of a tumor is noteworthy. Boas found it present in twenty out of twenty-one cases of cancer, and Klemperer in twelve out of fifteen cases. Rosenheim found lactic acid present in seventy-eight per cent. of the cases of cancer of the stomach examined by him. In about one-fourth of the cases it appeared only at a late stage in the disease. The eventual presence, therefore, of lactic acid in the contents of the stomach is an important sign of this disease. It is not infallible, however, since cancer may occur when lactic acid is absent, and lactic acid may be present in the contents of the non-cancerous stomach. Indeed, Rosenheim has found free hydrochloric acid, even superacidity, in twenty-seven per cent. of forty-seven cases of cancer examined.

The digestive power of the gastric juice may be directly tested by the addition of a small piece of the white of a hard-boiled egg to the filtered gastric contents and keeping them at the temperature of the body. If the gastric juice is normal, the egg albumen disappears in the course of an hour and a half. If free hydrochloric acid is absent, pepsin is usually present, although the albumen remains undissolved, in which case a few drops of the acid should be added to the contents of a second tube and the trial again made. If the albumen remains undissolved, a deficiency of pepsin is indicated.

If further inquiry is desired, the presence of peptones may be determined by the biuret reaction,—that of starch by the blue color and that of dextrine by the red color following the addition of iodine.

The absorptive power of the stomach may be shown by means of potassium iodide, two grains of which enclosed in a thin gelatin capsule are to be swallowed. Normally the iodine can be recognized in the saliva by means of the starch test fifteen minutes after its ingestion.

The motile power of the stomach may be determined when there is no stricture of the pylorus by means of salol, which is normally decomposed and absorbed in the intestine an hour after it has been swallowed in a gelatin capsule. The time of its absorption is shown by the appearance of salicyluric acid in the urine, as indicated by the production of a violet color on the addition of a few drops of a neutral solution of ferric chloride. If this reaction is delayed for several hours, or persists after twenty-four hours, motor insufficiency is indicated.

The evidence to be obtained from the microscopical examination of the contents of the stomach relates to the presence of numerous undigested muscle-fibres as a sign of defective digestive power, of blood-corpuscles or blood-pigment as a suggestion or confirmation of the existence of ulcer or of cancer, and of sarcina or ferment fungus in proof of fermentation of the contents of the stomach.

MALPOSITION OF THE STOMACH.

The normal position of the stomach in the fœtus is distinctly vertical. This position is sometimes found in the adult, and the pyloric end of the stomach may lie below the navel, as the result of the persistence of the congenital condition, or in consequence of the application of pressure, as from corsets. The irregularity is of no clinical importance unless it causes the duodenum to become angular, in which case dilatation of the stomach from obstruction to the passage of its contents results. In rare cases the stomach is to be found in the right hypochondrium, the cardiac end being at the right and the pylorus at the left of the organ.

Most important of the malpositions of the stomach is *gastroptosis*, the downward displacement. When it exists the smaller curvature of the stomach may be midway between the ensiform cartilage and the navel, while the greater curvature lies between the navel and the symphysis pubis. This prolapse of the stomach is dependent upon elongation of the gastrohepatic omentum, either existing at birth or in consequence of traction, muscular strain, or injury. A lax condition of the abdominal wall in consequence of repeated pregnancies and lacing acts as a favoring cause. Prolapse of the stomach occurs oftener in women than in men, and is usually associated with prolapse of other abdominal organs, especially the kidney. The displaced stomach may be of normal size, but is frequently dilated. The functional activity of the stomach may be normal, but is usually altered in consequence of atony of the wall or because of the disturbances of secretion to be found in gastric neurosis. Owing to these variations in the condition of the stomach, the prolapsus may give rise to no symptoms, or the complex disturbances found in gastric atony, dilatation of the stomach, and nervous dyspepsia may be present. The diagnosis is to be made by the examination of the inflated stomach, the percussion of which gives evidence of its position. The prolapsed is to be distinguished from the dilated stomach by the fact that both the upper and the lower borders are displaced downward. Atony of the wall of the prolapsed stomach is indicated by splashing on succussion in addition to the presence of symptoms dependent upon the retention of the gastric contents. The symptoms attributable to the prolapsed stomach are relieved by the treatment appropriate for nervous dyspepsia, and the prognosis of the two affections is essentially the same.

DILATATION OF THE STOMACH. GASTRECTASIS.

DEFINITION.—Increase in the capacity of the stomach from enlargement of its cavity.

ETIOLOGY.—Dilatation of the stomach occurs at all ages, more commonly in adults, and in both sexes. It is the result of interference with the passage of its contents into the intestine, from their increased bulk, from mechanical obstruction, or from weakness of the wall. Overloading of the stomach occurs in gluttons and in the insane, and from the excessive drinking of beer or of water. Obstruction is usually at or near the pylorus, and may exist at birth as a congenital stenosis, or as an angular bend of the duodenum from a vertical stomach. Acquired obstruction is usually the result of a scar from chronic ulcer of the stomach or duodenum or from corrosive poisons. Tumors of the pylorus, either cancer or localized hypertrophy, and frequently recurring spasmodic contraction of the pylorus, are also regarded as causes of obstruction. Tumors compressing the pylorus or the duodenum, or dragging upon the latter, or perhaps the floating kidney, are of importance in etiology, and the compression may be occasioned by fibrous bands or scars from a localized peritonitis.

Weakness of the wall may be the result of chronic inflammatory changes extending to the muscular coat from the mucous membrane in gastric catarrh, or from the peritoneum in perigastritis. Degenerative changes in the muscular coat in acute or chronic disease also act as causes of weakness or enfeeblement, and atony of the muscular coat is considered to exist where there is dilatation without evidence of organic disease.

Dilatation is either acute or chronic in accordance with the temporary or the continuous action of the cause. Acute dilatation is rare, but the stomach may be largely dilated from paresis of the wall in the course of a day or two after the occurrence of acute intestinal obstruction or general peritonitis.

Morbid Anatomy.—The stomach may be so distended as to fill the entire front of the abdomen, the greater curvature lying at the brim of the pelvis, and its capacity may be increased from the normal three pints to thirty pints. In acute dilatation the wall is stretched and thin; in chronic dilatation, especially from pyloric obstruction, compensatory hypertrophy may exist for a long time, and the wall of the stomach be found thickened. In chronic dilatation the mucous membrane usually presents the changes characteristic of chronic gastritis, and degenerative changes are likely to be found in the muscular coat.

Symptoms.—The appetite varies, but is usually feeble. There is frequent belching of odorless or offensive gas. Pyrosis may occur soon after eating, or take place several hours after food has been taken. The hot fluid regurgitated immediately after eating is irritating in consequence of the presence of acids from fermentation, while the acridity of that regurgitated later in the digestive process is often attributable to free hydrochloric acid. There is a sensation of fulness or distress in the epigastrium even after eating but little food, and nausea and vomiting are of frequent occurrence. Large quantities, perhaps three or four quarts, may be vomited even before breakfast, and may contain particles of undigested food, especially the skins and seeds of fruit eaten days or weeks previously. The salol test will show impaired motility, and the use of potassium iodide will indicate defective absorption. If the vomit is kept for a while in a suitable receptacle, a frothy layer forms on the surface, below which is a thin grayish-brown fluid, and at the bottom are particles of undigested food. Bubbles of gas are at times to be seen rising from the bottom of the dish.

In consequence of the prolonged retention and deficient absorption of the contents of the stomach, the bowels are constipated, the urine is scanty, and there is marked thirst. The general nutrition is usually lessened, the skin is dry and rough, and there may be extreme emaciation. Disturbances of the nervous system are at times complained of, such as headache and dizziness. These and the less frequent cramps, delirium, and coma are usually attributed to the absorption from the stomach of the products of the fermentation of its contents. In rare instances tetany has been observed, and has proved a cause of death.

On physical examination the abdomen is found distended either in the epigastrium or in the vicinity of the navel, or in both regions, and peristalsis is visible during the existence of compensatory hypertrophy, especially when the abdomen is palpated. On percussion the area of gastric resonance is markedly increased, and a change in the resonant area may result from an alteration in position of the patient. The increased area of resonance is most satisfactorily determined after inflation of the previously emptied stomach. On auscultation a splashing sound produced by palpation or succussion is to be heard over a wide area. The chemical examination of the contents of the stomach, according to the cause and persistence of the dilatation, shows every variation in the quantity of free hydrochloric acid, but more frequently in the protracted cases there is no free hydrochloric acid, but lactic, butyric, or acetic acid, carbonic acid gas, hydrogen, sulphuretted hydrogen, and even phosphoretted hydrogen, as shown by Ewald, may be present. On microscopical examination sarcina and various fungi are to be found. The urine is often alkaline, and contains an increase of phosphates.

DIAGNOSIS.—The increased area of tympany produced by the inflated stomach, splashing, and the possibility of removal by the tube from the stomach before breakfast of a considerable amount of contents, are characteristic of dilatation of the stomach. Moderate degrees of dilatation are easily confounded with simple atony or atony with prolapse of the stomach. In simple atony there may be temporary enlargement of the stomach, but the amount of contents is usually relatively normal, and the disturbance of nutrition is often inconsiderable. In dilatation enlargement of the stomach is permanent, the contents are indicative of fermentation, and emaciation is the rule. The upper border of the inflated prolapsed stomach lies between the ensiform cartilage and the navel, whilst that of the dilated stomach is to be found in the immediate vicinity of the apex of the heart. A mechanical cause of the dilatation is suggested by the previous history of ulcer, a palpable tumor at the pyloric end of the stomach, visible peristalsis, or extreme emaciation. Atony is suggested as a cause of dilatation by the previous history of the patient and by the absence of physical signs suggestive of a mechanical dilatation. Although the dilated stomach has been so large as to indicate an ovarian cyst and has been tapped, the use of the stomach-tube would render this operation or an exploratory laparotomy unnecessary for diagnosis.

PROGNOSIS.—The prognosis of dilatation of the stomach is based chiefly upon the cause and the duration. It is unfavorable in malignant disease, grave in case of fibrous stricture, and favorable when the dilatation is the result of atony. The greater the degree of dilatation and the longer its persistence the more unfavorable its prognosis, whatever may be the cause, since insuperable atrophy of the mucous membrane may result. The surgical treatment of dilatation of the stomach from mechanical causes, especially when performed before the dilatation has reached an extreme degree, renders the prognosis more favorable in case of fibrous stricture, and makes life more endurable and even prolonged in case of cancer.

TREATMENT.—The treatment of dilatation of the stomach, apart from the removal of the cause, consists in little more than washing out the stomach and careful regulation of the diet,—procedures which in many cases bring about a good result by relieving the basal catarrhal condition, and which when the cause of the dilatation is irremediable do good by removal of the fermenting mass of food and secretions which accumulate because they cannot escape through the pylorus.

The introduction of gastric lavage in 1867 by Kussmaul marked an era in the treatment of gastric diseases. In washing out the stomach it is better to remove the contents by siphonage than by the stomach-pump. The apparatus required is very simple, consisting of a long, soft stomach-tube or Nélaton's catheter, with sufficiently large side openings near the open or closed end, and united above to an india-rubber tube about a yard in length and terminating in a large funnel. After the stomach-tube has been introduced, the funnel filled with water is raised above the head of the patient, and when it has nearly emptied itself is lowered below the stomach of the patient, so as to produce a reversal of the current. The process is so easy that it is usually better to teach the patient to do it himself. At first it may be better to wash out the stomach two or three times a day, but when the viscus has once been thoroughly cleansed lavage should be usually practised only once a day, preferably at a time distant from a meal, as in the early morning or in the late evening. Pure water of about 100° F. may be commonly used; but when there is much fermentation a two per cent. solution of boric acid, or a one per cent. solution of salicylic acid or of resorcin, may be employed.

The feeding of a case of dilatation of the stomach should be that of severe chronic gastric catarrh, the food consisting chiefly or entirely of scraped beef, predigested foods, or cooked meat, which should always be very tender and thoroughly masticated. Milk diet also may be tried. It should be given at short intervals.

The power of contracting the muscular coat of the stomach has been assigned by various practitioners to certain remedies, notably strychnine and electricity. The alkaloid may be freely administered internally and the faradic current used locally, though the effect is probably very slight. When the dilatation is pronounced an elastic abdominal bandage is often serviceable.

For the treatment of pyloric stenosis three surgical procedures are in vogue. Loreta's operation consists in digital dilatation after gastrotomy; it is applicable only to non-malignant cases; according to Schroeter, it had up to 1894 a mortality-rate of about forty-six per cent. Moreover, in the greater proportion of successful cases there has been a recurrence of the stenosis.

Gastro-enterostomy consists in making an opening between the stomach and the intestines and uniting the two. According to Magill, in sixty-one gastro-enterostomies, made by the aid of plates or other mechanical devices (not sutures) between 1887 and 1894, the gross mortality was about twenty-three per cent.; the mortality with sutures seems to have been about fifty per cent.

According to Dreydorff, the mortality of pylorectomies up to 1894 was about seventy-five per cent.; but an improving technique seems to be reducing this greatly, as in nine cases Kocher had only two deaths. (See also Prognosis.)

GASTRITIS.

Definition.—Inflammation of the stomach.

Inflammation of the stomach usually results from a local irritation of its mucous membrane, and the resulting changes are both superficial and deep-seated, affecting the epithelium of the surface, the glands, and the interstitial tissue. In severe cases the inflammatory process extends towards the peritoneal coat of the stomach, which it sooner or later reaches, the intervening structures being involved to a greater or less extent. The distinction of clinical convenience is that between acute and chronic gastritis. Acute gastritis is either catarrhal, pseudo-membranous, or phlegmonous according to the anatomical changes, or toxic, mycotic, or parasitic according to the conspicuous features in etiology. Chronic gastritis is largely catarrhal, but is sometimes associated with proliferation or atrophy of the mucous membrane, and at other times with sclerosis of the wall of the stomach.

ACUTE CATARRHAL GASTRITIS. ACUTE GASTRIC CATARRH. ACUTE DYSPEPSIA.

Etiology.—Local irritation from food or drink is the usual exciting cause of acute catarrhal gastritis. The contents of the stomach when swallowed may be too hot or too cold, improperly prepared, or excessive in quantity. Of especial importance are fermented or decomposed food, as putrid meat or fish, sour milk, unripe or rotten fruit, ill-kept fermented liquors, and alcoholic excess. The bacterial infection of the food or drink is of especial importance, and epidemics of infectious gastritis have been reported in which a number of persons have simultaneously suffered from partaking in common of infected food or drink. Such instances of infectious gastritis are not to be confounded with the usual occurrence of acute gastritis in acute infectious diseases.

Predisposing causes are also important, since all exposed to the same exciting cause do not alike suffer. A vulnerable condition of the stomach prevails in certain families, and may be inherited. It is especially frequent among brunettes, so often characterized as bilious, in the very old, and in the very young, and may also result from bad hygienic surroundings and acute or chronic disease.

MORBID ANATOMY.—The appearances of the mucous membrane of the stomach in acute catarrhal gastritis are rarely observed, since the disease is rapidly recovered from. Our knowledge of them is based chiefly upon the direct observation by Beaumont of St. Martin's stomach, and is confirmed in rare cases of death from other causes during an acute catarrhal gastritis and by experiment. The mucous membrane is swollen, red, and either dry or covered with abundant viscid mucus. Minute hemorrhages may be observed, and superficial erosions are to be seen, especially along the projecting folds of the contracted stomach. Microscopical examination shows a granular condition of the glandular epithelium, sometimes associated with a cellular infiltration of the interstitial tissue. When the glands are decidedly swollen and granular the mucous membrane of the stomach is opaque gray, especially at the pyloric end, and the condition has been designated *parenchymatous gastro-adenitis.*

SYMPTOMS.—The symptoms vary in accordance with the mildness or the severity of the attack. There is a loss of appetite, or a desire for pungent, sour, saline, very hot or ice-cold articles of food or drink. Thirst is usually conspicuous, although the patient may be loath to swallow through fear of vomiting, and there is an unpleasant taste in the mouth. Nausea, belching, and vomiting are more or less constant, and hiccough is occasional. The regurgitation of a tasteless fluid, *water-brash*, or of an acrid, burning fluid, *pyrosis*, is frequent. There is more or less discomfort, sometimes intense pain, in the epigastric region, to which a faint, "all-gone" feeling is often referred, and constipation is the rule, but is sometimes followed by diarrhœa.

The patient complains of headache, is dull and sleepy or fretful, especially if a child, and in the infant delirium or convulsions may occur, suggesting a meningitis. In the mild cases there is little or no elevation of temperature, but in the severe cases the temperature may rise to 101° or 102° F., and the beginning of the attack be announced by a chill. The tongue is usually covered by a white coat, and the breath is offensive from the associated catarrhal stomatitis. Herpes of the lips is occasionally observed. The epigastrium is often distended, tympanitic, and tender. The vomit consists at first of undigested food, which may have been retained for many hours. With the persistence of the vomiting the contents of the stomach are either alkaline from deficient hydrochloric acid and abundant alkaline mucus, or acid from the presence of lactic acid or butyric acid in consequence of fermentation, or bitter from peptones or bile. The urine is scanty and high-colored.

The causes of a gastritis are often those of a gastro-enteritis, and the association of jaundice with the above-mentioned symptoms of catarrhal gastritis is indicative of extension of the inflammation to the duodenum. This gastro-duodenal catarrh is the usual cause of acute jaundice, bilious attacks, or "acute hepatic torpor." It is also to be remembered that acute pancreatitis is frequently preceded by the symptoms of a gastro-duodenitis. The advance of the irritant, perhaps a specific bacterium, to the ileum is manifested by profuse diarrhœa, which, combined with gastric symptoms and collapse, is significant of cholera, cholera nostras, or poisoning by putrid meat, fish, or milk products.

DIAGNOSIS.—Mild cases of acute catarrhal gastritis are readily diagnosticated from the immediate occurrence of the symptoms after indigestible food or other irritants have been taken into the stomach. The diagnosis becomes difficult in acute febrile gastritis of obscure etiology, since the condition may be due to the invasion of an acute infectious disease. The absence of characteristic symptoms in the course of a few days serves to eliminate the exanthemata. The early symptoms of typhoid fever often resemble those of acute catarrhal gastritis, but in the course of time the typical range of temperature, the enlargement of the spleen, the presence of a rash, and the diazo-reaction make clear the existence of typhoid fever.

PROGNOSIS.—Acute catarrhal gastritis is usually a mild affection, lasting from a few days to a fortnight. Rapid recovery often follows the vomiting of the irritating cause, or its expulsion from the bowels when diarrhœa is a complication. Gastritis complicated with duodenitis may last for weeks, and the prognosis of gastro-enteritis is stated in the consideration of acute enteritis. Frequently recurring attacks of acute catarrhal gastritis are likely to result in chronic catarrhal gastritis.

PSEUDO-MEMBRANOUS GASTRITIS.

This variety of acute gastritis is characterized by the presence of large or small patches of a membrane either loosely applied or intimately adherent to the mucous membrane of the stomach. The condition is rare. and occurs as a complication of severe infectious diseases, especially diphtheria, scarlet fever, small-pox, septicæmia, malignant endocarditis, and pneumonia. There are no distinctive symptoms unless portions of membrane be vomited.

PHLEGMONOUS GASTRITIS.

In rare instances a circumscribed or diffuse purulent infiltration of the submucous tissue of the stomach occurs. Single or multiple abscesses result, the former having been observed as large as the fist, and are discharged into the stomach or perforate the peritoneal coat. Phlegmonous gastritis has been found oftener in men than in women, more frequently in drunkards, and occurs without obvious exciting cause, or as a complication of ulceration of the stomach or in the course of a septic process, as puerperal infection or malignant endocarditis.

The onset is usually sudden, and is characterized by severe epigastric pain, high fever, with morning fall and evening rise of temperature, typhoidal symptoms, and eventual coma or collapse. The abscess has been felt as a tumor through the abdominal wall, and pus has appeared in the vomit and the dejections. The course of phlegmonous gastritis is usually acute, though sometimes chronic. The diagnosis rarely has been made, and the condition is most to be suspected when the symptoms of an acute perigastritis without obvious cause are present. The prognosis is unfavorable, death resulting from an extension of the suppuration to the peritoneum with the production of a general peritonitis, or in consequence of progressive emaciation and debility from the resulting destruction and associated disturbance of function of the mucous membrane of the stomach. In rare cases healing, with extensive deformity of the stomach from scars, may take place.

TOXIC GASTRITIS.

This variety of inflammation of the stomach is due to the introduction of poisonous chemicals, especially sulphuric, nitric, oxalic, and carbolic acids, caustic alkalies, phosphorus, arsenic, antimony, corrosive sublimate, and potassium cyanide. The resulting lesions resemble those described in connection with corrosive inflammation of the œsophagus. Phosphorus, arsenic, and antimony are likely to produce extensive parenchymatous degeneration of the glands of the stomach. The symptoms indicative of the entrance of the poisons into the stomach are localized epigastric pain, persistent vomiting of blood, and a swollen and tender epigastrium. The effect of acute poisoning from irritants swallowed is considered in the chapter on Poisoning, pages 360–362.

When gastritis is due to the growth of fungi in the stomach, as the favus fungus, or to that of anthrax bacillus in the wall, the condition is known as a mycotic gastritis. The larvæ of certain insects and intestinal parasites when present in the stomach may be productive of a gastritis to which the term parasitic gastritis has been applied. Further information on this subject is to be found in the chapter on Diseases due to Animal Parasites, page 321.

TREATMENT.—The treatment of acute gastritis varies with the causation. In the simplest cases all that is necessary is to restrict the food to milk with lime water and broths, and give a saline purge: such treatment being preceded by an emetic if the stomach contain undigested food. In severe cases it may be necessary to leech, to give aconite with antipyrin if there be fever, and to purge with a mercurial followed by salines.

When an acute gastritis is due to an irritant poison, the stomach after having been well washed out should be left for twenty-four hours or longer, according to the severity of the case, without food; leeches should be placed upon the epigastrium, followed, if necessary, by blisters; opium should be given freely, and bismuth subnitrate or subcarbonate carefully administered in small repeated doses. When the acute catarrh is the outcome of an alcoholic debauch, the patient should be well vomited by ipecacuanha given in five-grain doses repeated every ten minutes until the effect is produced, and afterwards should be freely purged by quarter-grain doses of calomel administered every hour, aided by a saline if necessary. The food should be broths, milk, or similar bland liquids, in small quantities, or should for the time being be entirely withheld from the stomach, the patient being sustained by nutritive enemata. Leeches to the epigastrium are rarely required, but the blister is often of great service. The use of stomachic bitters and similar irritant substances is strongly contra-indicated in gastric catarrh, except that in alcoholic subjects after the first few days hydrastine or fluid extract of hydrastine is often a very useful remedy; and that when the so-called acute catarrh of an alcoholic is really an exacerbation of a chronic gastritis with great relaxation of the vessels and benumbing of the gastric nerve-endings, local stimulants, such as tincture of Cayenne pepper and compound tincture of gentian, are often of service after the first few days of treatment.

CHRONIC GASTRITIS. CHRONIC CATARRHAL GASTRITIS. CHRONIC GASTRIC CATARRH. CHRONIC DYSPEPSIA.

Etiology.—The prolonged or frequently recurrent action of the causes of acute gastritis is an important factor in the etiology of chronic gastritis. Prominent among these causes are the long-continued use of unsuitable or improperly prepared articles of food, persistently irregular or hurried meals, insufficient mastication, and the abuse of tea, coffee, tobacco, and especially of alcohol. Chronic gastritis is a frequent accompaniment of cancer and dilatation of the stomach, and is sometimes associated with ulcer. It may follow chronic passive congestion of the mucous membrane not only from obstruction of the portal circulation, but also from chronic affections of the heart and lungs, and is of frequent occurrence in chronic tuberculosis, nephritis, gout, diabetes, and prolonged primary and secondary anæmias.

Morbid Anatomy.—In simple chronic catarrhal gastritis the mucous membrane is swollen, injected, of a bluish slate color from the presence of metamorphosed blood-pigment, and is covered with abundant grayish-white mucus. These alterations are seen especially in the chronic catarrhal gastritis due to passive congestion. The severe forms of chronic gastritis are those in which more conspicuous structural changes take place in the wall of the stomach. According to the nature and effect of these changes a distinction is drawn between hypertrophic or proliferating gastritis and atrophic gastritis. In *hypertrophic gastritis* there is a cellular infiltration of the mucous membrane of the stomach associated with enlargement of the glands, which become tortuous and even hyperplastic. Polypoid projections of the mucous membrane may be formed, and a lobulated or corrugated—the mammillated—condition of the surface results, in part from the contraction of the inflamed wall, in part from the abnormal growth of the interglandular tissue. The orifices of the glands are frequently constricted, causing dilatation of the ducts and the formation of cysts. The capacity of the stomach may be normal or somewhat increased. In *atrophic gastritis* a shrinkage of the inflamed mucous membrane occurs, in consequence of which it becomes thin, smooth, and dense, suggestive rather of a thickened serous membrane than of a mucous membrane, and an extreme degree of atrophy of the glands ensues. Sclerosis or cirrhosis of the stomach is the result of an involvement of all the coats of the stomach in the inflammatory process. In consequence of the shrinkage of the fibrous tissue the stomach may be so diminished in size as to hold but a few ounces. The wall is increased in thickness and in density. The gross appearances closely resemble those of fibrous cancer, so-called scirrhus, and a microscopical examination is often necessary to exclude the existence of this disease.

SYMPTOMS.—The local and general symptoms of chronic catarrhal gastritis resemble those of other affections of the stomach, especially acute catarrhal gastritis. They are, however, usually of gradual onset, at first being only occasional, but eventually are persistent. There is frequently a disagreeable taste in the mouth, which may be moist or dry, and the appetite is feeble, often perverse, and sometimes excessive. There is occasional thirst, especially when the mouth is dry, although often there is a profuse secretion of saliva. Nausea is frequent both before and after eating, and vomiting is of occasional occurrence. In alcoholic gastritis the vomiting of mucus before breakfast is conspicuous, and is attributable to the retching caused by efforts at clearing the pharynx from the abundant adherent secretion. Vomiting also takes place after food has been taken, and then consists of incompletely digested particles of food, which may be covered with mucus. Belching during and after meals is frequent, the gas raised being either odorless or offensive. Regurgitation of liquid, either bitter from the presence of peptones, or acrid from free hydrochloric acid or organic acids from fermentation, is a source of discomfort. The patient complains of a faint, "all-gone" feeling in the epigastrium before eating, but food usually produces a sensation of fulness, of weight, as from a piece of lead, or of tension, demanding relief from external pressure. There may be actual pain, sometimes severe, especially after eating, referred to the region of the stomach. The bowels are usually constipated, and flatulence is frequent. The evacuations sometimes contain undigested particles of food.

The patient complains of headache and of dizziness, particularly before eating. The action of the heart is often irregular, notably when the stomach is empty, and attacks of palpitation or dyspnœa may occur from apparently slight indiscretions of diet. The patient is frequently in a state of mental depression, or is nervous and irritable. He is often drowsy after eating, and wakeful at night. Although for a long time the general nutrition may be but little impaired, eventually emaciation, sometimes extreme, results. Extreme pallor and symptoms of a progressive pernicious anæmia have been observed in chronic gastritis with extensive atrophy of the mucous membrane.

The tongue may be somewhat coated, though not infrequently it appears normal. The breath is often offensive, either from an unclean condition of the mouth and teeth or from the escape of gases from the stomach. The epigastrium is frequently swollen and tender, and splashing may be heard on palpation at a time when the normal stomach should be empty. The contents of the stomach removed at a suitable time after a test meal has been taken may be increased in quantity and contain undigested food from impaired motility and defective digestive power. There is usually a diminution of hydrochloric acid, in which case lactic, acetic, or butyric acid is present. It is stated, on the contrary, that in proliferating gastritis there may be an excess of hydrochloric acid. There is abundant mucus in simple catarrhal gastritis, while there is little or no mucus in the contents of the stomach in atrophic gastritis. In the latter affection in addition to diminished or absent hydrochloric acid there is a marked lack of the digestive ferments. The urine is scanty, and either is high-colored with abundant urates or is pale and contains phosphates; crystals of calcic oxalate are sometimes observed.

PROGNOSIS.—Periods of temporary improvement during the course of chronic catarrhal gastritis are frequent, but recurrences of the symptoms are the rule. Complete recovery in primary gastritis is the less likely to occur the longer the condition has existed, because of the organic changes developing in the wall of the stomach. In gastritis secondary to grave disease the prognosis depends upon the conditions causing the gastric changes. The immediate outlook is extremely grave when the chemical examination of the stomach shows persistent absence of the digestive ferments.

DIAGNOSIS.—Important in diagnosis is a well-defined cause, the persistence of the symptoms, a constant lack of free hydrochloric acid, the presence of abundant mucus in the contents of the stomach, and evidence of deficient motility and absorptive power. Boas, however, has observed a series of cases characterized by the copious secretion of mucus and excessive acidity and accompanied by marked pain in the region of the stomach, to which he has applied the term *acid gastritis.* The diagnosis of pure chronic gastritis requires also the elimination of other affections of the stomach in which similar modifications of function may be present. Especially to be differentiated are nervous dyspepsia, dilatation, and cancer of the stomach. Even ulcer of the stomach, when pain is slight and hemorrhage lacking, may be regarded as chronic gastritis.

Nervous dyspepsia is to be eliminated by the existence of a satisfactory local cause for the gastric symptoms, by the greater or less uniformity of relation between definite kinds of diet and the occasioned disturbance, and by the freedom from various neuroses. Dilatation of the stomach is to be excluded by the results of the examination with the tube. In cancer the cachexia is greater and usually progressive despite treatment, lactic acid is generally found in the contents of the stomach, and, as a rule, a tumor eventually is appreciable. In ulcer of the stomach, even if there is no characteristic hemorrhage, localized tenderness is likely to be marked, and the gastric pain is commonly relieved by alkalies and by certain foods. The differential diagnosis is further considered in the articles on these several diseases.

TREATMENT.—The successful treatment of chronic gastric catarrh requires the most careful attention to minutiæ and watchfulness on the part of the physician to see that directions are closely carried out. A woollen or silk abdominal bandage to be worn day and night is in many cases essential. The diet must be regulated with the greatest strictness and care, an absolute diet-list being given to the patient. In the selection of the diet not only must the general rules be adhered to, but the individual peculiarities of the patient must be carefully studied, so that the diet-list shall conform not only to the needs of the gastric catarrh, but also to those of the individual suffering from the catarrh. The personal experiences of the patient are to be carefully weighed. In severe cases it may be necessary to commence the course of treatment with a rigid milk diet; there are many persons who assert that milk does not agree with them, there are a few who really cannot digest it; upon the latter it should not be forced. The milk may be warm, but never boiled. Ordinarily milk at the temperature of the room is to be preferred; ice-cold milk should never be allowed. It should be swallowed in small quantities, five to six ounces, every two hours during the day, and should have added to each portion one to two tablespoonfuls of lime water. It should be taken slowly, in draughts of not more than an ounce each, at intervals of a minute, or longer. It should not be too rich, and in some cases should be partially skimmed. Separator milk should never be used. The length of time during which a patient may be kept on a milk diet without injury is indefinite; from two to four quarts of milk a day are necessary to support the bodily functions.

Probably next to milk, and perhaps superior to it, in digestibility, is scraped beef given raw; it is, however, so disagreeable to patients that immediately following the milk diet, or in mild cases without a preceding milk diet, the practitioner frequently must content himself with restricting the food to Hamburg steak (made without egg and broiled lightly over a very quick fire), stewed sweetbread, white meat of chicken or game, and pulled bread or toast. Custards made with but little sugar or sweetened with saccharin, and junket, may be used for desserts. Milk-toast agrees well with some. Game may be allowed, but tame ducks, turkey, and squabs or young pigeons are to be interdicted. Among vegetables, thoroughly cooked spinach, macaroni stewed in milk, and rice, are the first to be given; in many cases certain farinaceous foods may be taken early, —never oatmeal, however. Wheat preparations are usually to be preferred, though some of the fine Indian-corn preparations are useful. The hull of the grain must be largely removed, and the farinaceous food must be thoroughly cooked. A principle of absolute importance is that not more than three or four different foods should be taken at one meal. As the case progresses, or the patient becomes utterly wearied, the diet may be extended by the addition of fresh young vegetables. Too long continuance of any diet may lead to loss of appetite with weakness and anæmia.

All vegetables that are eaten green, such as peas, should be very young and soft; ripe peas and beans are among the most indigestible of farinaceous foods. Potatoes are among the last articles to be allowed; they should always be roasted and mealy. Sugar is distinctly worse than potatoes. Artificial foods, peptones, etc., much used as they are, are, in our belief, not to be looked upon with favor, as it is impossible for any one to know what a pharmaceutical food really contains. The food must be thoroughly masticated.

The question of drink is an important one. In the first place, alcohol is an irritant to the stomach, and in chronic inflammation of this organ can only do harm, so that total abstinence must be enforced. In the second place, an excess of fluid with food dilutes the gastric juice, whilst any cold drink or food put into a diseased stomach during a meal interferes with its functions. Ice-water must be absolutely forbidden, and it is essential in many cases that the liquid taken during a meal be restricted to one teacup of weak hot tea or simple hot water. Coffee and chocolate are distinctly deleterious, though sometimes the demand for coffee from a patient is so urgent that it may be allowed once a day, preferably taken without cream.

Gastric lavage has been much used in the treatment of chronic gastritis; when there is any interference with the escape of food through the pylorus, or when there is dilatation of the stomach, it may be essential; but in the great majority of cases of catarrh lavage is unnecessary and even harmful. Its use is apt to develop into a very deleterious habit: thus, we have known a sufferer to take habitually forty or fifty lavages a day.

In most cases of chronic gastric catarrh there is constipation, and even when it is not pronounced, cure is facilitated by keeping the bowels in a soluble condition. It is probably by its action on the liver and the portal circulation that the Carlsbad water and salts have acquired their great reputation. It is doubtful whether these European waters and the salts prepared from them are in any way superior to the Bedford water of Pennsylvania or the stronger springs of the Saratoga district in New York. In our practice the Carlsbad salts used at home have not seemed superior to saline mixtures. (See formula 19.) In some cases it has been advisable to alternate this formula with that of the aloes and senna mixture. (See formula 20.) Formulæ 2 and 3 may also be used on occasion. The laxative should be administered daily, so as to maintain a steady impression, and in such doses as to produce semi-solid stools.*

As regards the direct medicament of the stomach, the first principle is to avoid doing harm. All irritating substances are injurious; quinine, gentian, quassia, and other so-called "simple bitters," strychnine, aromatics, spices, should all be rigorously avoided. In any dose and in all doses they are incapable of good (except in alcoholic cases), and may do great harm; in some highly anæmic cases minute quantities of iron, such as occur in some mineral waters, are not disadvantageous.

The one remedy that is useful is silver nitrate. Its action is purely local, and direct contact with the mucous membrane is essential. It should never be administered in solution, as when it is so given decomposition begins from the moment it touches the lips. Further, the mucous membrane should be prepared for its action; a tumbler of hot water containing ten grains of sodium bicarbonate should be taken half an hour before each meal, followed in ten minutes by a pill of one-quarter grain of the nitrate with half a grain of extract of hyoscyamus. In severe cases the nitrate should be exhibited for from one to two months, the best results often being obtained by breaking the treatment up into periods of two or three weeks. During these interruptions bismuth subnitrate may be used, but resorcin, one to two grains, in solution, is usually more effective; and even zinc oxide (dose one grain) often acts better. The inferiority of these drugs, however, to the nitrate is very great. During the prolonged exhibition of the nitrate the inside of the lips and the gums should be watched for the appearance of the first evidence of approaching argyria; any discoloration should be the signal for the immediate withdrawal of the drug. The danger of argyria is, however, we think, greatly exaggerated; in our own practice we have never seen but one case in which the slightest show of color appeared upon the lips.

When in any case of gastric catarrh there is pronounced tendency to fermentation of food which is not controlled by regulation of the diet, a capsule of naphtol (2.5 grains) and carbolic acid (1 grain) is often very serviceable.

Chronic gastric catarrh of alcoholic origin differs from that of a different etiology in that the mucous membrane by long habit has become accustomed to irritants and often bears bitters well, seemingly being for the time benefited. Even in these cases, however, the sooner such drugs are got rid of the sooner will complete restoration be brought about.

ULCER OF THE STOMACH.

This disease is variously designated according as the anatomical, the clinical, or the etiological characteristics are made conspicuous. It is called *round* from its frequent shape, *chronic* from its usual prolonged existence, *perforating* since it shows a tendency to destroy the wall of the stomach, and *corrosive* or *peptic* from the supposed importance in its production of the digestive qualities of the contents of the stomach. Similar ulcers are seen also in the duodenum, and more rarely at the lower end of the œsophagus.

---

* The student should also compare this article with that on hepatic congestion.

ETIOLOGY.—It is generally recognized that the factors of chief importance in etiology are a localized diminution in the blood-supply to the wall and superacidity of the gastric juice, and severe burns are sometimes followed by duodenal ulcer. Local causes of a diminished circulation are to be found in injuries to the surface, arterial obstruction from sclerosis, thrombosis, or embolism, and spasmodic contraction of the muscular coat of the arteries; and Rasmussen considers that pressure upon the stomach from a tight band around the waist may be of etiological importance. The local enfeeblement of the circulation becomes intensified in general deterioration of the blood, as in anæmia, chlorosis, and tuberculosis. Ulcer of the stomach is twice as common in women as in men, and, according to Welch, is oftenest found in the former between the ages of twenty and thirty years, and in men between thirty and forty. It is of relatively frequent occurrence, being present in five per cent. of a large number of autopsies. Female domestics, particularly cooks, are usually considered to be especially liable, but gastric ulcer is found in all classes in life. Ulcer of the duodenum, on the other hand, is more common in men.

MORBID ANATOMY.—The shape of the ulcer is round or oblong, and is often compared to that of a funnel, from the fact that one part is often shelving, even in terraces, towards the sharply defined deepest point. The terraced appearance is due to the more extensive destruction of the mucous than of the muscular coat, and of the muscular than of the subperitoneal coat. Eventually a like destruction of all the coats takes place, and the ulcer appears as if a hole had been punched in the wall of the stomach. The size of the ulcer varies from one a half-inch in diameter to another covering a surface of the size of the palm of the hand. One or several ulcers are to be found, and the largest result either from the confluence of two or more or are due to the progressive enlargement of a single ulcer. The edge is sharply defined, and often shows at one point the stump of an artery perhaps obliterated either by a thrombus or by endarteritis. The gastric ulcer is generally found in the pyloric half of the stomach near the smaller curvature, and on the posterior wall. Its proximity to the smaller curvature and its peculiar shape are commonly attributed to the arborescent distribution of the branches of an obstructed coronary artery.

The tendency of the ulcer is towards healing or perforation. The healed ulcer is manifested by a scar often radiating, which, if at the pylorus or in the duodenum, is likely to cause a stenosis, resulting in dilatation of the stomach. The healing of an ulcer of the middle third of the stomach extensive enough to involve a considerable portion of its circumference is one of the causes of an hour-glass shape of the stomach. The scar of a previous ulcer and the clean-cut destruction of an active ulcer are to be found not infrequently in the same stomach. The ulcer about to perforate is often prevented from causing the escape of the contents of the stomach by the formation of fibrous adhesions between the peritoneal coat of the stomach and the peritoneal covering of neighboring structures, as the liver, spleen, pancreas, and diaphragm, which make the base of the ulcer after perforation has taken place. The digestive action of the gastric juice takes place in them, and extensive destruction sometimes ensues. If destruction of the adhesions or of the peritoneum at the base of the ulcer ensues, the escape of the contents of the stomach causes a general or circumscribed acute peritonitis. Perforation of the diaphragm has occurred, leading to the passage of the contents of the stomach into the pleural and pericardial cavities, and in rare instances the wall of the heart has been perforated. Fistulæ have been formed between the stomach and the duodenum or the colon, and the abscess following perforation of the stomach has been evacuated into the intestinal tract or through the abdominal wall. Profuse and often fatal hemorrhage results from the perforation of arteries or veins which lie in the edge or at the bottom of the ulcer, as the pancreatic or the coronary artery, branches of the splenic artery, the hepatic artery, and the splenic or portal veins.

SYMPTOMS.—There may be no symptoms indicative or suggestive of the presence of a gastric ulcer. As a rule, excessive acidity or secretion precedes and accompanies disturbances which are more directly attributable to the ulcer: hence for a longer or shorter period complaint is made of epigastric distress, relieved by food, especially by albuminous substances, of pyrosis, particularly during the height of digestion, of vomiting of an acrid fluid when the stomach contains no food, and of excessive appetite and frequent headache.

The most significant symptoms are localized pain, tenderness, and hemorrhage. The pain is burning or gnawing in character, and usually becomes more severe within an hour after food has been taken. It is sometimes intense, especially when indigestible articles of food are taken, and may be greatly relieved by bland liquids, as milk, freely diluted with an alkaline water; when so severe as to cause vomiting, relief is often immediately experienced when the stomach is emptied. The pain is diffused over the epigastrium, and at times radiates in all directions, extending even to the shoulders. It is apt to persist throughout the process of digestion, and is not infrequently increased on motion and in certain positions. Pressure sometimes aggravates, at other times lessens, the pain.

In addition to the diffused and radiating pain, circumscribed tenderness is frequent, usually midway between the ensiform cartilage and the navel, and at times is perceptible only on deep pressure. A tender spot is sometimes to be felt on the left of the spine in the immediate vicinity of the lower dorsal vertebræ, especially when pain from the ulcer extends into the back. According to Boas, this dorsal tenderness is present in one-third of the cases.

Easily recognized hemorrhage occurs as a symptom of ulcer in at least one-third of the cases, but slight bleeding not manifested by characteristic symptoms or signs is probably of far more frequent occurrence. The blood is either vomited, *hæmatemesis*, or appears in the stools, *melæna*, and not infrequently the black discoloration of the stools continues for a number of days after blood has been vomited. Hæmatemesis may be the first symptom exciting the suspicion of an ulcer, and may be so severe as to prove an immediate cause of death. Usually, however, it occurs after the attacks of pain have existed for some time. The vomiting of blood may come on at any time and without obvious cause; on the other hand, it may follow an error in diet or muscular strain. It is often immediately preceded by a sensation of faintness, and the patient may be in a state of collapse before blood is vomited, and may die even without the blood making its appearance externally. When blood is vomited it is usually abundant, dark red, liquid or clotted, either clear or mixed with food, dependent upon the length of time it remains in the stomach and upon the period of digestion. The attack of hæmatemesis usually ceases when the stomach is emptied of its contents, although a number of recurrences at intervals of hours or days is not infrequent, after which months or years may elapse before this symptom returns.

For a long time the nutrition may be sufficient, but emaciation in protracted cases is often extreme. The disturbance of nutrition is due in part to the avoidance of food in consequence of the distress it produces, and in part to the complications occurring in the course of ulcer, especially to dilatation of the stomach from cicatricial stenosis. Anæmia is conspicuous while hemorrhage from the ulcer is taking place. The tongue is usually clean, and palpation of the abdomen, in addition to making known localized tenderness, in chronic cases sometimes discloses a circumscribed induration due to a thickening of the wall of the stomach from chronic perigastritis. The chemical examination of the contents of the stomach ordinarily shows superacidity, from two to three per cent. of free hydrochloric acid usually being found. Subacidity, however, may be present when there has been excessive loss of blood or a complicating chronic gastritis, with or without dilatation of the stomach. The chemical condition of the contents of the stomach is to be determined only by examination of the vomit, since the use of the tube may prove a source of danger by causing hemorrhage or perforation.

The healing of the ulcer is followed by relief to the characteristic symptoms, although these may be replaced by those of dilatation of the stomach when stricture of the pylorus is caused by the scar. The symptoms last to disappear are those of superacidity, but after longer or shorter intervals of freedom from disturbance recurrences are likely to take place. The persistence of the symptoms of ulcer usually results from its extension, especially to parts in the vicinity of the stomach after the wall has been perforated. The *perforation* of the stomach may be suddenly announced by intense abdominal pain, followed by a tense, swollen, tympanitic, and tender abdomen, with absence of hepatic dulness, and by a febrile temperature, rapid respiration, and vomiting. The symptoms indicative of peritonitis from perforation are more likely to occur when the ulcer is seated in the anterior wall. When the posterior wall is perforated the symptoms of acute perforation are usually absent, owing to the previous formation of more or less dense fibrous adhesions, and fever also is lacking unless a subphrenic abscess, an omental bursitis, or an abscess of the liver, spleen, or pancreas result from the perforation.

DIAGNOSIS.—The diagnosis of ulcer of the stomach may be very difficult, especially when there is no hemorrhage. The pain from ulcer of the stomach is the result of the action of the irritating contents of the stomach upon the gastric nerves in general, as well as upon those exposed by the ulcer. Pain referred to the stomach, however, is of very frequent occurrence independent of ulcer, and especially to be eliminated is the gastralgia occurring as a neurosis. This is found in persons of a neurotic temperament not infrequently suffering from other neuralgias, and occurs in paroxysms with intervals of freedom from symptoms of digestive disturbance, is neither induced nor relieved by any especial variety of food, and is often diminished by pressure and the use of electricity. Gastric crises of pain in locomotor ataxia may suggest those of ulcer, but this disease is to be excluded by the presence of normal pupillary and patellar reflexes. An attack of biliary colic has suggested pain from ulcer of the stomach, but the character of the pain is griping, vomiting affords no relief, the liver and the gall-bladder are enlarged and tender, and jaundice is likely to follow. No absolute importance is to be attached to the seat and time of occurrence of the pain as determining the position of the ulcer, although the occurrence of pain several hours after eating and of melæna is suggestive of the situation of the ulcer in the duodenum. Superficial tenderness is indicative of ulcer rather of the anterior than of the posterior wall, and deep-seated pain and tenderness, especially when the latter is to be found in the back, are suggestive of ulcer of the posterior wall. If continual relief to the pain is experienced in any one position, it not infrequently results that the affected part of the stomach lies uppermost in the position giving relief to pain.

The hæmatemesis may be the result of various causes besides ulcer, as cancer, fibrous hepatitis, the rupture of an aneurism, poisons, foreign bodies, and infectious diseases. The history of the case and the associated symptoms are usually sufficient to eliminate many of these causes. In cancer of the stomach the hemorrhage is likely to be more frequent but is less abundant than in ulcer, and generally is of a coffee-grounds character. The pain is not so intense, is rather continuous than paroxysmal, and is not so directly affected by the nature of the food. The tumor of cancer is larger and more irregular than the palpable induration from chronic ulcer. The symptoms are suggestive of subacidity, and the contents of the stomach, as a rule, show deficient hydrochloric acid. The hæmatemesis from fibrous hepatitis is to be excluded by the physical examination of the liver and spleen, which shows atrophy of the one and enlargement of the other. The hemorrhage in this disease is likely to be both intestinal and gastric, the blood in the stools often being fresh, and not black or tarry, as in ulcer of the stomach or of the duodenum. Hemorrhage from a ruptured aneurism is usually immediately fatal, and is generally preceded by symptoms indicative of the presence of an aneurism.

PROGNOSIS.—The prognosis of ulcer of the stomach as to life is ordinarily favorable, but the patient may suffer from continuous or recurrent symptoms for many years. Although the disease is more frequent between the ages of twenty and forty, the mortality is greater from forty to sixty. Death occurs in about fifteen per cent. of the cases. It is due to peritonitis from perforation in about one-half of these, and to hemorrhage in about one-third. Persistence of pain irrespective of the quality of food, and extreme tenderness, are serious symptoms, since they suggest an advancing ulcer and impending perforation. Peritonitis from perforation, however, though usually fatal, is not invariably so, especially when a circumscribed abscess is the result. Profuse hemorrhage is indicative of the perforation of a large blood-vessel, and, therefore, is evidence of a deep ulcer, the healing of which takes place with difficulty, and the tendency of which is towards perforation.

TREATMENT.—In the treatment of gastric ulcer it is often wise to put the patient to bed for a length of time, in order to favor quiet of the stomach and to save the general strength.

The first indication is to check gastric movements; the second, to render as far as possible the gastric contents unirritating to the stomach; the third, to make local applications which shall favor the healing of the ulcer.

Unfortunately, there is no known drug which has the power of checking peristaltic movements of the stomach, unless it is opium; indeed, it is far from certain that doses of this narcotic not sufficiently large sensibly to derange the general health have such power. The ingestion of food such as occurs at an ordinary meal undoubtedly calls into functional activity the glands and the muscular walls of the stomach; hence total abstinence from stomach-food for some days is often prescribed in gastric ulcer, the patient being sustained by the use of nutritive enemata. It is not clear, however, that small quantities of bland food, such as milk, excite the viscus, and according to our experience total abstinence from stomach-food is of doubtful utility, and should be enforced only in severe cases after the failure of other measures. It usually suffices to reduce the stomach-food to an ounce of milk with one tablespoonful of lime water taken every one or two hours. Even this restriction of diet cannot be with safety maintained for an indefinite time, so that after a few days the milk should be increased, or bouillon with egg (the egg to be rapidly stirred into the bouillon whilst it is boiling hot, immediately on its removal from the fire), and various purées,—as of chicken or of sweetbread,—junket, and custards, may be allowed. Next, scraped raw beef may be taken, then stewed sweetbread, then Hamburg steaks. Any food should be given in small quantity at short intervals.

The drugs which are used in gastric ulcer are bismuth subnitrate, silver nitrate, resorcin, zinc oxide, and oil of turpentine. Of these drugs we have never seen any distinct effects from bismuth. The oil of turpentine in the ordinary case will do great hurt; it is to be used only, and then very cautiously, in very old cases in which there is such a history as to warrant the belief that the ulceration has existed continuously for a great length of time. Silver nitrate is by far the most generally useful; it should be given as in chronic gastritis. Resorcin in doses of one to two grains is sometimes of service. Zinc oxide may be tried, but is usually inefficient.

In all cases of gastric ulcer constipation should be carefully guarded against; mercurials are occasionally of advantage; salines should be exhibited as in chronic gastritis.

Excessive vomiting often yields to very careful and restricted feeding, but if it continue is to be met by the use of cracked ice, by fomentations or a small blister upon the epigastrium, and by the exhibition of cocaine, or bismuth, or cerium oxalate. In obstinate cases lavage is frequently of great service; rarely opium suppositories are necessary.

Gastralgia may be so severe as to require active treatment. Sodium bicarbonate, in doses of ten grains or more well diluted, will sometimes bring relief. Hot applications and sinapisms applied to the epigastrium are often useful. The combination of prussic acid and cocaine (formula 26) is sometimes serviceable, but in severe paroxysms opium must be used. Antifebrin and antipyrin are very rarely effective in a gastralgia dependent upon ulceration. The use of irritating substances, such as chloroform and Hoffmann's anodyne, always endangers aggravation of the original disease.

Hemorrhage.—In cases of serious gastric hemorrhage all food should be temporarily withheld from the stomach, the patient being sustained by nutritive enemata; or milk and lime water should be given at short intervals in very small doses. Opium should be administered in such dose as to produce distinct quiet and obtunding of the nervous system; very commonly suppositories of extract of opium, one grain, and extract of belladonna, one-sixth of a grain, afford the best method of exhibition.

Of all the hæmostatic drugs, Monsel's solution is the most powerful; it should be exhibited in doses of one to two drops from every ten minutes to every hour, *pro re nata*, great care being exercised to avoid vomiting. Extract of ergot may be given hypodermically, as in hæmoptysis; ice should be freely applied over the stomach. If a hemorrhage from the stomach depends upon congestion of the portal circulation, a mercurial or saline purge may be of great benefit; if, however, the cause of the bleeding is ulceration, purgatives are to be avoided unless there has been pronounced and persistent constipation.

When the bleeding is from an ulceration there is a strong incentive to attempt its surgical arrest; the difficulties, however, of locating a bleeding spot, the uncertainty at any moment whether there will be further bleeding, and the danger of death from shock in an exsanguined person render the operation a very dubious procedure. Moreover, at present there is no sufficient evidence from recorded cases to guide the surgeon.

Perforation.—When perforation of the stomach occurs slowly, with the formation of abundant adhesions and a resultant secondary abscess, no other treatment may be needed than the evacuation and surgical treatment of this abscess. When the perforation is abrupt and accompanied with serious symptoms, radical surgical interference should be immediate; each hour of delay sensibly increases the risk to life. According to the statistics of Richardson, in forty-four such operations the mortality was 77.27 per cent., although the last twenty-one cases gave a mortality of only 57.01 per cent., the difference probably being due to improvement in surgical technique.

Surgical removal of an ulcer has been performed in a few cases, usually with fatal results. It seems to us not justifiable unless called for by excessive hemorrhage, by perforation, or by narrowing of the pyloric end of the stomach through progressive cicatricial contraction.

CANCER OF THE STOMACH.

Cancer of the stomach occurs in from thirty-five to forty per cent. of cases of cancer. According to Wyss, in middle Europe two per cent. of deaths are due to gastric cancer. The frequency of this disease somewhat varies, however, in different countries, and in New England the mortality from cancer of the stomach is about one-half of one per cent.

ETIOLOGY.—Cancer of the stomach occurs with nearly equal frequency in the two sexes. About three-fourths of all cases occur between the ages of forty and seventy years, and two-thirds between forty and sixty years. Heredity seems of importance in etiology in a certain number of cases. The significance of local irritation is suggested by the great frequency of cancer at the narrowest portion of the stomach, mainly at the pylorus, and by the previous occurrence of ulcer in about six per cent. of the cases.

Morbid Anatomy.—Cancer of the stomach is either primary or secondary, the former representing a growth arising in the deeper layer of the mucous membrane, the latter often extending inward from the peritoneal coat of the stomach. Primary cancer alone is of especial clinical importance. The varieties usually recognized are the medullary, fibrous, hyaline (gelatinous or colloid), and cylindrical-cell cancer. The growth occurs as single or multiple nodules, or as a diffused infiltration of the wall, which sometimes involves the entire stomach. The disease is seated at the pylorus in more than one-half of the cases, next in frequency at the smaller curvature, more rarely at the larger curvature, in the anterior or posterior wall, or in the vicinity of the œsophageal opening. Nodular cancer, as it increases in size, projects above the mucous membrane of the stomach, spreads laterally and in depth, its surface being smooth, lobulated, or papillate, and its edge often everted. The medullary variety of cancer is soft and often exceedingly vascular, while the fibrous and hyaline varieties are dense and contain few and comparatively small blood-vessels. As the nodules increase in size, hemorrhage or necrosis is likely to take place in them, and the portions of the tumor thus altered are readily corroded by the gastric juice, and the cancerous ulcer results, which tends to extend in depth until perforation of the stomach ensues. In about three per cent. of the cases perforation takes place into the general peritoneal cavity, and a fatal peritonitis follows. Usually, however, adhesions are formed between the stomach and the liver, spleen, pancreas, or diaphragm before actual perforation takes place, and these form the base of the cancerous ulcer when the wall of the stomach is destroyed. The cancerous growth extending into these structures in turn becomes softened and corroded by the gastric juice, and large cavities are formed outside of the stomach, but freely communicating with it. Food enters these cavities, and, becoming decomposed, causes an acute inflammation, which may result in abscesses and thrombophlebitis. The adhesive inflammation preceding perforation may take place also between the stomach and the abdominal wall, the colon, or the small intestine, and a cutaneous or gastro-intestinal fistula follow the perforation. A tumor at the pylorus often causes obstruction at this orifice, with secondary dilatation of the stomach, although occasionally a sufficient compensatory hypertrophy of the wall results. The mucous membrane of the stomach affected by cancer presents the characteristics of a chronic gastritis.

Infiltrating cancer of the stomach is usually either fibrous or gelatinous, and all the coats of the stomach are invaded, although the mucous and muscular coats are more especially altered. A limited portion of the stomach or the entire organ may be infiltrated, and the stomach is accordingly either enlarged or diminished in size. The diminution in size is most marked in case of the infiltration of the wall by fibrous cancer, and the gross appearances may resemble so closely those of the cirrhotic stomach of chronic gastritis that microscopical examination will be necessary to enable the differential diagnosis to be made. In fibrous cancer or scirrhus the anatomical diagnosis is not infrequently suggested by the recognition of evidence of cancer elsewhere.

The extension of the cancer from the stomach to other organs is frequent. The lymph-glands near the smaller curvature of the stomach are oftenest diseased, and from these the cancer may extend to remote lymph-glands, especially to the supraclavicular and to the inguinal lymphatic glands. Extension to the liver takes place in about one-third of the cases, either directly after adhesions are formed or by means of the portal circulation. The peritoneum often becomes cancerous by the extension of the disease from the stomach not only in the immediate vicinity of this organ, but also at remoter parts, frequently at the bottom of the pelvis in Douglas's fossa. Cancer may extend from the stomach through the peritoneum to the diaphragm, through the diaphragm to the pleura, and thence to the lungs, or it may invade the lungs, brain, and other parts of the body by means of the circulation.

SYMPTOMS.—Pain, vomiting, and cachexia, and tumor in the region of the stomach, are the conditions suggesting cancer of this organ. The development of the cancer is not infrequently preceded for a long time by symptoms of chronic gastritis, although occasionally there is no interference whatsoever with digestion until the onset of the symptoms attributable to the presence of cancer; and in elderly persons especially the symptoms may be exceedingly obscure. Pain is one of the earliest symptoms, is present in three-fourths of the cases, and is generally limited to the epigastrium, although sometimes extending into the sides, the back, and more rarely to the shoulders. It is usually constant, although often somewhat aggravated after eating. The pain is described as dragging, burning, gnawing, or cutting, and exceptionally is as intense as in ulcer of the stomach.

Vomiting is the most constant symptom, and occurs either early or late in the course of the disease in four-fifths of the cases. It is more likely to be absent when the cancerous growth is at some distance from the orifices of the stomach. Its occurrence is almost invariable in cancer of the pylorus, and the quantity vomited may then be large in consequence of secondary dilatation of the stomach. The frequency of the vomiting gradually increases with the progress of the disease, and eventually is likely to become extreme. The immediate act of vomiting has no definite relation to the taking of food; indeed, it may occur when the stomach is empty; nor does the time at which vomiting takes place bear any definite relation to the seat of the tumor. It is asserted, however, that vomiting soon after eating is frequent in cancer seated at the cardiac end of the stomach, while vomiting occurring an hour or later after eating is more likely to be connected with pyloric cancer. The vomit is sour, and consists of food, often incompletely digested, and of mucus, and may contain fragments of the cancer. Blood is present from time to time in nearly one-half of the cases, but is rarely so profuse as in gastric ulcer, and frequently resembles coffee-grounds in consequence of its gradual oozing, prolonged retention, and partial digestion.

Cachexia, the bad state or habit of the patient, begins early, progresses continuously, and is chiefly dependent upon the interference with gastric digestion and upon the hemorrhage: it is also influenced by the extension of the disease to other parts, especially to the biliary tract. The patient is usually thin, pale, at times somewhat jaundiced, the expression of the face is often one of suffering, and œdema of the skin, especially of the legs and feet, is frequent.

Tumor is to be recognized early or late in the course of the disease in about four-fifths of the cases, and the infiltrating cancer causes rather a diffuse resistance than a circumscribed induration. Tumors of the lesser curvature become apparent only when the stomach is dislocated downward, and then are made conspicuous often from the associated extension of the disease to the neighboring lymphatic glands. When the tumor is small it is frequently concealed beneath the left lobe of the liver, but is even then sometimes brought within reach by prolonged inspiration. As it increases in size, especially when free from adhesions, it is freely movable, and is generally to be felt and often to be seen in the epigastrium or in the hypochondriac regions, and exceptionally may descend so far as to lie in the vicinity of the symphysis pubis. The tumor is usually sharply defined, rounded, or irregularly nodulated, hard, and often tender. The tumor, though commonly movable, when non-adherent changes its position but little during respiration; indeed, its ascent on inspiration is easily prevented by pressure of the hand; but when the entire stomach is infiltrated with cancer the respiratory movements of the diaphragm are readily transmitted. When adherent to the liver its motility is limited by that of this organ, and when united to the pancreas or to the posterior abdominal wall it is rendered immovable. It frequently transmits the pulsations of the aorta when the patient is supine, but in the knee-elbow position the pulsations are not transmitted unless the tumor is adherent in the vicinity of the aorta. The movable tumor is sometimes more readily felt when the patient is on his hands and knees. When the stomach is inflated a tumor of the anterior wall is often made more conspicuous, while that seated in the posterior wall is no longer to be felt. The position of the tumor at the greater curvature of the stomach may be made evident by distention of the colon with air or water.

During the progress of cancer of the stomach the appetite is usually feeble, there is frequent nausea, and the disturbances of digestion found in chronic gastritis or in dilatation of the stomach are often present. There may be, however, a good appetite and but little disturbance of digestion. The bowels are usually constipated, and the dejections are often black from the presence of blood even when hæmatemesis is absent. The urine is often diminished in quantity, is usually alkaline or neutral, and frequently contains an excess of indican. A considerable leukocytosis is frequent.

Cancer of the stomach lasts from one to three years, the shorter period being the limit of life generally following the discovery of the tumor. Death is apt to result from progressive loss of flesh and strength essentially from starvation, unless extension to the serous membranes, pulmonary embolism, or an acute inflammatory complication, as peritonitis from perforation, supervenes. Towards the end of life frequent chills followed by fever and suggestive of malarial infection at times occur, and are due probably to foci of suppuration either in the vicinity of a perforating cancer of the stomach or in the liver or lungs. Profound disturbance of the nervous system occasionally occurs towards the end of life, the symptoms resembling those occurring in diabetic coma. The patient at first is restless and wakeful, the respiration becomes deep, and the pulse is rapid and weak. Drowsiness supervenes, and terminates in coma, in which death takes place. In such cases acetone and diacetic acid have been found in the urine.

Diagnosis.—The positive diagnosis of cancer of the stomach before a tumor is discovered or particles of cancer are to be found in the vomit is impossible. This disease is to be suspected if the symptoms of chronic gastritis persist without obvious cause and are associated with cachexia out of proportion to the severity of the digestive disturbance, especially in elderly persons. When pain and hæmatemesis also are present and tumor is not apparent, the differential diagnosis practically lies between chronic gastritis, ulcer, and cancer. Chronic gastritis is to be eliminated by a lack of cause, a cachexia greater than the digestive disturbances suffice to account for, considerable pain and tenderness, absence of hæmatemesis, and the usual inability to arrest the progress of the disease. Ulcer of the stomach is to be eliminated by the age of the patient, the absence of intense pain occasioned by certain foods and relieved by others and by alkalies, the lack of profuse hemorrhage, absent superacidity of the contents of the stomach, existing cachexia, and failure of relief from treatment. The diagnosis of cancer of the stomach is based upon the age of the patient, the persistence of the pain, the rapid progress of the cachexia, often after years of digestive disturbance, coffee-grounds vomit, or melæna, and usual failure to improve under treatment.

The discovery of a tumor in the region of the stomach is the diagnostic sign of greatest importance, and, whether present or absent, search should be made also for possible secondary tumors elsewhere, especially of the supraclavicular and inguinal glands and of the peritoneum in Douglas's fossa. Other neoplasms than cancer may grow from the wall of the stomach, but, with the exception of sarcoma, usually give rise to little or no disturbance of digestion. The distinction between sarcoma and cancer is rather histological than clinical. Foreign bodies in the stomach are generally to be excluded by the history of the case and by the absence of the extreme cachexia of cancer. The resistance due to acute perigastritis or acute pancreatitis is sudden in its origin and rapid in its progress. Cancer of the liver or gall-bladder changes position with respiration, and is not associated with the gastric pain, persistent vomiting, and hæmatemesis of cancer of the stomach. Impacted fæces in the colon are to be excluded by means of treatment. A movable kidney is to be replaced in the position of the kidney, and a tumor of the abdominal wall is unaccompanied by the gastric symptoms of cancer.

The importance of the results of the chemical examination of the contents of the stomach removed after a test meal is still a matter of opinion. Persistent subacidity or inacidity, from the lack of hydrochloric acid, usually exists, although in the rare cases in which cancer follows ulcer an excess of this acid has been found. The persistent presence of lactic acid, which is due to stagnation of the gastric contents, occurs oftenest in cancer of the stomach, and therefore is important in the way of suggestion. According to Boas, it is always absent in atony, in chronic gastritis, and in dilatation of the stomach from pyloric stenosis. Its asserted value as a test of the early stage of cancer is denied by Rosenheim, who regards it as evidence rather of a late than of an early stage of the disease. Since the presence of lactic acid in the contents of the stomach may result from other pathological conditions, and cancer may be present without lactic acid, it is evident that its presence in the contents of the stomach is of relative, not of absolute, value in the diagnosis of cancer.

PROGNOSIS.—Cancer of the stomach is fatal, and death occurs the sooner the greater the cachexia and the more persistent the hemorrhage, unless early extirpation by surgical treatment proves efficacious in preventing recurrence of the disease. The statistics recently given by Wölfler show an average mortality from resection of the pylorus of thirty-one and two-tenths per cent. in one hundred and seventy-three cases operated upon between 1888 and 1896, and of sixteen to twenty-five per cent. in the practice of a few surgeons. The mortality from the operation is twice as great when adhesions are present. Two patients have lived eight years after resection of the cancerous pylorus, four have lived more than five years, three have lived more than four years, and fourteen have lived from two to four years after the operation. The average duration of life after resection is one and a half years. Gastro-enterostomy, the mortality from which since 1888 in one hundred and ninety-five cases is thirty per cent., is usually inefficacious in prolonging life, though when successful it affords relief to symptoms and has been followed by more than two years of life.

TREATMENT.—The medical treatment of cancer of the stomach is purely palliative. By very careful feeding, by the use of lavage (see Dilatation of the Stomach), by the occasional administration of creosote, guaiacol, silver nitrate, or bismuth subnitrate, and by the cautious use of morphine when necessary to relieve pain, the patient should be kept as comfortable as possible. In a number of cases surgical exsection has been performed. When the tumor can be felt through the abdominal wall the probabilities are always that the disease involves so much tissue that surgical interference offers but little hope of permanent cure.

GASTRIC NEUROSES.

Definition.—Modifications of gastric function independent of anatomical lesion, and attributable to disturbances of the nervous system.

Etiology.—Essential in the production of gastric neuroses is undue excitability of the nervous system: hence they are more common in the young than in the old, in women than in men, in neurasthenia, hysteria, hypochondriasis, chlorosis, splanchnoptosis, and pregnancy, and in sufferers from Graves's disease. The disturbances of function indicative of gastric neuroses also occur as secondary or symptomatic conditions of the various diseases of the stomach and of parts in its vicinity, especially the biliary tract and the pancreas. They are direct symptoms in diseases of the central nervous system, both of the brain and of the cord, and reflex phenomena in diseases of the pelvis, and perhaps from intestinal parasites. They are of frequent occurrence in acute and chronic infectious diseases, in anæmia, uræmia, gout, and diabetes, and in consequence of the abuse of tea, coffee, alcohol, and tobacco. They are indicative of the toxic action of various drugs, as opium, quinine, and digitalis. The especial neuroses may be mild or severe, may occur alone or associated with others, and may produce slight or severe disturbance of nutrition. The patients, therefore, are either pale, thin, and weak in mind and body or stout and strong. When the mind is diverted or the patient is alone or in a state of rest the symptoms frequently disappear, to return when the patient is in company of other persons or is overworked or depressed. Important as suggesting the neurotic character of the gastric disturbance is the tendency of the patient to exaggerate it. The expression of suffering from gastralgia is extreme. Belching is vehement, regurgitation is constant, and vomiting is frequent and easy. The recognition of a gastric neurosis is essentially based upon the knowledge of the origin of a symptom, since in the one person the disturbance of function is a sign of disease in the stomach or elsewhere, and in the other there is no satisfactory evidence of such disease.

Gastric neuroses are usually divided into disturbances of sensation, motion, and secretion, and in general represent an excessive or defective exercise of the function concerned.

Neuroses of Sensation.—Loss of appetite, *anorexia*, *acoria* (loss of the feeling of satiety), and *hyperorexia*, excessive appetite, when not symptomatic of other disease, represent gastric neuroses which may be excited by emotional disturbances or occur as a simple manifestation of neurasthenia. Under hyperorexia are to be included *bulimia* and *polyphagia*, both terms indicative of an intense appetite. In bulimia the attack is paroxysmal, associated with burning pain referred to the stomach, is often associated with headache and a sensation of faintness, and is relieved by food. Polyphagia is permanent and unaccompanied by sensations of weakness. Boas applies the term *gastralgokenosis* to paroxysms of gastric pain occurring when the stomach is empty but which are easily relieved by food. Parorexia or *pica* represents the craving for unnatural, impossible, or disgusting articles of food, as dirt, chalk, and slate-pencils, and includes both the various longings of pregnant women and the coprophagy of the insane and hysterical.

Hyperæsthesia.—This term is applied to the occurrence of a sense of discomfort or pain provoked by food and frequently continuing for a long period of time. Paroxysmal attacks of epigastric pain independent of food are called *gastralgia*, or, when mild, *gastrodynia*, and are regarded as a neuralgia of the pneumogastric nerves. The paroxysms occur independently of food. The pain is often intense, may extend into the back, is somewhat relieved by pressure, is often associated with a sense of substernal constriction, and may end in spasms or a condition of semiconsciousness. Severe attacks may last for several hours unless relief is afforded.

TREATMENT.—For the relief of gastralgia, hypodermic injections of morphine are often required; but hydrocyanic acid in dose of three drops is sometimes efficient. When the attacks are frequent and persistent, the epigastrium may be blistered. When they are periodical, antiperiodic doses of quinine may be used. Antipyrin in full dose is especially useful when the gastralgia is due to a spinal sclerosis.

Neuroses of Motility.—Increased peristalsis as a motor neurosis is shown by eructation, rumination, habitual vomiting, cramps, and peristaltic unrest. Deficient peristalsis as a neurosis occurs in atony of the stomach and incompetency of the pylorus.

Eructation is to be regarded as a neurosis when air is swallowed in large quantity, *aerophagy*, and expelled from the stomach often with a loud noise. The air may be swallowed in such quantity as to cause distention of the stomach and the production of epigastric distress, palpitation, and anxiety.

Regurgitation in its most frequent form is represented by pyrosis, the entrance of the acid contents of the stomach into the œsophagus and mouth as a hot, thin fluid, often setting the teeth on edge. In *rumination*, or *merycism*, which is a rare motor neurosis, mouthfuls of food are forced from the stomach and again swallowed. The condition may exist for years without other disturbance of digestion, and the general nutrition suffer but little.

In *habitual* or *nervous vomiting*, which occurs both in children and in adults, a part or all of the contents of the stomach are easily and quickly expelled without preliminary nausea and independently of the quality of the food or of the stage of digestion. They are but little irritating if expelled soon after eating, but are acid at the height of digestion. In this neurosis longer or shorter intervals of freedom from vomiting occur, and, as a rule, the health of the individual is not seriously impaired. Exceptionally, especially in the hyperemesis of pregnancy, nervous vomiting may prove a source of progressive emaciation and debility and possibly a cause of death. Leyden has described the occurrence of periodical vomiting in persons free from other evidence of disease, and it is not infrequent as a reflex neurosis in menstruating women. The presence of blood in the periodical vomiting of the latter is to be doubted, as is also the assertion of the vomiting of the contents of the large intestine by hysterical or neurasthenic patients.

Spasm or *cramp* of the stomach may affect the stomach as a whole or be limited to either orifice. Cramp of the entire stomach is seen in the *peristaltic unrest* described by Kussmaul. In this condition the patient complains of distress from muscular contractions of the stomach, which are usually associated with borborygmus and splashing and cause mental distress to the patient, perhaps compelling her to lead a secluded life. The peristalsis is often visible when the stomach is prolapsed. In general, however, visible gastric peristalsis is the result of compensatory hypertrophy of the wall in stricture of the pylorus. Leo has observed in several instances hypermotility of the stomach causing an early expulsion of its contents, as shown by the examination after the use of test meals. The effect is essentially that of incompetency of the pylorus.

Spasm at the œsophageal end of the stomach is not to be discriminated from spasm at the lower end of the œsophagus. In each there are a sense of deep-seated obstruction in the vicinity of the lower end of the sternum, to be overcome by the passage of a full-sized sound, and the regurgitation of a tasteless, watery, sometimes frothy fluid. Spasm of the pylorus as a pure neurosis is with difficulty to be recognized. If it exists, the symptoms and signs would be those of atony of the stomach long preceded or accompanied by frequent attacks of localized pain and resistance in the region of the pylorus.

Atony of the stomach, though often the result of organic disease of the wall, may occur as a neurosis. It is manifested by diminished motility, in consequence of which the food is unduly retained in the stomach, which is distended temporarily, and is always in danger of becoming permanently dilated. There is a sense of fulness and weight in the epigastrium, and the use of a test meal shows that although the stomach is empty before breakfast it contains food three hours after Ewald's breakfast and seven hours after Leube's meal. The impaired motility is also to be determined by means of salol, as stated on page 822, and if present may delay the elimination of the salol for two days.

Pyloric incompetency is stated by Ebstein to occur as a neurosis; it permits the undigested contents of the stomach to enter the intestine prematurely, in consequence of which diarrhœa is likely to take place. Incompetency of the pylorus allows also the regurgitation into the stomach of the contents of the duodenum. The diagnosis is to be made by the visible passage of air or gas into the intestine when the stomach is inflated.

Neuroses of Secretion.—*Superacidity*, *hyperacidity*, and *peracidity* are terms applied to an increase of the hydrochloric acid of the gastric contents occasioned by the presence of food. The increase of the acid secretion from the stomach may also continue while the stomach is empty, either continuously or periodically. The periodical supersecretion is designated by Rossbach *gastroxynsis.* Superacidity causes diffused pain and tenderness in the epigastrium, nausea, pyrosis, and acid vomiting, associated in supersecretion with severe headache. Relief to the symptoms is often caused immediately by vomiting or by neutralizing the excessive acidity of the contents of the stomach. The appetite is unaffected, and albuminoids produce less disturbance of digestion than do starchy foods. Long-continued superacidity is likely to result in enfeebled motility and eventual dilatation, attributable perhaps to the production of pyloric spasm by the irritation of the excessively acid contents of the stomach. Superacidity is shown by the presence of more than two per cent. of hydrochloric acid removed after a test meal, and supersecretion is made evident by the constant presence of an ounce or more of gastric juice in the stomach which has long been deprived of food.

The existence of a constant subacidity due to a diminished quantity or to lack of hydrochloric acid is of doubtful occurrence as a pure neurosis, though frequent in consequence of disease. The contents of the stomach even at the height of digestion then show less than one per cent. of free hydrochloric acid.

TREATMENT.—Acidity of the stomach is to be temporarily relieved by the use of sodium bicarbonate, lime water, or ammonia in some form. Permanent relief is to be obtained by curing the cause of the acidity. The habit of using antacids in excessive dose is easily formed: in such doses they are irritant to the gastric mucous membrane.

NERVOUS DYSPEPSIA.

Nervous dyspepsia, a term introduced by Leube, denotes the occurrence of more or less complex groupings of the various gastric neuroses above mentioned. It is, therefore, a term of convenience, and its use may demand the recognition of as many varieties of nervous dyspepsia as there are gastric neuroses. In Leube's limitation of the term, however, the disturbances are moderate, confined to the act of digestion, and those of sensation predominate over the modifications of motion and secretion, whereas extreme disturbances of function, whether of sensation, secretion, or motion, are regarded as distinct diseases,—namely, gastralgia, superacidity and supersecretion, and nervous vomiting. The diagnosis of nervous dyspepsia depends largely upon the elimination of organic causes of the symptoms, since a purely nervous dyspepsia is considered to be independent of obvious anatomical changes. The appearance and behavior of the patient are often significant of extreme nervousness, though prolonged observation may be necessary to determine its presence. Although the symptoms resemble most closely those of chronic gastritis, they frequently bear no constant relation to the quantity or quality of food, and this disease is to be eliminated by the lacking etiology, the inconstancy of the symptoms, and the absence of mucus in the washings from the stomach. Ulcer may be suspected from the apparent severity of the gastralgia, and cancer may be suggested when excessive emaciation occurs. The positive evidence of these diseases as previously described is lacking. The prognosis of nervous dyspepsia and of the gastric neuroses is favorable for the primary or idiopathic varieties, although recurrences are frequent. The prognosis of the secondary gastric neurosis is that of the diseases in which they occur as symptoms.

TREATMENT.—In the treatment of gastric neuroses and nervous dyspepsia the first principle is attention to the general health of the patient. In some cases the atony of the digestive organs is the result of physical indolence, and is not to be lessened, except by progressively graduated exercise. If there is overwork or overstrain, it is essential to remove it. In many cases the dyspepsia is but a local expression of the general exhaustion, under which circumstances the rest-cure is to be carried out with a greater or less degree of rigor according to the necessities of the individual. When there is hyperacidity in a case of nervous dyspepsia, the best clinical results are to be obtained by the treatment laid down under Chronic Gastric Catarrh. Even when the gastric disturbances are seemingly connected with a general hysteria and constitute a neurosis, if there be hyperacidity, with or without epigastric tenderness, the treatment of gastric catarrh may be essayed.

In ordinary atonic dyspepsia without excessive acidity the regulation of the diet is very similar to that of gastric catarrh. It is essential to consult the past experience of the individual, as it is in these cases especially that substances not usually digestible are for certain individuals the most suitable food. Ice-cold water should never be allowed with or shortly after a meal. Hot water often acts as a stomachic, and if taken (one tumblerful) fifteen minutes before eating quenches thirst.

Among medicinal substances the simple bitters with aromatics, especially one of the peppers, are efficacious. In mild lack of digestive power, such as frequently occurs in convalescents, formula 23 will be found effective. In more serious cases formula 27 may be tried. When there is much flatulence, intestinal antiseptics, such as strontium salicylate, naphtol, and carbolic acid, may be of great service. (Formula 28.) As constipation is ordinarily the rule, laxatives must be freely used, and especially must care be taken, by the use of large enemata, to keep the colon completely emptied, so that there shall be no retention of scybala or other fæces.

There is much difference of opinion concerning the value of digestive ferments in atonic dyspepsia and other forms of indigestion. The fact that along with the ferments other remedies or measures are always used for the relief of the dyspepsia makes it very difficult to decide what part the ferments play in the relief of the patient, but our experience has not led us to attach much value to their influence ; nevertheless, the enormous quantities of them which are sold indicate that very many practitioners are of a different opinion, and, as the ferments are harmless, they may well be used as an addition to other treatment. If employed, they should be given much more freely than is usually done. The pure pepsin of the United States Pharmacopœia (not the saccharated) should be selected ; five grains should be given in the middle of the meal and five grains immediately upon completion of the meal, an amount equal to one hundred grains of the saccharated or ordinary pepsin. Pancreatin acts only in an alkaline solution, and any effect which it has when given by the mouth must be due to its escape into the intestinal tract.

CHAPTER III.

DISEASES OF THE INTESTINE.

ENTEROPTOSIS.

Definition.—Falling of the intestine.

Prolapse of the intestine occurs not infrequently in the visceral prolapse, *splanchnoptosis*, to which Glénard has directed especial attention. As in the prolapsed stomach, wandering spleen or liver, movable kidney, or displaced uterus, the abnormality is usually found in women accustomed to tight lacing, repeated pregnancies, or muscular strain. Both small and large intestines may become prolapsed, the coils of the small intestine lying largely in the lower part of the abdomen and pelvis. Prolapse of the colon (*coloptosis*) is more frequently observed, the freely movable transverse colon being the portion which is oftenest displaced. It may become elongated and tortuous, and be S- or M-shaped, the most dependent portion lying at the symphysis pubis.

Prolapse of the small intestine produces no symptoms, except those mentioned in connection with gastroptosis. Prolapse of the large intestine may cause constipation, flatulence, and colic. Glénard asserts that the transverse colon can be felt as a cord in the upper part of the abdomen. Boas and Ewald believe that this palpable cord is the pancreas. The symptoms associated with enteroptosis are more especially referable to the stomach, kidney, spleen, uterus, and the nervous system, and those referable to the intestine are either overlooked or attributed to disturbance of gastric digestion.

HEMORRHAGE.

Bleeding frequently takes place from the blood-vessels of the intestine, and when sufficient to cause bloody stools becomes a symptom of marked importance.

Etiology and Appearances.—The hemorrhage is the result of general and of local causes. Among the former are the various diseases of the blood, constitutional affections, and infectious diseases. Closely allied are the hemorrhages which occur in jaundice and in phosphorus poisoning. Among the local causes of intestinal hemorrhage are superficial lesions, especially the various forms of ulcer, polypus, and cancer. In this series are to be included the hemorrhages due to traumatism, to intussusception, and to intestinal parasites. Important also is obstruction to the venous circulation, as in fibrous hepatitis, portal thrombosis, valvular disease of the heart, and pulmonary emphysema. Dilated veins, especially in the rectum, aneurism, particularly of the aorta or its primary branches, and embolism and thrombosis of the mesenteric vessels are also causes. Nothnagel has called attention to the occurrence in phthisis of intestinal hemorrhage not dependent upon intestinal ulcers, and Grainger Stewart attributes certain cases of intestinal hemorrhage to amyloid degeneration. The most frequent causes are hemorrhoids, inflammation and cancer of the large intestine, and typhoid fever.

The appearances presented by the blood vary according to the quantity, the rapidity of the hemorrhage, the source, and the length of time occupied in passing through the intestine. Frequently hemorrhage is so slight as to be recognized only with the aid of the microscope, by which blood-corpuscles and blood-crystals are distinguished. The more abundant and the more rapid the hemorrhage the more likely is the blood to appear in clots, which may protrude several inches from the anus. The more gradual the bleeding the more constantly is the blood mixed with fæces, and the mixture is more intimate the higher up in the intestine the source of the bleeding. The less active the peristalsis the longer is the blood retained in the intestine, and the more likely is it to be of a dark color and relatively firm consistency. A dark, tarry appearance of the stools is suggestive of hemorrhage from ulcer of the duodenum.

Hemorrhage from affections of the mesenteric blood-vessels requires especial mention, and the publications of Elliot and Watson form an important contribution to our knowledge of the subject. The mesenteric artery—usually the superior—may become obstructed by an embolus which arises from a diseased aortic or mitral valve, or from a parietal thrombus of the left ventricle or auricle, or from a thrombus of the aorta, or from an aneurism of this artery. More rarely thrombosis of the mesenteric artery occurs in consequence of arterio-sclerosis. Thrombosis of the superior mesenteric vein is less frequent than is embolism of the corresponding artery, and may be caused by a twist of the intestine or by the strangulation of a hernia. We have seen it follow embolism of the splenic artery, being continued from a secondary thrombosis of the splenic vein. In certain cases no satisfactory explanation for its origin exists. The effect upon the intestine is the same in both thrombosis and embolism of these vessels. The slowness of the circulation in the mesenteric artery is such that obstruction of the trunk causes a hemorrhagic infarction of the part supplied, as in the case of a terminal artery, and the wall of more or less of the small intestine is thickened and of a purple color, the contents of the intestine being largely bloody. The longer the infarction has existed the more likely are necrosis and gangrene to occur. In the last event peritonitis results, and a bloody exudation is found in the abdominal cavity.

Watson, from the study of a collection of twenty-seven cases of embolism of the superior mesenteric artery, finds that the first symptom of this lesion is usually a violent abdominal pain, not sharply defined, and often associated with vomiting and diarrhœa. The stools were bloody in one-half the cases, and the temperature was subnormal in one-third. The stools are at times of a tarry character, and the affected portion of the intestine may be distinctly resistant on palpation. The patient may die within twenty-four hours after the onset of the symptoms, or may live nearly a fortnight. If the patient survive the immediate effects of the embolism, symptoms of peritonitis are likely to occur in the course of two or three days.

The diagnosis is based upon the occurrence of a sudden abdominal pain associated with vomiting, diarrhœa, bloody stools, and subnormal temperature in an elderly person previously well or having a history of antecedent embolism and presenting evidence of valvular endocarditis or of arterio-sclerosis, or in a person suffering from acute rheumatism. Other sources of the sudden pain and intestinal hemorrhage are to be excluded when possible.

Cases of embolism of the mesenteric artery have so constantly proved fatal that the prognosis has been recognized as of the gravest nature, although in rare instances spontaneous recovery has taken place. Elliot has shown that surgical treatment of this lesion may prove efficacious, a patient from whom he removed four feet of the intestine in a state of infarction having recovered.

TREATMENT.—The treatment of hemorrhage from the small intestine has been sufficiently described in the article on Typhoid Fever. (See page 143.) When the blood comes from the large intestine, efforts should be made to arrest its flow by local measures. Ice-water injections, or ice itself, may be introduced into the large intestine; or astringent injections may be employed, especially the injection of a drachm of silver nitrate in two quarts of water. If there be reason to suspect mesenteric embolism, a surgical consultation should be held, and, if circumstances favor, laparotomy may be performed.

Hemorrhage from the rectum must be treated locally.

ENTERITIS.

DEFINITION.—Inflammation of the bowels.

Inflammation of the intestine is characterized by the appearance of an exudation within its wall, upon its surface, or in both situations, and pursues an acute or a chronic course. It is often confounded with diarrhœa, since the causes of each are largely the same, and diarrhœa—frequent loose dejections—is the conspicuous symptom of enteritis. Diarrhœa, however, represents largely an increase of intestinal peristalsis, especially of the colon, in which the intestinal contents remain normally from twelve to twenty hours. The increased peristalsis is due not only to the action of irritants, but also to peculiarities of the individual. Two or three movements daily are physiological for some persons, and an unusually sensitive nervous system often so reacts to mental or bodily excitants that diarrhœa results.

ETIOLOGY.—The principal causes of acute enteritis are to be included under irritation and infection, although the latter usually acts by means of the irritation produced by its products. Conspicuous among the irritating causes are improper food and drink. The food may be improper in consequence of its nature, as unripe fruit, or from putrefactive or fermentative changes such as occur in spoiled meat, fish, fruit, and vegetables, or from excess in quantity or bad cookery. In like manner, milk and milk products may be so altered by the growth of bacteria as to excite enteritis. Various chemical irritants, many of them medicinal in suitable dose, as arsenic, antimony, and mercury, produce enteritis when their action is intensified. Foreign bodies, especially when serving as the nuclei of fæcal concretions, may be productive of inflammation.

The infectious causes of enteritis are those giving rise to cholera, cholera nostras, cholera infantum, and dysentery, in which the enteritis is to be regarded as primary. In typhoid fever, pneumonia, tuberculosis, measles, septic infections, and peritonitis a secondary or symptomatic enteritis is frequent. Acute enteritis also may result from injury, including that due to severe burns. It occurs in the course of nephritis, tuberculosis, diabetes, and other chronic affections. Children, especially infants, are more frequently affected with acute enteritis than adults, largely in consequence of unclean feeding-bottles and of improper food. The prevalence of enteritis in summer is largely due to the favoring influence of heat in the production of putrefaction and fermentation. Importance is often assigned to exposure to cold as a cause of enteritis, but its influence is to be regarded, like that of heat, rather as predisposing and aiding than as actually producing the inflammation.

VARIETIES.—The anatomical varieties of enteritis are the catarrhal, follicular, pseudo-membranous, ulcerative, diphtheritic, phlegmonous, and gangrenous. The lesions are circumscribed or diffuse, and, according to the part of the intestine affected, a distinction is drawn between duodenitis, jejunitis, ileitis, appendicitis, typhlitis, colitis, and proctitis. Such a distinction, however, is practicable for only certain portions of the intestine ; for example, inflammation of the stomach and inflammation of the duodenum are frequently combined, and the disease is regarded as gastro-duodenitis or gastro-duodenal catarrh. Inflammation of the jejunum presents no characteristics by which it is to be differentiated from inflammation of the ileum, and the entire small intestine is often simultaneously diseased ; hence enteritis or ileitis is commonly used to designate inflammation of the small intestine, irrespective of the part conspicuously diseased. In like manner colitis is applied to inflammation of the colon although inflammation of the rectum (proctitis) and inflammation of the cæcum (typhlitis) may be associated. Inflammation of the cæcum rarely occurs as an independent condition ; and although the term typhlitis was originally applied to indicate what was supposed to be an inflammation of the cæcum, the symptoms of typhlitis are now known to be almost invariably the result of appendicitis, or inflammation of the vermiform appendix. Inflammation of the rectum not infrequently occurs without associated inflammation of the colon, although often combined with it. Most important of all the localized inflammations of the intestine is that of the appendix, designated appendicitis. When the inflammation exists throughout the large and the small intestine the term entero-colitis or ileo-colitis is applied.

ACUTE CATARRHAL ENTERITIS.

MORBID ANATOMY.—The changes due to acute catarrhal inflammation of the intestine are redness and swelling of the mucous membrane, with increased secretion. Post-mortem changes produce such alterations in the distribution of blood in the vessels of the intestine that but little importance can be attached to redness as a sign of inflammation unless it is caused by extravasated blood. More important is the swelling of the mucous membrane, chiefly due to œdema, and especially noticeable in the valvulæ conniventes in the upper part of the large intestine. In severe acute enteritis coherent flakes of epithelium are detached and abundantly appear in the liquid contents of the intestine, forming the rice-water stools of choleraic enteritis. The presence of any considerable quantity of mucus is rare in acute inflammation of the small intestine, although a somewhat opaque, perhaps blood-stained, mucus is often found in acute catarrhal colitis or proctitis. In *follicular enteritis* there is swelling of the solitary follicles and Peyer's patches, the individual follicles being surrounded by injected blood-vessels. The follicles are either translucent or opaque, and in severe cases become transformed into abscesses, which break and give rise to ulcers.

SYMPTOMS.—Diarrhœa is the conspicuous symptom of enteritis, although frequent loose movements of the bowels may occur in the absence of inflammation, and enteritis may exist without diarrhœa being present. The number of dejections varies from three or four to twenty and upward in the course of the twenty-four hours, and the quantity of the movement diminishes with the increase in the number of dejections. They contain abundant liquid, either as a result of diminished absorption from increased peristalsis or in consequence of serous exudation from the wall of the intestine. They are either homogeneous or contain particles of undigested food, milk-curds being frequently recognized. The color of the evacuation depends upon the quantity of bile or blood present and the changes undergone by their pigment. The absence of bile gives rise to a colorless or clay-colored dejection, while grass-green stools result from the rapid passage of the bile through the intestinal canal. With less frequent movements the color is yellow or yellowish brown. In simple catarrhal enteritis the presence of blood in the stools is rare. The color is also modified by the nature of the food, being of a lighter hue from a milk diet and of a darker tint when meat-juice is taken. The effect of medicines, especially of bismuth and iron, in blackening the stools is a familiar fact. The consistency of the evacuations varies from that of a thin watery to that of a soft pudding-like material, and, in general, the more frequent the evacuations the thinner is the consistency likely to be. The dejections are not infrequently frothy, from the intimate admixture of bubbles of gas. Mucus is a more constant characteristic of chronic than of acute enteritis, and when present in the latter is usually the result of the presence of the inflammation in the large intestine, in which case the mucus may be blood-stained. The reaction of the stools is generally alkaline, and on microscopical examination, particles of undigested food, epithelial cells, mucous corpuscles, bacteria, and often various crystals are to be found. When parasites are suspected as the cause of the enteritis, search should be made for them and for the eggs.

Abdominal pain is frequent, usually of a spasmodic character, and often associated with rumbling borborygmus as gas or a mixture of gas and liquid is moved along the course of the intestine. The pain is usually referred to the lower abdomen, and, when the large intestine is the seat of the inflammation, may follow the course of the colon or be referred to the region of the sigmoid flexure. The presence of tenesmus is indicative of the seat of the inflammation in the rectum. In the milder cases there is no fever, but in the severer forms of enteritis, especially those of infectious origin, slight or severe chills early occur, and the temperature is elevated two or three degrees. In the severe attacks loss of appetite, thirst, nausea, and vomiting are usually present, but they may be absent. The strength of the patient may be so little disturbed that he is capable of pursuing his daily occupation, or there may be so much weakness and prostration that he is confined to the bed and suffers from symptoms of collapse.

The abdomen is either distended, usually moderately, or flattened, according to the presence of a greater or less quantity of gas and the frequency of the dejections. Tenderness is present only in the severer cases, especially when the inflammation is limited to the colon. Albuminuria and casts have been found in severe cases of acute enteritis, and enlargement of the spleen has been observed. The duration of the attack varies from a few days to a fortnight, or the enteritis may become chronic; irregularity in the action of the bowels for weeks or months is a frequent result.

The acute enteritis of infants has an enormous mortality, especially in the summer among the children of the poor in cities. As already stated, this variety is largely due to the use of unclean feeding-bottles, in consequence of which putrefaction or fermentation of the intestinal contents takes place. The diarrhœa is associated with vomiting. Convulsions are at times present, there is considerable elevation of temperature, the child frequently cries from paroxysms of colic, and the abdomen is swollen and painful. The dejections are often of a greenish color, frothy, and contain abundant curds. This variety of acute enteritis may be soon recovered from or may gradually prove fatal from exhaustion. It, however, may give evidence of involvement of the colon (entero-colitis). In this event the stools are more fæcal in character, but contain abundant mucus, often stained with blood, and the attack pursues the course described under Dysentery. (See page 215.)

The severest variety of acute enteritis in children is known as *cholera infantum.* The vomiting and diarrhœa are excessive, the dejections rapidly become watery, and a condition of collapse readily supervenes. The incipient abdominal pain soon ceases, but painful cramps in the muscles of the extremities may take place. The superficial temperature is often subnormal, although the thermometer in the rectum indicates the presence of fever, and there may be hyperpyrexia shortly before death. The infant lies still and indifferent, the face is pinched, the fontanelles are sunken, the skin is cool and moist, the pulse is small and rapid, and in fatal cases death occurs suddenly, perhaps after a convulsion, or gradually at the end of prolonged coma. The prognosis in cholera infantum is always grave, and death may occur within forty-eight hours after the attack, or at the end of a few days. In cases of recovery convalescence is usually protracted over a period of several weeks.

DIAGNOSIS.—The distinction between simple diarrhœa and enteritis is essentially one of degree. Both may be due to the same causes, but the difference in result is dependent upon the intensity of the irritant, the persistence of its action, and the degree of vulnerability of the patient. In diarrhœa the cause of the increased peristalsis is quickly eliminated, in enteritis it persists. Diarrhœa may be caused by emotional excitement, and merely represents increased peristalsis and consequent defective absorption, the degree of which often varies in individuals. The clinical diagnosis of enteritis demands the presence of diarrhœa in connection with one or more of the cardinal symptoms of inflammation. The character of the stools offers evidence as to the predominant seat of the inflammation in the large or in the small intestine. In enteritis as contrasted with colitis the dejections are more watery, yellow or green, and a sediment forms on standing. In colitis, on the contrary, the dejections are more homogeneous, and contain fæcal matter, either in scybala or flakes, and easily differentiated particles of mucus. Vomiting is a more frequent accompaniment of enteritis, abdominal pain is more frequent in colitis, and tenesmus is the characteristic of proctitis. An excess of indican in the urine in enteritis is indicative of a localization of the inflammation rather in the small than in the large intestine.

TREATMENT.—Acute diarrhœa dependent upon enteric irritation should be treated as acute enteritis, and all irritating or actively astringent drugs are contra-indicated. On the other hand, the sudden diarrhœa of summer, attended with colicky pain, without tenderness, and with free serous discharges, and the paroxysms of serous purging without pain which are often induced by anxiety or other emotions, are usually to be relieved by stimulating prescriptions containing camphor, chloroform, and volatile oils, with or without opium. (See formula 9.) In many of these cases, especially when there is no emotional disturbance and the discharges are very large, watery, and free from color, no remedy will compare in therapeutic activity with mercurials: from one-eighth to one-sixth of a grain of calomel should be given every hour until the passages become greenish or brownish; very commonly when this has happened the diarrhœa ceases spontaneously; if this does not occur, astringent or stimulating local remedies may be expected to act immediately. (See also Cholera Nostras.)

In serous diarrhœas all active exercise should be avoided; indeed, if the symptoms be severe, the patient should be put to bed. Care should be taken to protect the abdomen from cold. The food should be reduced to broths or milk foods. Sometimes partially predigested milk, in the form either of ordinary peptonized milk or of junket, is very suitable. Milk thickened with flour and thoroughly cooked has in our experience acted better than milk thickened with arrow-root or other starches formerly recognized by the United States Pharmacopœia. An excellent food which is very binding is made by filling a pint bag tightly with flour, sewing it up, throwing it into boiling water, and after five or six hours of cooking taking out the contents, cutting off the outer sodden rind and grating the inner baked core, and then incorporating the gratings with very hot milk.

The treatment in acute enteritis must vary in its rigor with the severity of the disease. In pronounced cases the patient should be put to bed and confined to a diet composed of animal broths, milk, and milk foods. (See above.) In milder cases tender meats may be allowed, but broken or ground grains and vegetables are to be forbidden. Rubefacients often distinctly relieve pain, and are to be freely used. In bad cases a poultice containing from ten to twenty per cent. of mustard should be placed over the whole abdomen, or the spice plaster be used. As a substitute for the latter, spongiopiline, or a cloth, may be wrung out of a tincture made by macerating one ounce each of Cayenne pepper, cloves, and allspice in a pint of alcohol; the cloth may be covered with oil-silk, and is often preferred to the poultice on account of its lightness. A flannel bandage should be worn both day and night when there is no other local application to the abdomen. Hot-water bags are often very comforting.

In the beginning of the attack, especially if the passages are small and contain abundant mucus or if there is a history of the recent ingestion of indigestible food, a full dose of castor oil and laudanum may be given, or one-fourth to one-sixth of a grain of calomel may be administered every two hours until free bilious discharges are produced. A mixture of equal parts of castor oil and aromatic syrup of rhubarb (U.S.P. 1880) given in small doses every hour sometimes acts most happily. After the purgatives have acted, a mixture of bismuth and carbolic acid (see formula 5) should be exhibited; with it may be combined, as a slightly astringent antacid, simple prepared chalk. When with the continuing diarrhœa there are persistent tenderness and no improvement under the use of bismuth and carbolic acid, silver nitrate with extract of opium or of hyoscyamus may be given in pill covered with a thick capsule. Salol is often advantageous as a sedative to the intestinal mucous membrane and as an intestinal antiseptic. When there are marked evidences of fermentation, or when there is much flatulence, naphtol (two grains) or strontium salicylate (three to five grains) may often be combined advantageously with the bismuth and carbolic acid.

The diarrhœa which exists in enteritis seems naturally to indicate the use of astringents, and when the discharges are very free this apparent indication grows correspondingly in force. It must, however, be remembered that all vegetable astringents are irritant, and that the discharge from the bowels which is provoked by inflammation usually does more good by relieving the local disease than it does harm by exhausting the patient. The effort of the practitioner should be to cure the condition which provokes the diarrhœa, and not to arrest the diarrhœa. If an astringent seems to be necessary to control excessive diarrhœa, lead acetate is more sedative and less irritant than the vegetable astringents. In the latter stage of the disease the condition of the intestines may be one of relaxation following acute congestion and inflammation, so that an astringent is really indicated: under these circumstances the sulphuric acid mixture (formula 6) will usually be found to act happily, or the chalk mixture (formula 8) may be tried.

Treatment of Acute Diarrhœa of Young Children.

The acute diarrhœa of childhood is for therapeutic purposes divisible into two classes of cases,—those which are typified in acute enteritis and those which are typified in an acute cholera infantum; the one with small mucous discharges, the other with large serous passages. We think every practitioner who has seen much of infantile diseases will recognize that the line between the cases, considered as a whole, is not sharp, but that the two typical forms grade into each other. Nor yet, in accordance with our experience, is the etiological difference in the two classes absolutely diverse. The typical cholera infantum and the typical enterocolitis of the child may be produced in different children by similar causes. In the summer the chief cause of severe and fatal diarrhœa among young children is elevation of temperature. The mortality-curve among children in our great cities closely follows the temperature-curve; the rise of temperature to 100° F. at mid-day, continuing for several days, always being soon followed by a rise in the mortality-rate.

It is evident, further, that the eating of improper food, sour milk, bad fruits, stale vegetables, fermented and altered by heat, is a fruitful cause of the summer diarrhœa of children, and that an increase of the general temperature, by increasing the activity of change in animal and vegetable foods, augments the danger to the poor of great cities of bowel complaints from improper ingesta.

In the clear recognition of the vitality of these two etiological facts lies, we believe, the secret of the proper management of the acute infantile diarrhœas under consideration. If the disease occurs during the hot spell, and the temperature of the child is above the normal, the reduction of the temperature by the cold-water or cold-air bath affords the one rational method of treatment. The temperature of the young child should always be taken in the rectum, as the internal temperature may be very high at a time when the external or axillary temperature is comparatively low. Again, the sudden unconsciousness, with disturbed respiration and other evidences of cerebral paralysis, which frequently ends the scene in cholera infantum in the summer months, is due to hyperthermia. Early reduction of the temperature by the use of cold in such cases is followed by immediate improvement of the nervous symptoms. Peevishness and prostration during the day, with intense restlessness at night and convulsive startings, are often put an end to by cold bathing. The bath should be repeated every two to four hours, should be at a temperature of 90° F., cooled to 80° F. if necessary, and should be sufficiently prolonged to reduce the temperature. From time immemorial the effects of change of air in the treatment of cholera infantum obtained by taking the child to the sea-shore or over some large body of water have been considered most remarkable. The effects reached have really been due more to change of temperature than to change of air, and are not, therefore, so mysterious. Steady cooling by constant immersion in cool air is, of course, when obtainable, to be preferred; but intermittent cooling by occasional immersion in cool water is often the best that can be obtained. When the cholera infantum patient can be sent to the sea-shore it should be done; but probably the shaded country spring-house would be nearly as effective as the cool sea-breezes. An electric fan forcing air over suspended blankets kept continually wet we have known apparently to save life when a fevered patient could not be taken out of the heat. The hydrencephaloid congeries of symptoms with which is especially associated the name of Marshall Hall are in some cases of infantile diarrhœa the outcome of exhaustion, to be met by stimulants and concentrated digestible foods. As they occur, however, in children in American cities during summer months, they are usually the result of hyperthermia. Whenever the temperature reaches 101° F. the child should be bathed in tepid water; at 102° F. cool bathing is urgently needed; at higher temperatures failure to use the bath is a crime. The rise of the bodily temperature in these cases is no more due to inflammation or a measure of the severity of the intestinal irritation than is the high temperature of a stevedore who falls sunstruck on the wharf where he labors. Large injections of ice-cold water are very serviceable, acting locally well and aiding in reducing the general temperature.

As in many cases of acute diarrhœa occurring in the young child the intestines contain a fermenting mass of undigested food and disordered secretion, it is commonly well to commence the treatment by a full dose of castor oil or a mixture of equal parts of castor oil and spiced syrup of rhubarb. When the passages are very large and serous the purgation may be omitted. Small doses of calomel (one-twelfth to one-eighth of a grain) may be given every two hours, with or without three to five grains of bismuth subnitrate, according as the practitioner believes that the attack is or is not accompanied with pronounced irritation or inflammation of the mucous membrane. Carbolic acid, by arresting fermentation and by its anæsthetic influence on the intestines, often acts happily when conjoined with the bismuth. The vegetable astringents are almost always useless remedies, and frequently do harm. Opium is often indispensable, but should be used with the greatest caution, and with the full remembrance of the fact that the momentary checking of a symptom is not curing the disease. Quinine, strychnine, and all other remedies which are capable of irritating the alimentary canal are harmful in most of these cases. Drugs like strychnine and digitalis may be used in times of collapse, but under such circumstances should be given hypodermically. Alcohol is often indicated, and, given sparingly in the form of fine old brandy, is incapable of doing harm, except it be in those cases in which the local inflammation is severe. Salol, naphtol, strontium salicylate, and other drugs which act as intestinal antiseptics are often of service, especially in those cases in which the discharges are not sufficient to clear out thoroughly the alimentary canal and in which there is reason for believing that fermentation is going on within the intestines. When the passages are serous and very large, or when, as in a typical cholera infantum, they are apparently paralytic in origin, camphor, chloroform, and the volatile oils may be used cautiously as in other forms of nervous diarrhœa.

The feeding of a child suffering from an acute summer diarrhœa is a matter of the gravest importance. In severe cases the only thing allowed at first should be water rendered albuminous with the white of egg: the white of one or two eggs should be thoroughly shaken with a pint of water; in many cases brandy should be added to this solution. Animal broths, especially chicken broth, without rice or other farinaceous ingredients, or pure beef-juice obtained by expression, should be the first foods given after the albuminous water. Wine whey sometimes acts well. Milk, if employed at all, should be of absolute purity and freshness when possible; if not, it should be sterilized. Pure milk should be partially predigested. Junket is probably superior to simple milk, it being, indeed, milk in an early stage of digestion. Not rarely lime water may be added to the milk, in the proportion of one to four or one to eight, with advantage: by delaying coagulation in the stomach it tends to prevent the formation of hard curds. In any case of acute intestinal diarrhœa occurring in a breast-fed child there is no food superior to the breast-milk, if it be of good quality; it may be right for a few hours to confine the child to the albuminous water, but the nursing should not long be interrupted. If, however, as is often the case, excessive thirst leads the child to drain the breast too frequently and too thoroughly, due caution must be exercised to see that there is no overfeeding.

CHRONIC ENTERITIS.

Definition.—Chronic inflammation of the mucous membrane of the small intestine, very frequently also involving that of the large intestine.

Chronic enteritis is anatomically divided into chronic catarrhal enteritis, in which the inflammatory change is of the catarrhal type; pseudo-membranous enteritis, in which the inflammation is accompanied with the formation of large quantities of mucoid secretion, taking the form of membranes or casts; and ulcerative enteritis, in which the formation of ulcers is the conspicuous feature.

CHRONIC CATARRHAL ENTERITIS.

Chronic catarrhal enteritis is either the result of acute enteritis or is chronic from the outset, in which case inspissated fæces, ulcers, and tumors of the intestine, intestinal parasites, chronic passive congestion of the portal system, and chronic wasting diseases are important in the etiology.

Morbid Anatomy.—The mucous membrane is swollen, opaque gray, perhaps slate-colored from the presence of modified blood-pigment in the interstitial tissue. The pigment often lies in the villi and in the vicinity of the follicles. The mucous membrane is thickened from an increase of its fibrous tissue, which may be diffused or cause polypoid projections from the surface or extend into the submucous and muscular coats. The outlet of the glandular crypts may be obstructed and dilatation of the ducts result. There is an abundant formation of mucus, especially in the large intestine, and it may be copious in the small intestine, in which normally little or no accumulated mucus is to be found. Occasionally, especially in children, the mucous membrane becomes atrophied and the cryptic glands to a large extent are destroyed.

Symptoms.—In chronic enteritis there is a frequent alternation between constipation and diarrhœa. The daily number of loose dejections is in the vicinity of half a dozen, and pain may or may not be associated with the movements, but the patient often complains of a sense of weight and discomfort in the abdomen. The dejections contain abundant mucus, and not infrequently a slimy dejection with but little fæcal matter is followed by a scybalous movement, or mucus and fæces are intimately mixed. When the abdomen is distended and tympanitic, borborygmi are often heard. There may be tenderness on palpation. There may be but little disturbance of nutrition, or marked emaciation may exist. In children there are likely to be extreme emaciation and weakness. Adults suffering from chronic enteritis are often irritable or depressed, easily fatigued, and frequently hypochondriacal. Chronic enteritis is of long duration in the adult, with periods of exacerbation and remission, at times prolonged intervals of freedom existing. In young children and in elderly persons it may prove a cause of death from progressive exhaustion.

TREATMENT.—The hygienic management in a case of chronic catarrhal enteritis is extremely important, and must be attended to in the minutest details by the practitioner. At no time should any chilling of the surface of the body be allowed ; a heavy, well-fitting woollen or silk abdominal bandage is vital, and should be worn continually, it being changed day and night. A woollen or silk long-sleeved under-vest or under-shirt must be worn day and night ; the ankles also should be well protected from draughts, the patient on no account being allowed to put the naked foot upon even a carpeted floor, and in cool weather shoes should be worn instead of slippers even in the house. The drinking must be carefully attended to. Ice-cold liquids are to be forbidden, no sweet drinks are to be allowed, and wines are in large part to be excluded from the dietary. In some cases the stronger wines, such as port and madeira, may be given very sparingly ; but ordinarily if any alcohol is taken it should be in the form of well-diluted spirit, pure brandy being usually preferable to whiskey. Coffee should not be taken at all ; tea may be used in moderate quantities. Sometimes it is advantageous to confine the patient temporarily to a skim-milk diet. In other instances it is better to allow Hamburg steaks or broiled or baked tender meats,—veal, turkey, pork, and tame duck being absolutely forbidden. Starchy foods are rarely allowable ; toast or pulled bread may be given, but potatoes and the various farinaceous dishes are contra-indicated. Macaroni stewed in milk without cheese agrees with most cases, and rice may be used if necessary to satisfy the craving for vegetables. Generally no vegetables should be taken. Custards, and bread and other simple plain puddings, without much sugar, are to be put on all except the strictest diet-lists. Eggs cooked (not fried) or raw may be given in moderate amount.

The amount of exercise allowed must be carefully suited to the individual case. Not rarely there is pronounced exhaustion, and rest in bed with massage is essential. On the other hand, an old enteritis is sometimes very happily affected by carefully graded exercise.

In most cases the discharges can be temporarily arrested by the use of astringent remedies, but this arrest of the diarrhœa is followed rather by an increase than by a betterment of the intestinal condition. Astringents are not curative, are very capable of doing harm, and when largely and actively employed are always an evil. The acid diarrhœa mixture (see formula 6) is the most serviceable and least harmful that we have ever employed. Guarana, twenty grains an hour after meals, is sometimes well borne. Certain astringent or alterative mineral waters, especially the Oak Orchard Acid Spring water, and when there is much intestinal indigestion the Franzenquelle water of Europe, may be of service. In many cases of catarrhal enteritis purgatives are from time to time indicated, and complete irrigation of the colon is often very useful. Among purgatives the castor oil and glycerin mixture is usually to be preferred; occasionally small doses of calomel act well. The enemata should be two quarts of simple water, or may often with advantage be medicated. Occasionally scybalous masses will be found in the stools produced by the enemata, fæcal retention being not impossible in chronic enteritis; the entire removal of such bodies from the alimentary canal is essential to recovery. In other cases, even when there is considerable diarrhœa, black, slimy, mucoid discharges are provoked by the injections, so that it would appear as if the large intestine was covered by a fermenting mass of old retained secretions and fæcal matter, through which ran a central current. In such cases the medicated injections, especially the silver nitrate (ten grains to two quarts), should be repeated at intervals of from two to six days until the bowel is cleansed.

For the purpose of directly affecting the inflamed mucous membrane, the most effective remedy we know of is the tar-water mixture (see formula 22): a wineglassful (a fluidounce and a half) should be given from one to two hours after each meal. Silver nitrate is much used, but has afforded us little satisfaction; if given at all it should be in pill enclosed in a double capsule, so as to avoid as far as possible its destruction before it reaches the intestine. Bismuth, especially bismuth with carbolic acid, or the combination of bismuth, naphtol, and carbolic acid, is valuable. Other aromatic products than tar, such as turpentine, oil of cubeb, or oil of copaiba, may be used in various cases. Any remedy given for the purpose of affecting the mucous membrane of the small intestine should be taken from one to two hours after eating, at a time when the current is naturally setting from the stomach into the intestinal tract.

Chronic Diarrhœa of Young Children.

Chronic intestinal catarrh of infancy is in the great majority of cases the result of improper feeding. When it occurs in breast-fed infants the milk of the woman should always be looked upon with suspicion; although simple irregularity of feeding, and especially excessive frequency of feeding, will sometimes derange the digestion of the feeble child. No artificial food has as yet been made that compares, in its accord with the digestive system of the infant, with human milk; and therefore, when chronic catarrh in the bottle-fed child does not yield to careful treatment, it may be essential to procure a wet-nurse, even if the proper sustenance of the child of the rich parents involve the improper feeding of its less fortunate *confrère.*

The proper hygiene of the wet-nurse should always be looked after. Much harm is often done by pampering. Accustomed to a life of plain food and hard work, the wet-nurse is often overfed, under-exercised, and suddenly in every way led into a life of luxurious ease that cannot do otherwise than derange her general system.

It is evident that very many children must be brought up upon artificial food. In attempting this the child should, if it be possible, live rather in the country than in the town; good being achieved not only by the fresh air and out-door life of the country, but also by the freshness and purity of the staple food of infantile life, milk. The most generally applicable substitute for human milk is that of the cow, which is usually believed by experts to be improved by a process of sterilizing,—heating for fifteen to twenty-five minutes at a temperature of 165° F. to 170° F.,—but which is probably injured by boiling. In our largest cities the attempt is being made to furnish to physicians milk which has been modified by the subtraction or addition of various substances, so as to render it more closely allied in its composition to human milk; indeed, it is proposed that the physician shall order for the individual babe milk of a certain composition to suit its especial needs. It is, however, very doubtful whether any of these refinements of chemical activity yield products which equal absolutely fresh milk obtained by having the cow within a few hundred yards of the infant. For the first three months of the babe's life the milk should be diluted with two parts of boiled water; for the second three months of life equal parts of boiled water may be added; after this half as much water may be put with the milk; and after nine months the milk can be given undiluted. Excessive dilution sometimes leads to curious results, as in a temporarily baffling case in which the only symptoms were that the child was constantly crying, constantly taking food, and constantly urinating; the whole difficulty was at last found to be the excessive dilution of the milk, which caused the starved child to cry and drink from excessive hunger, and to urinate to get rid of the water. When any intestinal catarrh refuses to yield to treatment whilst the child is being carefully fed with cow's milk, artificial foods may be tried. Of these the most popular are largely composed of grape-sugar. A study of the various artificial foods, sufficiently detailed to be of value, would far exceed the limits proper in a volume like the present, and the reader is referred especially to works on diseases of children.

The hygienic management of the child suffering from chronic intestinal catarrh is important. It should be daily bathed in cool—not cold—water, should in the summer season be protected from the heat and in the winter from the cold, and should at all times wear a woollen abdominal bandage.

The medicinal treatment of chronic intestinal catarrh in childhood can be outlined in a few words. The most important principle is to avoid all astringent remedies as far as possible, and to attempt to cure the catarrh, and not the diarrhœa which is its symptom. Mercurials are of value. Minute doses of calomel or of gray powder may occasionally be given for several days at a time with advantage. Bismuth subnitrate is much used, and is often temporarily of value; very frequently it may be given with advantage associated with chalk (five to ten grains of each three to four times a day). The intestinal antiseptics are important: salol, creosote, carbolic acid, naphtol, strontium salicylate, may be used from time to time, alone or in combination with bismuth, often very advantageously. The one drug, however, which we have seen yield the most beneficial results is sodium phosphate; it is rather laxative than astringent, but evidently favorably modifies the intestinal secretions. From five to ten grains of it should be given with each bottle of milk or immediately after the taking of the food.

PSEUDO-MEMBRANOUS ENTERITIS.

This variety of chronic enteritis has been designated also *pseudomembranous colitis* and *mucous colic.* It oftenest occurs in neurasthenic or hypochondriacal persons, usually in women, and sometimes in children. It is possible that two distinct conditions exist, the one inflammatory, the other a neurosis. The essential characteristic is the discharge from the intestine of a gray mucus, translucent or opaque, in the form of membranes or of cords, sometimes a foot or more in length, and of tubular casts of portions of the intestine, often discolored by the intestinal contents and even by blood. On microscopical examination the membranes contain epithelium, degenerated or not, and a homogeneous intercellular substance, the basis of which is either mucin or other albuminoid substance. It is only in rare instances that the membranes have been observed *in situ,* and in these they were found in the colon, the mucous membrane of which presented no distinctive alteration.

Symptoms.—Pseudo-membranous enteritis is characterized by attacks of colic followed by the evacuation of the typical discharge. The attacks of colic may last for several days, when relief is experienced, and intervals of months may elapse without a recurrence of the symptoms. There is no obvious exciting cause for the immediate attack, but with the repeated occurrence of the attacks depression of spirits, hysterical manifestations, and neurasthenic symptoms are frequent. The general nutrition and appearance of the patient may be but little affected. Pseudo-membranous colitis or mucous colic is usually an affection of long duration, and treatment is generally of but little avail. Exceptionally the disease has come to an end after one or a few attacks. Errors in diagnosis are likely to arise only from mistaking the remains of undigested food or vegetable or animal tissues for the characteristic membrane, but microscopical examination enables their nature to be quickly determined.

TREATMENT.—For therapeutic purposes cases of pseudo-membranous disease of the intestine may be divided into—first, those in which there is habitual constipation; second, those in which there is a tendency to relaxation of the bowels; third, those in which constipation and diarrhœa alternate. The management of these varieties of the disease differs, but at the same time has much in common.

In every case the hygienic management must be in accord with the general condition. Probably in the majority of bad cases there is a more or less pronounced neurasthenia, under which circumstances the rest-cure should be enforced with a rigor proportionate to the needs of the individual. Sometimes, even from the outset, graded increased exercise is required. Under all circumstances the abdominal bandage should be used day and night, and care should be taken to see that it is well fitting and continually in place. The bathing habits should be looked after, —the daily cool or tepid bath being employed as indicated.

The diet should be carefully watched, and should be nearly the same as in chronic enteritis. The experience of the individual as to what does not agree with the digestion should be thoughtfully consulted. Sugar should be reduced to the minimum. Oatmeal should be denied, though corresponding wheat-foods are in some cases suitable. Potatoes, beets, and other vegetables which grow under the ground should be strictly forbidden; whilst spinach, young peas, or Lima beans may be sparingly eaten. Macaroni cooked without cheese, rice, and milk foods are usually suitable, and plain puddings and custards may sometimes be allowed. Hot bread and griddle-cakes are to be interdicted, and even stale bread must be used sparingly; pulled bread and toast are preferable. Tea may be allowed, but coffee and chocolate are on the doubtful list. Alcohol in any form should be used with caution; malt liquors are especially injurious.

During the paroxysms of exacerbation the patient should be kept quiet, even confined to bed if not robust, should use free counter-irritation along the whole length of the colon by means of iodine or sometimes even of flying blisters, and should take full doses of castor oil until its effects have become manifest, at the same time using large injections as spoken of in the following paragraph. Relief cannot be expected until the membranous masses are thrown off. Between the exacerbations the treatment varies with the case. The tar mixture (formula 22) may be given continuously for weeks in every form of the disease. When there is distinct diarrhœa, carbolic acid and bismuth (formula 5) are very useful. No astringent should be employed more severe than the sulphuric acid mixture, with occasionally guarana after meals. When there is constipation it is essential that the bowels be kept freely open day after day, and no hesitation should be felt in the use of laxatives. In many cases the senna mixture (formula 20) acts happily. If it does not suit, or if it has been taken for months and is losing its power, the daily use of the glycerin and castor oil mixture (formula 24) will in some cases be very effectual. These laxatives should, however, be varied: the sodium phosphate mixture (formula 19), cascara sagrada, the A.B.S. (aloes, belladonna, and strychnine) pill of the hospitals, and various combinations of the vegetable cathartics, with eserine, may from time to time be used. Many individuals are greatly benefited by taking sweet oil after each meal, a dessertspoonful to two tablespoonfuls, with or without one to two teaspoonfuls of whiskey. Any derangement of digestion by the oil must be the signal for its withdrawal.

Perhaps the most important part of the treatment in these cases is the habitual use of large enemata, which at first may be employed three or four times a week, afterwards once a week, or at irregular intervals, according to the necessities of the case. Two quarts of water, at 105° F. in the receptacle, variously medicated, should be given at a time. The remedies used in these enemata should vary as do local applications to mucous membranes in other portions of the body, and should have about the same range. Solutions of common salt or borax (varying from two per cent. to saturation, *pro re nata*) may do good. The most generally useful is silver nitrate (five to ten grains to the quart). Often it is better to give the silver injection at intervals of a week or more, with milder injections between. Judgment as to the effect of these injections is to be made by noting the character of the passages produced by them, the pain at the time of the injection, the tenderness along the colon after the injection, and the effects upon the passages for the next few days. In most cases it is essential to get rid of the mucus; increase of the mucus, however, for several days after the injection, especially if it be associated with increased colonic tenderness, is evidence of irritation of the gut.

ULCERATIVE ENTERITIS.

Ulcers of the intestine arise usually in the mucous membrane, but sometimes develop in the serous coat, and are the result of a variety of causes. Many of them are of merely secondary importance, while others are the conspicuous characteristics of the disease in which they occur. The ulcers occurring in acute and chronic infectious diseases, as typhoid fever, dysentery, tuberculosis, and syphilis, in constitutional affections, as scurvy, gout, diabetes, malignant disease, and especially cancer, and those due to sharply defined causes, as strangulation of the bowel in acute intestinal obstruction, or localized disturbances of circulation, as ulcers of the duodenum, and those from thrombosis and embolism, are mentioned elsewhere. Those due to disturbances of innervation and to amyloid degeneration are of such rare occurrence as to be of little clinical importance.

As a result of catarrhal enteritis two varieties of ulcer occur. The one, called catarrhal, extends from the surface downward; the other, the follicular, proceeds from an abscess of the lymph-follicle in the intestinal wall. The *catarrhal ulcers* are found especially in the large intestine, may be few or many, and when widely distributed tend to become confluent and give rise to extensive loss of substance. Islets of mucous membrane remain perhaps undermined or with polypoid projections. Extension in depth may lead to inflammation of the mesocolon, or even to perforation of the bowel. Healing of the catarrhal ulcer is possible, although unlikely to take place when they are numerous.

The *follicular ulcer* usually occurs also in the large intestine, though sometimes present in the ileum. It represents one of the results of follicular enteritis, in which the inflamed lymph-follicles become abscesses and are discharged into the intestines: hence the ulcer from the outset is deep-seated. The wall is early undermined, and extensive destruction of the mucous membrane results, when the numerous follicular ulcers become confluent. The longer the process continues the less possible is it to distinguish by the anatomical appearances between catarrhal and follicular ulcers: indeed, many writers consider that they are essentially the same.

Stercoral ulcers occur in various parts of the large intestine in consequence of long-continued retention of inspissated fæces, especially when containing lime salts and forming fæcal concretions. This variety of ulcer is of especial importance in appendicitis. Formerly ulceration of the cæcum was thought to be a frequent result of retained fæces, but it is now recognized that most ulcerations of the cæcum of a non-tubercular or cancerous nature are due to a perforation from without of a peritoneal abscess caused by appendicitis. Multiple and small ulcers resulting from the irritation of retained fæces are sometimes observed at the flexures of the large intestine. They tend to girdle the intestine, and in healing may cause stricture.

Amyloid ulcers have been described by a number of observers, although their occurrence is to be regarded as extremely rare. Indeed, considering the frequency of amyloid degeneration of the intestine and the infrequency of this variety of ulcer, it would seem as if the loss of substance might be the result of conditions of which the amyloid degeneration is a complication. It is not unlikely that ulcers originally due to tuberculosis or syphilis, in which amyloid degeneration is frequent, may have been attributed to this affection of the intestine.

SYMPTOMS.—Ulcers of the intestine, whatever may be the cause, have but few characteristic symptoms; indeed, extensive ulceration of the intestine may exist and there be no symptoms indicative of this lesion. As a rule, the more numerous and the larger the ulcers, the more likely is diarrhœa to be present, and the course of the affection is that of a mild or a severe form of acute or chronic catarrhal enteritis. When the ulcers are limited to the large intestine the condition is regarded often as a catarrhal dysentery. Ulcers may occur in the large intestine with either no diarrhœa or with alternating constipation and diarrhœa. Pain is an inconstant symptom, but when complained of it is colicky in character and produces persistent discomfort. If sharply localized and associated with constant tenderness in the region concerned, it is suggestive of the extension of the ulceration to the vicinity of the peritoneum. Ulcers are more especially indicated by the discovery of blood, pus, or shreds of tissue in the dejections, but the hemorrhage is often so slight as not to be detected even on microscopical examination. In like manner pus, though still more important as a characteristic of ulcer, may be so small in quantity as to be overlooked. Large quantities of pus in the intestinal evacuations are less suggestive of ulceration than of the perforation of a neighboring abscess into the intestine. Shreds of tissue are absolutely characteristic, but are rarely found, except in the rapidly progressing ulcers of acute dysentery.

The effect of intestinal ulcers upon the general nutrition is often slight, unless they are numerous in the small intestine or there is extensive destruction of the mucous membrane of the large intestine. More important in the progress of the ulcers is the occurrence of perforation, the immediate significance of which depends largely upon its seat. If it takes place along the line of attachment to the mesentery, a mesenteric abscess results; if at the part of the bowel immediately covered by peritoneum, general peritonitis is likely to follow. Embolism of the portal vein or stricture of the bowel at times follows ulceration.

TREATMENT.—The treatment of ulcers in the small intestine is practically that of chronic catarrhal enteritis. When the ulceration is in the large intestine the general management of the case is that of chronic enteritis; but the main reliance must be upon local treatment. Intestinal antiseptics are chiefly of value as they remedy complicating conditions of the upper bowel. Bismuth and perhaps the tar preparations, when given by the mouth, may to some extent reach the large intestine.

The most remarkable effects are at times to be obtained from a large injection, two quarts of water containing from one-half to one drachm of silver nitrate. Such injection may be repeated in three or four days, and perhaps a third or even a fourth time at intervals of a week. Between these injections the bowel may be washed out with a saturated solution of borax, not oftener than once in three days. It would appear probable that in obstinate cases various other local applications besides the silver nitrate might be advantageously applied to the intestine, notably weak solutions of zinc sulphate, or fluid extract of hydrastis, or mixtures containing bismuth subnitrate; in our experience, however, no other local application has approached the silver salt in effectiveness.

DIPHTHERITIC ENTERITIS.

This variety of inflammation of the intestine is characterized by superficial necrosis of the mucous membrane, the production of which is probably intimately connected with the presence of bacteria and with fæcal retention. The alterations are found at first in particular portions of the intestine, especially of the large intestine, and on limited parts of the surface, but rapidly extend in width and depth.

A primary and a secondary diphtheritic enteritis are recognized. The former is the especial characteristic of diphtheritic dysentery; the latter is the result of various infectious diseases, especially of pyæmia and septicæmia of puerperal or of non-puerperal origin, cholera, typhoid fever, scarlet fever, and variola. It also occurs in certain chronic diseases, as tuberculosis, nephritis, cancer, and diabetes, and has been observed as a result of poisoning with corrosive sublimate.

The anatomical appearances are sufficiently described in the article on dysentery, page 214, in which disease a diphtheritic colitis is frequent.

The symptoms of a primary diphtheritic enteritis are those of dysentery. There may be no symptoms indicative of secondary diphtheritic enteritis, the lesions often being found unexpectedly at a post-mortem examination. On the other hand, especially in uræmic and mercurial cases, diarrhœa, colic, and even tenesmus, may be present, but the stools are usually free from blood. The presence of a secondary diphtheritic enteritis may be inferred when symptoms of a severe colitis occur in connection with the above-mentioned causes.

TREATMENT.—The treatment of diphtheritic enteritis is largely that of its cause, with the addition of such general and local measures as have been described under the head of acute and chronic enteritis and of dysentery.

PHLEGMONOUS AND GANGRENOUS ENTERITIS.

When the mucous membrane is infiltrated with pus, the condition is known as *phlegmonous enteritis*. It is of rare occurrence, and may be the result of a primary infection of the wall, as in malignant pustule. More often it occurs in consequence of ulcers, intestinal obstruction, strangulated hernia, or fæcal impaction. The symptoms are those either of a severe enteritis or of a peritonitis.

Gangrenous enteritis occurs when putrefaction of the necrotic mucous membrane occurs. It therefore represents a stage in the progress of ulcerative, diphtheritic, or phlegmonous inflammation of the intestine. It is oftenest present in dysentery, and is indicated by the discharge of discolored sloughs of an extremely offensive odor with considerable blood. Its further consideration is to be found in the article on dysentery, page 214.

Phlegmonous and gangrenous enteritis must be looked upon as secondary or complicating disorders, for which there is no other treatment than that of the original cause, with the use of opiates or laxatives or astringents and of various local remedies to meet symptoms as they arise.

APPENDICITIS.

The importance of recognizing the vermiform appendix as the usual source of the inflammations in the right iliac fossa, whether designated *iliac abscess*, *iliac phlegmon*, *typhlitis*, *perityphlitis*, *paratyphlitis*, or *typhloenteritis*, led Fitz to offer the term appendicitis to indicate the primary disease whose results were so variously named. Despite the barbarism of the term, its practical importance has made it welcome. He showed by the comparison of a large number of cases of perforation of the vermiform appendix with those receiving the clinical diagnosis of typhlitis or perityphlitis that the symptoms, course, and results of all had so many points in common as to indicate that inflammation of the vermiform appendix was the essential feature; "that, for all practical purposes, typhlitis, perityphlitis, typhlitic tumor, and perityphlitic abscess meant inflammation of the vermiform appendix; that the chief danger of this affection is perforation; that perforation, in the great majority of cases, produces a circumscribed suppurative peritonitis tending to become generalized."

ETIOLOGY.—According to Toft, the vermiform appendix was found diseased in one hundred and ten out of three hundred post-mortem examinations, and Hawkins found a like condition in sixteen out of one hundred autopsies. The causes of the great frequency of inflammation of the appendix which is indicated by these figures are due both to congenital peculiarities of structure and to conditions acquired after birth. Among the former are unusual length and abnormal position of the appendix, and irregularities in the development of its mesentery, which abnormities tend to favor the accumulation of material within the canal. The important causes acquired after birth are adhesions due to a localized peritonitis, either proceeding from the appendix or arising elsewhere in the abdomen, in consequence of which the appendix becomes adherent and is prevented from expelling its contents. Most important of all is the presence of fæcal concretions or foreign bodies, the former being found in about one-half and the latter in at least one-quarter of the cases. Moulded, inspissated fæces, however, are often found in a normal appendix, and therefore are to be regarded rather as a favoring than as the exciting cause of the inflammation. The same is true, though to a lesser degree, of the foreign bodies, which are various, and include seeds, bristles, worms, shot, beans, pills, and gall-stones. Digestive disturbances, or a strain or jar, such as may take place in lifting, jumping, falling, or from a blow, are of etiological importance in at least one-third of the cases. Usually, however, an attack begins without any obvious exciting cause. Appendicitis occurs oftener in males than in females, and especially in healthy youths and young adults, although it has been observed in an infant of twenty months and in a person seventy-eight years of age.

MORBID ANATOMY.—The varieties of inflammation which may be found in the appendix are the catarrhal, ulcerative, and gangrenous, each of which may be circumscribed or diffuse. The catarrhal and ulcerative forms of inflammation are acute or chronic, and end in resolution, perforation, stenosis, or obliteration, while the gangrenous variety always ends in perforation. The appearances of catarrhal appendicitis are the same as those of catarrhal inflammation elsewhere in the intestine. But the tendency of all inflammation of the appendix is so strong to a rapid extension to the submucous, muscular, and peritoneal coats that the term infectious has been suggested by Morris to indicate the nature of acute appendicitis. When the appendix is removed within twenty-four hours after the onset of the symptoms, it is found often reddened and swollen throughout, with a cellular exudation in its wall. Ulceration of the mucous membrane of the appendix may be the result of a catarrhal inflammation and occur in the absence of a concretion or foreign body, even without symptoms, and is occasionally found in chronic appendicitis. The base of the ulcer is formed by the submucous or muscular coat, and the surrounding mucous membrane is opaque gray and covered with mucus. The ulcer may heal, or, gradually extending in depth, eventually lead to perforation and the production of a circumscribed appendicular peritonitis. Stricture or partial or complete obliteration of the canal, with dilatation beyond the point of obliteration, may follow healing of the ulcer. Gangrenous appendicitis is of the greatest gravity, from the constancy with which it is associated with perforation, and in this variety a concretion or foreign body is often present. Gangrene and peritonitis, however, may result in the absence of foreign bodies or ulcer from the invasion of the wall of the appendix by bacteria present in the intestinal contents. The wall in contact with or in the vicinity of the concretion or foreign body is thin, of an opaque gray or greenish-yellow color, and often surrounded by a sharply defined line of demarcation. One or more openings, either pin-hole in size or large enough to admit the passage of a pea, are to be found when perforation exists, and the rest of the appendix may show but little alteration or may be reddened and swollen even to the size of the little finger. Frequently the entire appendix becomes detached, forming a slough. Chronic appendicitis is manifested by an enlargement of the appendix either from a thickening of its walls, especially of the mucous membrane, which is opaque gray and corrugated, or from hypertrophy of the muscular coat. The peritoneum likewise is often thickened and opaque, either throughout or in patches. The altered appendix may lie free in the abdominal cavity, or be adherent to the surrounding parts, or be embedded in dense fibrous tissue, often causing the thickened appendix to follow a tortuous course.

A localized peritonitis is the usual result of the severer forms of appendicitis, and is manifested at the outset by a dull, velvety surface of the appendicular peritoneum. Fibrinous exudation soon makes its appearance as a gray or yellowish-gray membrane, which can be readily stripped from and sometimes forms a mould of the appendix. This fibrinous exudation causes adhesions between the appendix, the coils of intestine, and the abdominal wall. Soon a liquid exudation appears around the appendix and more or less rapidly increases in quantity. At first it is serous or fibrino-serous and is slightly opaque, but as it increases in quantity it becomes an opaque yellow pus, which, from its usual presence in the immediate vicinity of the cæcum, has been designated perityphlitic abscess. This circumscribed appendicular peritonitis, however, is to be found in the neighborhood of the appendix, and, according to the position of the latter, may be present in the region of the right kidney, the gall-bladder, the navel, the left iliac fossa, the hypogastrium, Douglas's fossa, or the inguinal canal. The peritoneal abscess may become large enough to contain several quarts of pus, and tends to break into the general peritoneal cavity, or into the intestine, especially the cæcum. It may be discharged into the bladder or the vagina, or the pus may escape through the abdominal wall either near the navel or in the lumbar region, or from the inguinal canal, the thigh, or the hip. Fæcal concretions or the sloughing appendix may pass through the opening and appear in an intestinal evacuation or in the discharge from the wound. In case of the communication of the abscess with a mucous canal and with the skin, the resulting fistula may remain open for months. The abscess sometimes extends to the space between the diaphragm and the liver, presenting the characteristics of a subphrenic abscess, perhaps to be followed by pleurisy or pericarditis. If the abscess lies in the vicinity of the internal iliac artery, the wall of this vessel may be perforated and fatal hemorrhage result.

Perforation of the appendix at times takes place before the general peritoneal cavity has been walled off by adhesions. In such cases a diffuse peritonitis results, the general peritoneum being injected and ecchymosed and a fibrinous or fibrino-serous exudation diffused throughout the abdominal cavity. Exceptionally an abscess resulting from perforation of the appendix lies in the subperitoneal tissue, forming a paratyphlitic abscess. This localization is likely to occur when the appendix is attached to the abdominal wall, either in consequence of developmental peculiarities or because of adhesions from a previous attack of appendicitis. If perforation takes place along the line of attachment, a retroperitoneal abscess arises which may extend in various directions and find outlets as various as those of the intra-peritoneal abscess.

Among the occasional complications of appendicitis is abscess of the liver. This is the result either of a pylephlebitis from the extension of a thrombophlebitis of the mesenteric vein leading from the appendix to the portal vein, or of embolism of the branches of the portal vein within the liver.

SYMPTOMS.—The recognition of symptoms of appendicitis is by no means so frequent as might be inferred from the observations of Toft and Hawkins of the prevalence of this disease. It is certain that many attacks of appendicitis are so latent as to produce either no symptoms or such slight disturbance as not to attract particular attention. The practitioner, however, is concerned with those instances in which positive symptoms are present: such cases may be conveniently grouped under acute and chronic appendicitis.

Acute appendicitis is characterized by abdominal pain, tenderness in the right iliac fossa, elevation of temperature, circumscribed resistance, and digestive disturbance. Most important is the unexpected occurrence of the pain in a person previously well or suffering for a day or two from slight malaise, manifested by loss of appetite, nausea, constipation, or diarrhœa. Although the pain is generally unexpected, it may follow an obvious exciting cause, as an error in diet, a jar or strain, or the action of a purgative, and is sometimes associated with a chill or chilliness. It varies in character from a sense of discomfort to one of agony compelling the patient to make a sudden outcry. It is usually constant, though sometimes paroxysmal. At the outset it is often referred to the abdomen in general or to the hypogastric, umbilical, epigastric, or other region, but is soon localized in the right iliac fossa. The severe pain is probably due to the extension of the inflammation to the peritoneum, and we agree with Richardson in regarding it as evidence rather of an actual or threatening perforation of the appendix than of a simple catarrhal inflammation. The surgeon often has found a perforation of the appendix at this early stage, when the pain resembled in character and severity that occurring in appendicitis recovering in the course of a few days under medical treatment.

Of greater diagnostic importance than pain is localized tenderness, often exquisite, produced by either superficial or deep pressure. The seat of the tenderness is usually found in the right iliac fossa, within a radius of two inches from the anterior superior spine of the ilium. McBurney has observed it oftenest near the outer edge of the right rectus muscle, on a line between the navel and the anterior superior spine of the ilium ("McBurney's point"). With the variation, however, in the position of the appendix the point of greatest tenderness may be found elsewhere in the right iliac fossa, or even in the umbilical or the lumbar region, in the left iliac fossa, in the groin, or in the pelvis.

Elevation of temperature, however slight, is a most significant symptom of appendicitis, since it indicates the inflammatory origin of the pain and tenderness. Within twenty-four hours after the onset of the pain the temperature may be less than 100° F., or it may rapidly rise above this point, especially in children, and throughout mild cases of appendicitis it may not exceed 101° F. In general, in a typical case of appendicitis an elevation of two or three degrees is to be expected, but a subnormal temperature may be present in the severest cases of acute appendicitis, in which general peritonitis is present from the outset. The pulse is quickened usually in proportion to the elevation of temperature, but is much accelerated in the grave cases even when the temperature is low.

Resistance on palpation of the wall of the right iliac fossa is next in importance to localized tenderness and elevation of temperature. During the first twenty-four hours after the incipient pain, especially when severe, the abdomen is often flattened, even retracted, and the tense right rectus abdominis muscle resists palpation, rendering it difficult, if not impossible, to distinguish a localized tumor if present. The abdomen, however, soon becomes distended and tympanitic, and though at first only moderately swollen it is afterwards considerably so. The circumscribed induration in the region of the appendix soon becomes apparent, and is usually found "in the right iliac fossa below the line extending from the anterior superior spine of the ilium to the navel, nearer the former, and two finger-breadths above Poupart's ligament." The position of the induration varies, however, in accordance with the difference in the position of the appendix already mentioned. This induration is sometimes superficial, in close proximity to the anterior abdominal wall, but is more often deep-seated, and covered by the distended and usually tympanitic cæcum or by distended coils of ileum. The induration may be diffused or circumscribed, and, if originally diffused, tends eventually to become defined. It sometimes represents a resistant mass of the size and shape of the little finger, or is ovoid in outline. This circumscribed resistance is due to the swollen appendix and the surrounding peritoneal exudation, upon the abundance of which depends the size of the tumor. It is dull on percussion when near the surface, but at the outset is usually covered by resonant intestine, and later may be tympanitic from the mixture of gas with the exudation. Fluctuation becomes apparent only at a late stage in the disease, when the exudation so increases in quantity as to lie near the anterior abdominal wall. Pelvic examination in a case of suspected appendicitis in which there is doubtful resistance in the right iliac fossa should never be omitted, since a tumor as well as tenderness may thus be detected.

The respiration is but little affected. There is loss of appetite, and vomiting is of frequent occurrence at the outset, but is usually temporary unless general peritonitis is present. Diarrhœa, though sometimes preceding the attack, is generally absent, except at a late stage in protracted cases. Constipation is the rule. The examination of the blood usually indicates a leukocytosis, although Richardson states that too much weight should not be laid upon this sign. Increased frequency of micturition is sometimes an early symptom, but retention of urine, perhaps requiring the use of a catheter, not infrequently takes place for a while after the first twenty-four hours. The urine is high-colored and may be albuminous.

In the further progress of acute appendicitis the tendency is towards resolution or perforation, with its resulting localized peritoneal abscess or general peritonitis. In these days of the frequent treatment of appendicitis, whether mild or severe, by laparotomy, it is impossible to obtain statistical evidence on a large scale of the relative frequency of these alternatives. According to the experience of most physicians in large practice, the termination in resolution is frequent. Of the one hundred and eighty cases designated typhlitis or perityphlitis analyzed by Fitz in his first communication, one-third ended in resolution. The statistics on this point of the greatest value are those given by Hawkins, who states that of two hundred and sixty-four cases of appendicitis admitted into St. Thomas's Hospital in consecutive order there was no suppuration in about seventy-two per cent., while there was a localized abscess or a general peritonitis each in about fourteen per cent. Richardson reports that of one hundred and thirty-seven cases of acute appendicitis seen by him thirty-six per cent. were mild cases and recovered without operation. In the personal experience of Fitz the course was mild in at least one-half of the cases.

In the mild cases of appendicitis terminating in resolution the pain soon becomes localized, and is easily relieved by hot or cold applications or by small doses of morphine, although occasional twinges occur. The temperature is usually slightly higher at each evening observation than on the previous day until the third or fourth day, when it drops, often suddenly, sometimes gradually, to nearly the normal point. The abdomen is only moderately distended, and there is usually but little nausea or vomiting. The localized induration in the region of the appendix shows no tendency to increase in size, and its sensitiveness rapidly diminishes. Although the action of the bowels is arrested and catheterization may be necessary to empty the bladder, the intestinal peristalsis and the function of the bladder are readily restored as the temperature falls. Spontaneous action of the bowels is often easily accelerated by the use of an enema.

The severe as contrasted with the mild cases of appendicitis are those in which the pain requires repeated doses of an opiate for its relief, and in which the painful area increases at intervals of a few hours. There is but little fall in the morning temperature, and that of the evening is higher than on the previous day. Neither gas nor fæces escape from the rectum, and there is often retention of urine, although there may be a frequent desire to empty the bladder. The abdominal distention rapidly increases, and the region of tenderness spreads in all directions, frequently into the pelvis.

In those severer cases which present the characteristics of a localized peritonitis, two possibilities are especially to be anticipated: the one is the circumscribing of the inflammation to the vicinity of the appendix, resulting in the formation of a sharply defined, usually intra-peritoneal abscess, and the other is the generalizing of the peritonitis.

The evidence of the formation of an abscess is furnished by the presence of a tumor at the seat of the pain and tenderness in connection with the persistence of the elevation of temperature. The tumor is usually deep-seated, and sometimes to be felt with difficulty in consequence of the abdominal distention. The various positions it may occupy have already been stated, and the importance of a rectal examination in determining its pelvic seat is obvious. With the formation of the abscess temporary improvement often takes place, although the temperature remains still elevated. The general abdominal distention and localized pain may subside, and intestinal peristalsis be restored, and there may be so little constitutional disturbance that frequently patients with a large quantity of pus in the abdomen have walked into the hospital.

Small abscesses may be absorbed, though slowly; large abscesses tend to become discharged into the cæcum, rectum, bladder, or vagina, or through the abdominal wall. A threatening evacuation into the rectum is often indicated by a frequent desire for defecation, and the escape of a gelatinous mucus, often blood-stained; impending perforation into the bladder is at times indicated by frequent, scanty, and painful micturition. Sudden perforation of the wall of the abscess, with the escape of pus into the peritoneal cavity, is always to be dreaded. Even if the abscess diminishes in size by the gradual absorption of pus, the danger of the formation of an embolic abscess of the liver should always be recognized. In this event chills, exacerbation of temperature, increased area of hepatic dulness, and pain in the region of the liver are likely to occur.

Generalizing of the peritonitis sometimes takes place at the outset of the attack of appendicitis. The initial pain then is of extreme violence, and extends over the entire abdomen. There is often a severe chill. The temperature usually is subnormal, but the pulse is rapid and feeble. The abdomen is tense and retracted. The skin is cool, moist, and at times mottled with livid spots; the eyes often are sunken, the face pinched, the voice husky. The patient may die during this stage of collapse, but not infrequently he rallies temporarily, the skin becomes hot, the abdomen distended, tympanitic, and fixed during respiration, and the pain and tenderness may diminish. Persistent vomiting is likely to occur, at times of a material resembling beef-juice, and death follows in the course of two or three days. These are the fulminating cases which offer so little hope from any form of treatment. More often the generalization of the peritonitis takes place more gradually. Without any considerable change in the course of the temperature, as observed in the severe cases, the pain and tenderness rapidly and progressively spread from the starting-point, and require increasing doses of opiate for relief.

The pulse gradually increases in frequency, and its force weakens. There is inability to take nourishment, and vomiting is frequent and eventually fæcaloid. With progressive loss of strength the patient may be comparatively comfortable, but rarely survives beyond the end of the first week, death not infrequently taking place suddenly and unexpectedly, often when the mental condition of the patient was so steadily improving as to make the outlook appear hopeful.

DIAGNOSIS.—A sudden attack of pain and tenderness in the right iliac fossa, associated with an elevation of temperature, however slight, in the great majority of cases is due to an attack of acute appendicitis. The evidence is strengthened if the symptoms are present in a young man. If the pain is intense, the tenderness exquisite, the abdomen retracted, and the right rectus muscle rigid, it is probable that perforation of the appendix is present or imminent, and the appearance of a circumscribed resistance at the usual seat of the appendix within twenty-four hours strengthens this probability. The attack of pain caused by disease of the appendix may be simulated by renal colic, whether due to the passage of concretions or to an acute hydronephrosis. Appendicitis is distinguished, however, from renal colic by the presence of fever, the gradual formation of a tumor, and the absence of hæmaturia. Attacks of biliary colic due to the passage of gall-stones rarely simulate the pain from appendicitis, but pain, tenderness, tumor, and fever due to acute inflammation and distention of the gall-bladder may closely resemble the symptoms of appendicitis. The pyriform shape, superficial seat, and mobility of the tumor, and the frequently associated jaundice, are absent in appendicitis. An acute attack of pelvic peritonitis, especially of tubal or of ovarian origin, may be mistaken for an attack of appendicitis. The previous history of the patient and the results of pelvic examination may suffice for the exclusion of these sources of error in diagnosis. Acute intestinal obstruction, particularly when strangulation exists, or when caused by intussusception, may suggest the general peritonitis caused by perforation of the appendix. The tumor of intussusception is less tender, and the frequent tenesmus and bloody stools of this affection are lacking in appendicitis; in internal strangulation from intestinal obstruction the symptoms are not sufficiently characteristic to eliminate appendicitis. The severity of the symptoms is such, in case of doubt, as to demand surgical treatment. In rare instances typhoid fever has been regarded as acute appendicitis. The localized peritonitis from tubercular mesenteric glands has proved a source of error in diagnosis, and in more frequent instances attacks of appendicular colic have been mistaken for those of inflammation of the appendix. The simulation of appendicitis takes place late in typhoid fever, and the previous history of the patient renders the diagnosis of appendicitis improbable. The progress of the disease or the presence of characteristic changes elsewhere may be necessary for the exclusion of the tuberculous condition, and the absence of fever in appendicular colic suffices to exclude this symptom as evidence of inflammation of the appendix.

PROGNOSIS.—That appendicitis is frequently recovered from under medical treatment is a fact familiar to all physicians. The mild cases are usually not reported, and the mortality as given by the surgeon relates rather to the disease as affected by operation than to the disease alone.

According to Porter, in a collection of four hundred and forty-eight cases the average mortality was about seventeen per cent., the death-rate in ninety-five cases treated medically being nearly fourteen per cent., while of three hundred and thirty-nine acute cases operated upon the mortality was about eighteen per cent. The figures of Hawkins are of greater value in determining the average mortality, since they represent experience at St. Thomas's Hospital under relatively uniform conditions. According to him, of two hundred and sixty-four cases the mortality was about fourteen per cent. All the cases ending in resolution, seventy-two per cent. of the whole, recovered, but of those ending in abscess twenty-six per cent. were fatal, and of those resulting in general peritonitis seventy-five per cent. died. Richardson reports that of one hundred and thirty-seven cases of acute appendicitis seen by him thirty-six per cent. were mild cases and recovered without operation; of the cases operated upon, two-thirds recovered and one-third died. His experience closely corresponds with the result of the analysis of one hundred and eighty cases designated typhlitis and perityphlitis made by Fitz in 1886,—viz., recovery by resolution in thirty-two per cent.

Although the average mortality of appendicitis may be stated as about fourteen per cent., the important question relates to the prognosis in the individual case. All mild cases recover under medical treatment, and the risks of surgical treatment lessen with the mildness of the symptoms. The surgical operation attended with the least mortality is that done after the patient has recovered from an acute attack,—as is commonly but erroneously stated, "in the interval between the attacks." While the symptoms are those of a mild appendicitis the individual prognosis is favorable, but they may suddenly or rapidly change, and the outlook in severe appendicitis is always uncertain.

With symptoms of apparent equal severity in two patients, the one will die of general peritonitis while the other quickly recovers: "the progress of the disease needs to be watched with knife in hand." In mild cases of appendicitis the temperature usually falls by the third or fourth day, intestinal peristalsis is restored, pain and tenderness disappear, and recovery takes place in the course of a week or ten days. In the severe cases death from general peritonitis is especially to be feared. Of such cases sixty-eight per cent. died during the first eight days, one-third of these previous to the fourth day. The prognosis as to the individual depends, therefore, upon the presence or absence of the symptoms of an extension of the peritonitis,—namely, rising pulse and temperature, and increasing distention, with or without a tumor. The persistence of the temperature after the third or fourth day, and the presence of a sensitive tumor, even with a falling temperature, are indicative of a localized suppurative peritonitis, from which the pus may be absorbed, but following which liability to recurrent attacks is frequent.

CHRONIC APPENDICITIS.

In nearly one-half of the cases of acute appendicitis seen by Fitz there was more than one attack of the disease, separated by longer or shorter intervals of freedom from discomfort, and from his experience, therefore, the patient is as likely as not to have another attack. The recurrent has all the characteristics and possibilities of the original affection. The symptoms are the same, either mild or severe, and the prognosis does not materially differ, except that the more numerous the recurrences the less severe are they likely to be. If the intervals are long, perhaps months or years, each subsequent attack is regarded as a *recurrent appendicitis.* If the attacks are frequent, occurring at intervals of weeks or months, and in the mean time the patient is comparatively free from uncomfortable sensations in the region of the appendix, the condition represents a chronic appendicitis with a tendency to relapses, or simply a *chronic* or *relapsing appendicitis.* It is possible, however, for a chronic appendicitis to exist without relapses, although these usually occur, and the lesions characteristic of a chronic appendicitis may be present as the result of an acute attack, and there be no symptoms indicative of this condition.

The disease chronic appendicitis, however, is to be recognized clinically by a series of symptoms localized in the region of the appendix. The essential feature in these symptoms is their persistence, intervals of relief being comparatively few. As Talamon has stated, chronic appendicitis is rather an infirmity than a malady menacing to life, and he has given the term appendicular colic to the frequent attacks of temporary pain in the region of the appendix. The patient is in a condition of more or less pronounced invalidism. Overwork or trivial disturbances of digestion produce pain and sensitiveness in the region of the appendix, compelling the patient to remain quiet for a day or two. With the pain and tenderness there may be a slight elevation of temperature. Sometimes constipation is associated with or precedes the discomfort, and occasionally a dull, resistant mass of considerable size is to be felt in the region of the cæcum, due to the retention of fæcal matter. This combination of retained fæces and a painful and tender appendix is the stercoral typhlitis of the older writers, and is evidently the result of a mild attack of appendicitis associated with constipation. In such cases relief often follows evacuation of the bowels, perhaps from the removal of a mechanical obstruction at the mouth of the appendix. On physical examination of the right iliac fossa in the interval between the attacks of pain, the enlarged appendix is often to be felt as a distinct tumor, perhaps of the size of the little finger, either directly beneath the abdominal wall or deep-seated in the iliac fossa. At such times there may be even tenderness on palpation, and the patient is usually conscious of the localized resistance offered. The more frequent the recurrence of the symptoms and the shorter the interval between them, the more enfeebled the patient becomes. He is not infrequently prevented from continuous work; he is debarred from the pleasures and profits of travel through fear of an attack of pain and its possibilities while at a distance from competent medical or surgical treatment. In addition to the constant uncertainty as to freedom from discomfort, there is always danger of the occurrence of an acute attack of inflammation resulting in perforation. He is often nervous and irritable, and becomes self-centred and timid. Pepper has characterized this condition as one of the most troublesome of curable affections. The symptoms may be protracted over a period of years, and we are indebted to Treves for first advocating the removal of the appendix when the patient has recovered from an acute attack.

Although the diagnosis of chronic appendicitis is usually easy, from the localization of the pain and tenderness and the frequent possibility of palpating the enlarged appendix, errors in diagnosis occasionally arise. In hypochondriasis and hysteria the patient often complains of pain in the right iliac fossa, and refers any disturbances of digestion or his general symptoms of nervous derangement to the diseased appendix. From such patients a normal appendix has been frequently removed. Critical observation shows that localized tenderness is often absent when the attention of the patient is diverted, and that there is no palpable tumor or localized resistance when the physical examination of the region of the appendix is made. In such cases oxaluria is not infrequent, and it is possible that irritation of the right ureter by the passage of crystals of calcic oxalate, as mentioned by Cabot, may explain the localizing of the discomfort. Patients with an inherited or acquired tendency to gout may have repeated attacks of renal colic from the passage of uric acid closely simulating relapses of discomfort in chronic appendicitis, and sometimes irritation in the course of the right ureter may occur in chronic appendicitis in consequence of the adherence of the appendix to the peritoneum overlying the ureter. In such cases repeated examination of the urine becomes necessary, and the presence of crystals of uric acid or calcic oxalate or of blood-corpuscles is suggestive of the renal nature of the attack. Cases of chronic appendicitis sometimes closely simulate those of cancer of the cæcum, for there is a condition of progressive loss of flesh and strength, failure of appetite, weakness of digestion, irregular action of the bowels, sometimes abundant mucous discharges, and a resistant tumor, not especially tender, in the region of the cæcum. To eliminate this possible error in diagnosis, importance is to be attached to an accurate history of the beginning of the attack and to the frequent observations of the temperature. In such cases the diagnosis may first be made by means of an exploratory laparotomy. The prognosis of chronic appendicitis, though in general favorable as to life, is always uncertain. The relapses or recurrences may gradually diminish in severity and the appendix become obliterated or destroyed perhaps during some severe recurrent attack.

TREATMENT.—The treatment of the individual case of appendicitis is almost always surrounded with great anxiety, on account of the difficulty, in fact, in many cases the impossibility, of determining in the onset of a case whether it should be looked upon as one of fæcal accumulation in the cæcum with associated inflammation of the appendix, or as one of mild catarrhal appendicitis, or whether ulceration or perforation exists.

The methods of treatment which have their advocates are not only various, but antagonistic, at least so far as the giving of drugs is concerned. All are in accord in inculcating absolute quiet in bed, with total abstinence at first from food other than chicken or other broths without rice or similar material in them, followed, when the time comes, by the addition of raw eggs or other albuminous liquid foods, pure milk being avoided on account of the tendency which it has to produce curds, although when diluted with carbonic acid water it is sometimes agreeable and useful.

The points in regard to which there are great differences of opinion are : first, as to the use of local measures ; second, as to the use of opium ; third, as to the employment of calomel, and of saline or other purgatives ; fourth, as to operative procedures.

Local applications consist in the use of heat and cold, of leeches, and of blisters. So far as concerns the use of heat or of cold, I believe that the sensations of the patient are the safest guide. If the continuous application of the hot-water bag gives the greatest comfort, it should be preferred. If the application of ice reduces the pain and is agreeable to the patient, it should be selected. Except in rare cases, the only objection which can be urged against the proper use of leeches is the trivial influence the leech-bites may have on any surgical procedures that afterwards become necessary. I do not believe that this objection has much force ; it requires only a little more care thoroughly to disinfect the leech-bites than surgically to cleanse the sound skin. The effect of the leeches upon the disease varies with the character and the cause of the attack : if the attack is the outcome of ulceration or gangrene of the appendix, or if the appendix is the centre of an active infective process, leeches have no influence upon the local inflammation ; on the other hand, if the inflammatory action is the outcome of a typhlitis stercoralis and is of slow development and of comparatively little force, leeches may be very useful, especially in gaining time for the employment of salines. Blisters I do not believe to be of any value in acute appendicitis. The blister increases the suffering of the patient and has little or no effect upon the spread of the inflammation; it also interferes with the work of the surgeon.

The question of opium is an exceedingly important one, concerning which there has been much discussion, which, so far as I am concerned, has led to some alteration of views. I still believe that opium does good in these cases by controlling pain and restlessness, and also acts antiphlogistically in some unknown way. On the other hand, there is great force in the surgical contention that opium interferes with intestinal secretion and peristalsis, and especially so masks the symptoms as greatly to enhance the difficulties of deciding the progress of the case and the time at which surgical interference should be adopted. I believe, therefore, that unless opium is called for by the presence of excessive pain it is best to avoid it, and that when used it should be in the form of hypodermic injections of morphine.

The difficulty surrounding the question as to the administration of salines is largely one of diagnosis. If the appendicitis is connected with fæcal accumulation in the cæcum, the administration of salines until the bowels have been thoroughly emptied is strongly indicated. If on the first day of a mild appendicitis there is the sense of the presence of a tumor imparted to the fingers on palpation, salines should always be given, and in many cases their use should be combined with that of calomel. It is better to give repeated small doses than a single large dose,—the large dose being much more apt to be vomited or to cause distress. Magnesium citrate is probably the best of the salines, on account of the pleasantness of its taste, though sodium sulphate is probably more certain in its action; its bitterness, however, makes it more nauseating.

It is a significant fact that whilst formerly scientific physicians utterly abandoned and condemned the use of calomel in such diseases as diphtheria, it was largely used by country practitioners, and through their influence has been forced back upon the leading members of the profession. The same class of practitioners have so often affirmed to me that they have seen an appendicitis improve simultaneously with the coming on of ptyalism that I regard their evidence as of practical value. In the class of cases of appendicitis now under consideration, when there are no perforation and no gangrene or hopeless septic infection, in my opinion calomel should be administered. It acts as a laxative, and also, according to my belief, as an antiphlogistic remedy.

At alternate half-hours the patient may take an ounce to an ounce and a half of magnesium citrate solution and a half-grain of calomel, the calomel being dropped when from seven to ten grains have been taken, even if no action of the bowels has occurred, and the saline being administered hourly, day and night, until a free passage has been obtained or until the impossibility of so doing is demonstrated. When ulceration, perforation, or gangrene is present in an appendicitis the salines can do no good, and may readily do harm : so that they should not be exhibited. It is, however, impossible in most cases to determine positively when perforation or ulceration occurs, so that it may be considered as rule-of-thumb practice to use the saline in the beginning of an appendicitis which is not foudroyant or explosive in its type. If, however, the practitioner should believe that there is probably ulceration or gangrene of the appendix, the purgative should be used only if absolutely necessary to overcome demonstrable fæcal retention. The opinion of many of our best surgeons that the presence of fæces in the colon greatly increases the danger from the operation upon the appendix cannot be properly disregarded. In almost all cases of appendicitis high enemata are valuable : if there is reason to suspect that there is ulceration or perforation, they alone must be depended upon to clean out the colon ; if these complications are absent, they may be used to assist the saline laxatives.

The most vital problem in any case of appendicitis is as to the propriety of surgical interference. Resolution after ulceration and formation of an abscess is such a rarity that the possibility of its occurring in any case should not be taken into consideration. When, therefore, there is reason to believe during an acute appendicitis that perforation or the local formation of pus has occurred, or that the appendix has become gangrenous, immediate operation should be performed. On the other hand, very frequently, perhaps in the great majority of cases, it is impossible to diagnose accurately the condition of the appendix : so that the question naturally presents itself as to what would be the result of operating upon every case as contrasted with the result of using the expectant treatment, with selection of cases for the surgeon. There are, however, no sufficient statistics to warrant definite conclusions on these points. The opinion put forth by some surgeons that the operation is free from danger is, in my opinion, erroneous. The question of the skill of the operator is in appendicitis a most important one : the operation should be undertaken with the greatest sense of responsibility, and only by those who by previous training are thoroughly prepared : it should be carried out with the most absolute asepsis. It being understood that a proper surgeon is available, the following rules seem the best that can be laid down for guidance in this matter.

First, when in the onset of the attack the pain, the tenderness, and the tympany are excessive, and the fever and pulse rapidly rising, the probabilities of an acute perforating appendicitis are such that an immediate operation should be performed, each hour lost sensibly increasing the danger.

Second, when in a case of mild appendicitis sixty hours of careful treatment have gone by without distinct abatement of the symptoms, the operation should usually be performed, except in the rare cases in which masses apparently fæcal in character have been detected at the beginning of the attack in the head of the colon and still remain there to some extent, especially if there has been tenderness along the course of the colon away from the immediate neighborhood of the appendix.

Third, an immediate operation should be performed when in a hitherto mild case a sudden increase in the local and general symptoms points towards the occurrence of perforation or the formation of pus, this rule being imperative if the acute symptoms are accompanied by such wide-spread general tenderness and marked increase in the fever and pulse-rate as to indicate the coming on of a general peritonitis. In such a case minutes are important, and unless the operation can be performed before the full development of septic peritonitis the result will almost certainly be death.

During convalescence from appendicitis great caution should be exercised in getting the patient back to ordinary food, and laxatives must be used freely if needed. Even after recovery care should be taken to avoid indigestible food, fruits containing seeds, violent exercise, or any exertion which will throw strain upon the abdominal muscles and which might, by breaking up an adhesion, stir up a slumbering inflammation. The bowels should be kept perfectly soluble; if there be a remaining induration, persistent mild counter-irritation, especially with iodized oil, may be used locally. No drugs, except laxatives, are of any avail.

Recurrent appendicitis often finally gets well without operation, but certainly very grave risks attend leaving the case to nature. There are no reliable statistics which enable us to estimate accurately the mortality of operations between attacks of appendicitis, but Bull is probably not far from the truth in putting the rate at from five to six per cent. I believe this mortality to be far less than that which is the result of leaving the cases to nature, and that an operation should be performed if the past attacks have been very numerous, especially if they are increasing in frequency or severity, or if the attack last recovered from has been very alarming, or if the persistent severity of the local symptoms between the attacks makes it probable that there is an abscess. Indeed, I am strongly inclined to go further, and to make it a rule to operate upon all cases directly after the recovery from a second attack, unless there is some very distinct reason for not doing so. The necessity for a person who has had two attacks of appendicitis always keeping within reach of a first-class surgeon is most pronounced, and sometimes is embarrassing.

At the operation the appendix should be taken out, provided it can be done without too much injury or without such manipulations as may rupture possible adhesions or bring about the escape of septic matter into the peritoneum. The question of removal or non-removal must be settled during the operation by the surgeon.—H. C. W.

Recognizing the impossibility of satisfactorily determining at the outset how an attack of appendicitis is to end, but convinced that the large majority of cases can recover quickly, easily, and safely under medical treatment, it seems to me advisable to advocate such treatment as shall favor the predominant tendency of this disease to terminate in resolution.

From this point of view the essentials are to check peristalsis above the cæcum and to relieve pain. Repeated instances have occurred of the aggravation of the symptoms soon after the administration of laxatives by the mouth, often by an anxious mother who attributes the abdominal pain to the presence in the intestine of indigestible food or retained fæces. All cathartic medicines are, therefore, to be avoided until convalescence is established, and only the blandest liquid diet is to be permitted. If constipation has preceded the attack or the colon is distended with gas, a rectal enema often gives relief, and does not threaten the tearing apart of delicate adhesions whose influence is protective, or risk the perforation of a weakened appendix.

The relief of pain is best accomplished locally by means of hot or cold applications. If these are ineffectual, morphine should be given, beneath the skin, by the mouth, or in suppository, in such quantity as to keep the patient comfortable. Small doses are usually sufficient for this purpose.

If resolution is to occur, it is likely to take place by the third or fourth day: hence, when the condition of the patient permits, an operation should be delayed until this time. The surgical treatment of the acute attack is always to be avoided when possible, because it is unnecessary in the majority of cases, and is followed by the risk of a subsequent hernia. If eventually required, it is more safely employed in the absence of acute inflammatory symptoms, and there is afterwards less likelihood of hernia.

The removal of the appendix for chronic inflammation is to be recommended in those cases in which recurrences are frequent or the tendency to relapses is such as to produce a state of semi-invalidism.—R. H. F.

INTESTINAL OBSTRUCTION.

Definition.—An internal mechanical interference with the action of the bowels, causing complete arrest of evacuation.

In the usual restricted use of the term intestinal obstruction only the mechanical causes within the abdomen are considered. Acute is to be distinguished from chronic obstruction, although the causes of the latter may produce acute outbreaks.

VARIETIES.—The internal mechanical causes of acute intestinal obstruction in two hundred and ninety-five cases were as follows: strangulation by bands and cords, by slits and fissures, and by peritoneal pouches, in thirty-four per cent., intussusception in thirty-two per cent., abnormal contents in fifteen per cent., twists and knots in fourteen per cent., and strictures and tumors in five per cent. Since cases of acute obstruction from abnormal contents and from strictures and tumors are of the least practical importance, and as obstruction from knots is extremely rare, it is convenient to remember that acute intestinal obstruction is due to strangulation or to intussusception, each, in about forty per cent., and to twists in about twenty per cent.

Strangulation.—In seventy per cent. of the cases of strangulation by bands and cords a previous peritonitis was important in etiology, either by producing fibrous adhesions or by causing the adherence of the appendages of the intestinal or genital tract. The intestinal appendages include the epiploic appendages, the vermiform appendix, persistent vitelline remains, the omentum, and the mesentery. The vitelline remains are Meckel's diverticulum (the vitelline duct) and the patent or obliterated vitelline blood-vessels. The diverticulum is usually connected by the blood-vessels with some part of the abdominal wall or contents, but may be adherent only in consequence of a localized peritonitis. The vitelline blood-vessels or their remains may likewise form a strangulating cord in the absence of the diverticulum, and are connected with the mesentery or the anterior abdominal wall, usually in the vicinity of the navel. Omental adhesions may result in the formation of a strangulating cord, or a loop of intestine may pass through a slit or fissure of the atrophied or aplastic omentum. The rare strangulating slit of the mesentery and abnormal peritoneal pouches are attributable to defective development. Extremely rare is strangulation from rupture of the diaphragm. Seventy per cent. of the cases of strangulation occur in males, and forty per cent. in persons between the ages of fifteen and thirty. Strangulation in early youth is relatively uncommon, but when occurring it is usually due to vitelline remains. The small intestine is obstructed in nearly ninety per cent. of the cases, the lower abdomen is the seat of the strangulating object in eighty-three per cent., and the right iliac fossa in sixty-seven per cent.

Intussusception.—In this variety of acute intestinal obstruction, according to the experiments of Nothnagel, a contracted portion of the intestine forces its way into the relaxed portion immediately below. The upper portion is thus invaginated into the lower, and more and more of the intestine may be forced into the sheath, which also often becomes simultaneously inverted, until the invaginated portion lies in the rectum. In seventy-five per cent. of the cases the ileum was invaginated into the cæcum or into the colon, the *ileo-cæcal* and *ileo-colic* varieties. The small intestine was invaginated into itself, forming the *enteric* or *ileal* variety, in twelve per cent. of the cases, while the invagination of the colon into itself or of the cæcum into itself occurred in a like number of cases. Two-thirds of the cases of intussusception occurred in males, fifty-six per cent. in children under ten years of age, and thirty-four per cent. in infants less than twelve months old. Although diarrhœa or constipation is often present, and indigestible food, violent exertion, or injury immediately precedes intussusception in a certain number of cases, these factors are not of sufficient importance in etiology to require especial consideration. The immediate effect of the invagination is the production of a tumor, the appearances of which vary according to the duration of the intussusception. The tumor forms a sausage-like mass of intestine, and the inverted portion after death is often withdrawn with difficulty from the sheath, at the mouth of which the vermiform appendix, in the ileocæcal variety, often protrudes. On opening the sheath the invaginated portion has a crescentic outline, in consequence of the traction of its mesentery. From the compression of the blood-vessels of the invaginated portion as it enters the sheath, the intestinal veins become distended with blood, the mucous membrane is of a purple color, and hemorrhages within or from the intestinal wall are frequent. The opposed peritoneal surfaces are covered with a fibrinous exudation forming adhesions, the presence of which is a means of distinguishing the intussusceptions occurring in the death-agony from those which are the cause of death. If the patient survives the immediate effects of the intussusception, necrosis of the invaginated portion takes place, and several feet of the intestine may be discharged as a slough. In such cases fusion of the mouth of the sheath with the intestine above the point of separation takes place, and eventually healing may result, usually ending in the formation of a fibrous stricture.

Twists and Knots.—The large intestine is involved in eighty-seven per cent. of all cases of twist. About one-half of the twists of the large intestine are located at the sigmoid flexure, and one-third in the region of the cæcum. The formation of the twist is promoted by the elongation of a loop of the intestine in consequence of hernia, the traction of adhesions, or the prolonged accumulation of fæces. The affected part of the bowel is usually twisted along its axis for a half-turn, a whole turn, or even more, and a strangulation of the intestine is the result. The coil of intestine below the point of strangulation is distended and purple, death in fatal cases usually resulting from general peritonitis. In rare cases a loop of the small intestine may be twisted about another portion. Nearly seventy per cent. of the cases of volvulus or twist are in males, and about one-third of them occur between the ages of thirty and forty years, although this cause of intestinal obstruction has been found at the age of six and beyond that of seventy years.

Knots as a cause of intestinal obstruction are so rare as to be of no practical importance. They are formed by the encircling of a coil of intestine by a neighboring loop, the free end of which so passes beneath the attached portion as to form a knot. The effect is to produce strangulation of the intestine.

Strictures and Tumors.—Acute obstruction from strictures or tumors of the intestine or from abdominal tumors outside the intestine occasionally occur. The large intestine is usually obstructed, and the cause is generally seated in the lower half of the abdomen. These causes of obstruction are more often found in women, in consequence of the prevalence among them of abdominal tumors, and four-fifths of the cases occur after the age of forty. Cancerous tumors of the intestine cause acute obstruction more frequently than do fibrous strictures. Strictures of the intestine may exist at birth and be manifested by complete obliteration of the canal, as in imperforate rectum, or by the separation of the stomach from the duodenum. Acquired strictures result from the healing of ulcers, especially of tubercular or syphilitic ulcers. A stricture may also result from the healing of the ulcer due to intussusception and of stercoral ulcers at the flexures of the colon. Although stricture of the intestine is of frequent occurrence in chronic dysentery, this variety is rarely sufficient to produce acute obstruction.

Abnormal Contents.—Various foreign bodies introduced into the intestine by the mouth or by the rectum may become causes of acute intestinal obstruction. These are taken in either accidentally, especially by children, or intentionally, as in the insane. Generally they are solid masses, as stones, coins, glass, nails, or false teeth. The common abnormal contents which cause intestinal obstruction are biliary calculi, impacted fæces, and enteroliths. The *enteroliths* are composed in considerable part of undigested material, such as hair, thread, fruit-stones, or bits of bone. The deposition of calcium and magnesium phosphates takes place within or about these substances, and calculi are formed which may be as large as a hen's egg. Intestinal calculi may be composed of lime or magnesia taken as medicines for a long period of years, and cases have been reported of obstruction resulting from a mass of round worms.

Gall-stones are found to be the cause of obstruction in three-fourths of the cases due to abnormal contents. The majority of the patients are women, all are adults, and six-sevenths of them are more than fifty years old. Obstruction from impacted fæces occurs with equal frequency in the two sexes and at all ages.

Chronic obstruction is due to strictures, to tumors, or to fæcal impaction. The strictures are oftenest of a cancerous nature, though sometimes due to tubercular, syphilitic, or stercoral ulcers, to the healing of an ulcer following the detachment of the slough in intussusception, or to a localized chronic peritonitis. The non-malignant tumors causing chronic obstruction are polypoid fibromata or lipomata.

SYMPTOMS.—The principal symptoms of acute obstruction are pain, vomiting, tympany, and tumor. Stoppage of the bowels is an important symptom, though not always present throughout, and may be so complete that neither gas nor fæces escape. Frequent loose movements may be present at an early stage of intestinal obstruction, and are often a characteristic symptom of intussusception. When fæcal retention is the cause of obstruction, numerous ineffective dejections are common.

The pain is usually sudden and severe, and often colicky. In most cases it is referred to the abdomen in general, but occasionally it is localized in one or another region of this cavity. In intussusception the initial pain may be of gradual onset, and is often manifested as tenesmus.

At the outset tenderness is not an especial feature, and, indeed, it is not conspicuous throughout the attack.

Vomiting occurs with great frequency. At first the food last taken is expelled, later a bile-stained fluid is ejected, and eventually a yellow fluid, the contents of the duodenum, appears, at first odorless, but becoming, on or about the third day, of an offensive odor, the so-called fæcal or stercoraceous vomiting. There is no satisfactory evidence, however, to warrant the idea that the contents of the large intestine are ever vomited, and in intussusception fæcaloid vomiting is rare. Tympanitic distention of the abdomen, as a rule, soon makes its appearance. It may be enormous, but is usually moderate, and is less conspicuous in obstruction from intussusception than from strangulation. A palpable tumor is of great frequency in intussusception, but is only occasionally observed in the other varieties of acute obstruction. The tumor is composed of circumscribed distended intestinal coils, often with visible outline, and may be felt as a dense object in the case of abnormal contents. In acute obstruction from cancer the tumor is sometimes palpable, either by the rectum or through the abdominal wall, although acutely obstructing cancer is usually of the annular type, causing rather a stricture than a tumor. The presence of a tumor is most constant in intussusception, being apparent in nearly two-thirds of the cases. It is to be recognized on examination of the abdomen or of the rectum, more commonly of both, and is of early occurrence, being found within the first three days in more than three-fourths of the cases. It is usually felt in the course of the large intestine, especially in the region of the descending colon, as an elongated cylindrical mass, not freely movable, and often temporarily increasing in density during spasmodic and painful peristalsis. If the tumor is low down in the rectum there is often a relaxed sphincter, and the finger introduced into the rectum usually readily feels the slit-like opening of the lower end of the invaginated intestine. When the abdominal tumor is due to obstruction from fæces, it may occupy the entire abdomen or be limited to the course of the colon. It is elongated, rounded, nodular, slightly movable, hard, and dull on percussion. The fæcal nature of the tumor is often to be determined by a rectal examination.

The temperature is frequently elevated, usually only to a moderate extent, especially after the first day of obstruction from strangulation, and may be subnormal, particularly when symptoms of collapse occur. On the contrary, if peritonitis supervenes the temperature continues to rise. The pulse at the outset is but little affected, but soon becomes quickened and feeble. Hiccough is only occasional, though the cause of much discomfort. The urine is usually high-colored and scanty, especially when there is excessive vomiting, and may contain albumin and indican.

Chronic obstruction is characterized by persistent constipation, extending over a period of months or years. Weeks may elapse without a movement of the bowels, or scanty dejections may pass through a tunnelled or channelled fæcal mass in the colon. Very frequent in chronic obstruction by impacted fæces are repeated mucous discharges. Enormous fæcal accumulations may exist and very few symptoms arise, or pain, nausea, and vomiting may result. In rare cases chronic obstruction from impacted fæces may be followed by death from perforation of the intestine. Chronic intestinal obstruction from stricture or tumor is indicated not only by long-standing constipation, but also by abdominal pain or discomfort often referred to a definite point, which frequently proves to be the seat of the obstruction. There is progressive loss of flesh and strength, with not infrequently attacks of acute obstruction, which are relieved by appropriate treatment. As the patient becomes emaciated the outlines of the distended coils of intestine can be seen beneath the wall of the abdomen. There is visible peristalsis, associated with borborygmi. A rectal examination may enable the seat of the stricture or tumor to be determined, and palpation of the abdominal wall may indicate the presence of the tumor. In cases of irremediable chronic obstruction death results from progressive exhaustion, from a supervening acute obstruction, or from peritonitis following perforation above the point of obstruction.

DIAGNOSIS.—The symptoms suggestive of acute intestinal obstruction —namely, pain, vomiting, tympany, or tumor, and arrested alvine discharges—may be due to external herniæ: hence such sources of obstruction should be excluded before internal causes are considered. The symptoms of acute intestinal obstruction so closely simulate those of peritonitis that a differential diagnosis between the two affections is often of extreme difficulty, and frequently is made first by means of an exploratory laparotomy. At the outset the early presence of fever and general abdominal tenderness are suggestive rather of peritonitis than of intestinal obstruction. Persistent vomiting is more frequent in obstruction than in peritonitis. The causes of peritonitis are to be eliminated as far as possible: hence evidence of antecedent disease should be sought in the gastro-intestinal, genital, and urinary tracts, in the biliary passages and the pancreas, in suppurating or necrotic lymph-glands, in embolism of the mesenteric arteries, in latent appendicitis, and in suppurating inflammation in the vicinity of the peritoneum.

The symptoms of acute intestinal obstruction are sometimes simulated by the results of a severe blow upon the abdomen, the after-effects of difficult and prolonged laparotomies, the reduction of a hernia, and attacks of biliary or renal colic. The history of the case, the localization of the pain, and the absence of jaundice and hæmaturia are usually sufficient to exclude these possible sources of error in diagnosis. Time often is necessary to determine whether the condition is one of acute internal mechanical obstruction or a localized or diffuse peritonitis.

The part of the bowel obstructed is suggested by the history of the case, the appearance of the abdomen, and rectal examination. In obstruction of the small intestine the abdominal distention is present at first in the epigastric and umbilical regions, and the tension is often temporarily reduced by vomiting. The urine is scanty and contains an excess of indican, and the symptoms of collapse usually occur within four days after the onset of the symptoms. In obstruction of the large intestine fæcaloid vomiting is generally absent or of late occurrence, the abdomen is enlarged at first in the flanks and in the region of the transverse colon, and a palpable tumor is often to be recognized. Tenesmus and discharges of bloody mucus are frequent.

Digital exploration of the rectum may disclose an intussusception, a tumor, a stricture, or hardened fæces when the large intestine is obstructed. The determination of the capacity of the large intestine by injections of warm water is of value when examination by the finger is negative. A bulb syringe usually suffices for this purpose. The patient, anæsthetized if necessary, should be placed on the back, with the hips raised, or on the right side. A soft rubber tube should be introduced into the rectum for several inches, leakage through the anus being prevented by compresses around the inserted tube, which is connected with the syringe. The capacity of the large intestine of an adult is six quarts, that of the rectum three pints. The entrance of the larger quantity of water would indicate that obstruction was at or above the cæcum, whereas obstruction in the region of the sigmoid flexure would be suggested if only the smaller quantity of water could be introduced. The earlier the capacity of the large intestine is thus determined, the safer, since Thomas reports rupture of the intestine in a case of intussusception from an enema injected under light pressure on the third day. Exploration of the large intestine by means of a flexible tube is of but little value, since its entire length may be passed through the anus, and the tube be found, on digital examination, coiled within the rectum. The attempt to introduce a rigid tube may be dangerous, although rarely an unusually long rectum may permit the tip of the tube to be felt through the abdominal wall above the pelvis.

In determining the cause of the obstruction it is to be remembered that practically sixty per cent. of all cases of acute internal mechanical obstruction are due to strangulation from bands, cords, twists, or knots, and forty per cent. to intussusception. The nature of the cause is to be inferred from a knowledge of the part of the bowel affected, the age of the patient, the special symptoms, and the relative frequency of the several causes of obstruction. Acute obstruction of the large intestine is due to intussusception or twists in eighty per cent. of the cases. If the patient is under thirty years of age, intussusception is more common than twist, and is indicated by tenesmus, tumor, and bloody stools. The capacity of the colon as determined by injection is likely to be greater in intussusception than in twist, since the former is near the cæcum in seventy-five per cent. of the cases, while twist is at the sigmoid flexure in fifty per cent. If the patient is over thirty years of age and the evidence of intussusception is lacking, the obstruction is likely to be due to strangulation or a twist in which the symptoms are acute, or to a tumor or stricture, acute obstruction from which is preceded by symptoms of chronic obstruction, and the presence of which may be indicated by rectal examination. Strangulation affects that part of the large intestine near the sigmoid flexure, while obstruction from cancer or stricture usually takes place below this region. Tumor and stricture are the commonest causes of acute obstruction of the large intestine, but, as already stated, are usually preceded by symptoms of chronic obstruction.

Acute mechanical obstruction of the small intestine not due to gallstones or foreign bodies is the result practically of strangulation. Gallstones are to be eliminated by their usual occurrence after the age of fifty, the previous symptoms of cholelithiasis in one-half the cases, the late occurrence of tympany, and the occasional recognition by palpation of a hard, movable nodule in the abdomen. The history of the case when the resistance is palpable may indicate the presence of a foreign body. Strangulation is due to adhesions in seven-tenths of the cases and to vitelline remains in one-fifth. It is of importance to remember that the causes of strangulation of the small intestine are to be found in the lower abdomen in four-fifths of the cases.

The diagnosis of chronic obstruction is readily made from the persistent constipation. The presence of impacted fæces may be recognized on physical examination of the abdomen and rectum, and freedom from discomfort and constipation after their removal makes the diagnosis clear. If relief is not experienced, the presence of a stricture or tumor is obvious, especially when distended intestinal coils are visible. Abdominal palpation may reveal the presence of a tumor, or rectal exploration may disclose or make probable a stricture or a tumor, the nature of which is usually to be determined only by an exploratory laparotomy.

PROGNOSIS.—Acute intestinal obstruction is a grave affection, but the prognosis varies especially in accordance with the cause. Obstruction from strangulation is almost uniformly fatal unless timely surgical treatment has afforded relief. Symptoms of collapse early arise, and death usually results between the second and the fourth day. Possible exceptions to the rule are to be admitted, for in rare instances relief from the symptoms has followed medical treatment. A spontaneous reduction of the strangulated intestine and the reversing of the twist are conceivable, though not sufficiently probable to be offered in evidence against surgical treatment. In obstruction from intussusception death occurs with the greatest frequency between the third and the fifth day, although spontaneous recovery sometimes takes place by a reduction of the intussuscepted intestine or by its discharge as a slough. The mortality is least severe in case of the rectal variety. Of thirty-five cases of other varieties of intussusception treated medically, seventy per cent. died and thirty per cent. recovered. The surgical treatment of this affection in thirty-six cases showed a mortality of eighty per cent. In obstruction from gall-stones two-thirds of the cases medically treated recovered. Of the cases treated surgically, one-fifth recovered. Obstruction from fæces sometimes proves the cause of death by perforation of the bowels or from the progressive enfeeblement of a person already debilitated by age or disease. In obstruction from strictures and tumors, although recovery from the immediate symptoms may take place under medical treatment, a radical cure is to be obtained only by surgical measures. The frequent malignant nature of the stricture or tumor usually makes such treatment merely palliative, even if the patient recovers from the effects of the operation.

Treatment.—In acute intussusception all food should be withdrawn for the time being, and the bowels should be kept completely at rest by full doses of opium, which should usually be given in suppositories. As the formation of adhesions practically puts an end to the possibility of withdrawing the intussuscepted intestine, immediate efforts should be made to get the gut into its normal position. The best method of doing this is to put the etherized patient in an inverted position, and administer, by means of a fountain-syringe, elevated from six feet for an infant to fifteen feet for an adult, warm saline solutions of olive oil, the nozzle of the syringe being inserted up to the sigmoid flexure; whilst full of fluid, under pressure, the bowel should be systematically compressed and kneaded from below upward, great care being exercised not to use undue force, whilst from time to time the patient is well shaken. If no success attends this procedure, dilatation of the intestines, by forcing into them from a large india-rubber bag two to four gallons of atmospheric air, or, as preferred by Senn, hydrogen gas, may be tried. The practice of injecting into the intestines a solution of sodium bicarbonate followed by one of tartaric acid seems to us somewhat risky, on account of the difficulty of controlling the amount of gas evolved. After the first twenty-four hours of intussusception, hydrostatic and pneumatic treatments are alike dangerous; the case should be at once operated upon, or be left to nature, aided by careful feeding with small quantities of strong broths, warm local applications, and the persistent use of opium to mild narcotism. If during the treatment the stomach becomes very much distended, it may be washed out, often with advantage.

In the cases collected by Fitz the mortality without laparotomy was sixty-nine per cent., with operation eighty-three per cent. In Ashhurst's statistics the mortality-rate with operation was about seventy per cent., practically the same as that of a large number of cases not operated upon collected by Leichtenstern. Improving technique will probably lessen somewhat the surgical mortality, but at present the exact value of laparotomy in intussusception, especially if the patient is an infant, has not been determined. The operation, if performed at all, should not be postponed longer than twenty-four hours. Enterotomy may be practised as a late operation when the abdominal distention and distress are very great.

In cases of chronic obstruction of the bowels, if relief is not afforded by careful regulation of the diet, treatment of catarrh or other causative or complicating disorder, and the use of very mild laxatives and enemata, with opium and belladonna as required, laparotomy should be performed, followed, if the obstruction be found irremovable, by enterectomy, if the patient be strong enough, or by the formation of a false anus.

CANCER OF THE INTESTINE.

Although cancer of the intestine is the most frequent mechanical cause of chronic intestinal obstruction, it often occurs without producing this result, and therefore it requires separate consideration.

Etiology.—Intestinal cancer is more common in men than in women, especially after the age of fifty. The importance of local causes, particularly the mechanical action of fæces, is suggested by the almost invariable presence of the disease in the large intestine, its occurrence in the rectum, according to Leube, in four-fifths of the cases, and its limitation to the cæcum, or to the sigmoid, splenic, and hepatic flexures, in the remaining fifth.

Morbid Anatomy.—For practical purposes all malignant tumors of the intestine are included in the description of cancer, since it is the latter that is oftenest present, and its symptoms do not differ from those produced by sarcoma or lymphoma. From the easily recognized physical characteristics, soft, hard, and gelatinous cancers are differentiated. The distinction between malignant adenoma and cancer is one of but little value; since the epithelioid cells of cancer are often cylindrical, as are those of adenoma and malignant adenoma. Malignant adenoma presents the clinical characteristics of cancer, and the distinction between adenoma and malignant adenoma is to be determined only by the lapse of time. The gross appearances of cancer of the intestine do not differ essentially from those of cancer of the stomach or of the œsophagus. It arises in the deeper portion of the mucous membrane, and as it increases in size extends laterally and in depth and projects in the form of a nodule. This continues to enlarge, and may exist above the surface of the intestine as a flattened induration with sharply defined, perhaps everted, edges, either isolated or associated with smaller nodules. The cancerous growth may also encircle the intestinal tube and form a broad or narrow ring. The overlying mucous membrane becomes destroyed, and superficial portions of the cancer die, and are rubbed off or torn away, an ulcerated surface remaining. As the disease extends to the submucous and muscular tissues it invades the neighboring tissues or organs, the ulcer becomes deepened, and perforation of the intestine may result, with the establishment of communication between adjacent coils of intestine or with the bladder or the vagina. Multiple nodules are likely to appear in the peritoneum, and to be found in the liver, in the lungs, and even in the more remote portions of the body.

SYMPTOMS.—Cancer of the intestine, as a rule, causes no suggestive symptoms referable directly to the intestine until ulceration, stricture, or tumor is apparent, and the disease exists, therefore, often for a long time before its presence is suspected. Irregular action of the bowels may be present for years without other disturbance until localized pain, slight tenderness, and perhaps ill-defined induration, occur. The pain at the outset is manifested as a sense of constant though slight discomfort, but in the course of time becomes colicky in character, its severity increasing, and when in the rectum has the characteristics of tenesmus. In rectal cancer a dull ache may be referred to the sacral or coccygeal region, and the desire for defecation is frequent and irresistible, evacuations of the bowel being followed by temporary relief. Of especial importance in exciting suspicion of cancer in connection with the above symptoms is loss of flesh and strength out of proportion to the discomfort from which the patient suffers. Cachexia results despite the good appetite, the normal digestion, and the freedom from change in the quality and quantity of the excrement. The fæces are sometimes ribbon-like or furrowed when the lower part of the intestinal canal is narrowed by a cancerous stricture. The serious nature of the symptoms is often first suggested by the presence of blood or of blood-stained mucus in the dejections, and the suspicion of an ulceration becomes strengthened by the presence in the intestinal discharges of pus or shreds of tissue, the cancerous nature of which is sometimes to be recognized on microscopical examination. With the occurrence of ulceration pain and diarrhœa become constant and severe, the appetite fails, digestion weakens, and there is rapidly increasing pallor of the skin.

The symptoms indicative of the progress of cancer of the intestine towards the formation of a stricture have already been mentioned in the article on intestinal obstruction. It may be repeated, however, that an attack of acute intestinal obstruction may first excite the suspicion of a cancer, and that repeated attacks of acute obstruction with intervals of comparative comfort are oftenest due to cancer of the intestine. As a rule, however, constricting cancer of the intestine produces symptoms rather of chronic than of acute obstruction.

The formation of a visible or palpable tumor in cancer of the intestine belongs usually to the late stage of its progress, and is more likely to take place when the cancer affects either the cæcum or the rectum. The tumor is largely due to the extension of the disease to the tissues outside the intestine. It may be composed in part of coils of intestine united by cancerous adhesion and in part of retained intestinal contents. Variations in the shape and consistency of the tumor thus arise, and not infrequently rapid modifications in size result from diminution or increase in the mass of the accumulated intestinal contents. The tumor may be small, dense, nodular, floating or fixed, superficial or deep-seated. It may be large, rounded, and smooth, causing a projection of the abdominal wall, especially conspicuous in an emaciated person. There may be but little change in the apparent size of the tumor for weeks or months, or the mass may attain the size of an infant's head in the course of a few weeks, the growth being so rapid and the consistency so soft as to suggest the presence of fluid.

If stricture is present, the abdomen is enlarged, and distended coils of intestine are often easily recognized. If the cancer progresses without a tendency to stricture, there may be nothing abnormal in the appearance of the abdomen, or it may be even flattened.

On rectal examination the cancerous growth is often found within reach, and secondary tumors in Douglas's fossa are frequently felt when palpation of the abdomen fails to reveal the primary cancer.

When the significant conditions of ulceration, stricture, and tumor become apparent the course of cancer of the intestine is usually rapidly progressive, and a fatal termination may be expected within six months or a year. A speedily fatal issue follows perforation, which sometimes takes place; usually death results from progressive loss of flesh and strength, with eventual œdema of the lungs. Frequent complications are hydronephrosis and fibrous nephritis, due to stenosis of one or both ureters from the extension of the cancerous infiltration to their wall. A fatal termination is often hastened by the production of a cancerous peritonitis, or by the occurrence of a septic infection in consequence of the formation of a recto-vesical fistula. Death sometimes occurs suddenly and unexpectedly from embolism of the pulmonary artery secondary to a thrombosis, which occasionally occurs in the iliac vein.

DIAGNOSIS.—The cancerous nature of the ulcer or stricture of the intestine which is invisible or beyond the reach of the finger is usually to be diagnosticated only by means of an exploratory operation, and the cancerous nature of a tumor of the intestine is often to be arrived at only by exclusion, unless the removal of a fragment of the larger and softer growths by means of an aspirator has led to the positive diagnosis of its nature. Fæcal tumors are eliminated by appropriate treatment. An occasional source of error is chronic appendicitis, which may produce an induration in the vicinity of the cæcum and be combined with fæcal retention and slimy dejections. In doubtful cases exploratory laparotomy is to be recommended, since a cure is to be expected if the disease is of inflammatory origin, and the prognosis is not changed if cancer is present. Most important in the exclusion of other abdominal tumors which might simulate cancer of the intestine is inflation of the bowel, which serves to show the relation of the tumor to the course of the large intestine, its usual seat.

PROGNOSIS.—The prognosis of cancer of the intestine when treated medically is invariably fatal. When treated surgically the result varies largely in accordance with the seat of the tumor and the time of its discovery. If low down in the rectum, where it is often early recognized, permanent or prolonged freedom from the disease has repeatedly been observed after operation. The prognosis of cancer in other portions of the intestinal tract is to be considered as fatal, since the symptoms of its presence usually become manifest at a time when the disease has extended to other parts and complete removal is impossible.

TREATMENT.—Medical treatment of cancer of the intestine consists solely in the relief of pain by opiates and the combating of obstruction by suitable diet, laxatives, and enemata, and, in case of eventual need, by punctures of the intestine with a hollow needle. J. G. Blake succeeded in keeping a patient alive for eighteen weeks during which there was complete obstruction. During this time the intestine was punctured one hundred and fifty times, some eight ounces of liquid fæces being removed each time.

CONSTIPATION. OBSTIPATION. COSTIVENESS.

DEFINITION.—Sluggish action of the bowels.

ETIOLOGY.—The intestinal contents are forced onward as the result of peristalsis ; from twelve to twenty hours are necessary for their passage from the cæcum to the anus, although but four hours are required for their journey from the pylorus to the cæcum. The arrest of the peristalsis by mechanical obstruction is considered in the article on intestinal obstruction. When peristalsis is checked by atony of the muscular coat from congenital weakness or acquired degeneration, by deficient nervous excitability, or by peculiarities of the contents, persistent constipation is the result. Congenital weakness may be the cause of the enormous enlargement of the colon which is at times seen in young children, and which persists despite the induction of free evacuations by means of appropriate treatment. Acquired degeneration of the muscle is of frequent occurrence in chronic catarrhal enteritis, in chronic peritonitis, and in amyloid disease of the intestine. Deficient nervous excitability may be due to organic disease of the brain or spinal cord, or to functional derangement, as in neurasthenia, hysteria, and certain forms of insanity, or to local affections of the intestine, as chronic passive congestion or intestinal catarrh. The excitability of the nervous apparatus of the intestine varies in individuals, and is weakened by sedentary habits and negligence. The intestinal contents become abnormal and cease to produce the necessary excitation both from an excess and from a diminution of vegetable constituents. A deficiency of liquid, whether due to a dry diet or to profuse sweating, as in excessive muscular work or fever, is of marked importance in the causation of constipation; but an abundance of milk in some persons produces this result. Muscular spasm in the lower part of the rectum, oftenest excited by a painful fissure of the anus and sometimes by ulceration of the mucous membrane, irritable prostate, a retroflexed uterus, or a displaced ovary, at times proves a cause of obstruction. Many writers assign importance to weakness of the abdominal muscles resulting from repeated pregnancies or due to the excessive accumulation of fat.

SYMPTOMS.—The effects of habitual constipation vary extremely, but are most exaggerated in persons of a nervous temperament, who complain of headache, dizziness, mental sluggishness, depression of spirits, and wakefulness, with loss of appetite and a coated tongue. The nervous symptoms are frequently attributed to the absorption of the toxic products of decomposition in the intestine. Fæces and putrefactive bacteria, however, are the normal contents of the large intestine, in which the fæcal retention takes place, and there is no exact evidence that any undue absorption of putrefactive products occurs.

The tendency of prolonged constipation is to the accumulation of fæces, resulting in fæcal impaction. Increasing distention of the abdomen then takes place, and distended coils of intestine are at times to be seen, especially in a thin person. The accumulated fæces are found especially in the rectum, sigmoid flexure, descending colon, and cæcum, more rarely at the splenic and hepatic flexures, and may be present simultaneously in various parts of the large intestine. Palpable tumors but little sensitive to the touch result, and are to be felt through the abdominal wall, and the nature of the mass when in the rectum is readily determined on digital examination. The local effects of the fæcal tumors vary considerably. The impaction of fæces in the rectum usually gives rise to frequent distress from the constant desire for evacuation, although only a small quantity of slimy matter escapes. In consequence of the pressure of the mass upon the wall of the rectum there is passive congestion, indicated by piles and leucorrhœa, or pain when the pelvic plexus of nerves is compressed. Impacted fæces elsewhere in the large intestine may prove discomforting from their weight and mobility, and may be mistaken for an abdominal neoplasm. Ulceration of the mucous membrane in contact with the fæcal mass may occur, and attention has been called to the possibility of the production of strictures in the healing of such ulcers. Ulceration of the cæcum rarely results from the presence of fæces in this part of the bowel, but painful tumors in the right iliac fossa may be due to the association of appendicitis with fæces in the cæcum. Retention of scybala in diverticula of the colon may be followed by an inflammation of the wall, extending to the peritoneum or into the mesocolon. Fæcal retention in the sigmoid flexure is an important element in the production of twist of this part, partly by elongation of the loop resulting from the long-continued traction, and partly because the weight of the loop facilitates its turning. The occurrence of attacks of acute intestinal obstruction from impacted fæces has already been mentioned.

Diagnosis.—The diagnosis of chronic constipation is usually readily made from the history of the case and from the effect of treatment. It is to be remembered, however, that, although one daily evacuation of the bowels is the custom of most healthy adults, exceptional persons are found in whom one movement every three or four days is considered to be normal. It is also important to bear in mind that frequent movements of the bowels and abundant slimy discharges may be associated with and result from chronic constipation. The discharges may appear normal when the impacted fæcal mass is tunnelled or channelled, but are usually hard, dry, and lumpy, sometimes resembling sheep-dung.

Treatment.—In the treatment of chronic constipation it is a matter of the first importance to remove the cause. In a large proportion of cases constipation in part or altogether depends upon sedentary habits, so that systematic graded exercise must be insisted upon, in the open air if possible, in the gymnasium or training-quarters if necessary. Along with the general exercise, which should be carried far enough to get the person into good muscular condition, should be associated movements which are especially adapted to strengthen the abdominal muscles and increase the activity of the abdominal circulation; many of the so-called "Swedish" movements are valuable for this purpose. They should be taught to the patient and rigorously carried out daily. In some cases good is achieved by the patient lying on his back and rolling around and around on the abdomen daily for ten to twenty minutes a large ball of heavy wood or iron. The habit of defecation at a certain time must also be formed; for most persons the morning hour is the most convenient; the time of day, however, is a matter of no importance from the purely medical stand-point, though the daily regularity is essential.

Constipation is very often associated with hypochondriasis, so that care must be exercised not to enhance the importance of the symptom in the sensitive consciousness of the patient.

If, as is frequently the case, the subject habitually uses fluids in small amount, the habit of free water-drinking should be formed, in the hope that the intestinal as well as the other secretions will be rendered more abundant. At least in the United States, it is well to caution the patient against the taking of very large quantities of ice-cold drinks, which are especially deleterious when there is chronic gastric catarrh or atony of the digestive organs. From eight to sixteen ounces of water drunk at bedtime, or upon rising in the morning, or, better, at each time, are often distinctly effective in promoting morning defecation.

The character of the food taken must be adapted to the individual case. The law is that the greater amount of the residue incapable of digestion in the food the greater its laxative influence; witness the contrasting habitual conditions of the dog and the cow: hence laxative articles of food are—fresh or dried fruits, all green vegetables, and various grains ground entire, that is, without separation of the hull from the starchy interior. Sugars and substances containing them are laxative, although they are altogether digested. Among the individual articles of food, cracked oats and rolled oats stand pre-eminent; they are, however, probably not superior to rolled wheat as a laxative, and are distinctly less digestible. Graham and other forms of bread made of unbolted flour are much superior to white bread. Rice is scarcely laxative. Oils, especially vegetable oils, such as that of the olive, are mostly laxatives, and when they can be digested are very valuable additions to the diet. Especially is this the case when along with the constipation there is a tendency to failure of the general nutrition. Some persons with very feeble digestion can assimilate considerable quantities of sweet oil, while others whose general digestion seems much superior reject the oil. Trial in the individual case affords the only test; we have seen excellent results achieved by giving one to two tablespoonfuls of sweet oil after meals. Often a dessertspoonful of whiskey may be advantageously given with the oil. A practical difficulty in the food management of constipation is that in most cases the condition is associated with feeble digestion, and that to digest food containing a large amount of indigestible matter is beyond the power of the patient. Very careful regulation of the diet in relation to the individual case is therefore essential.

Medical treatment of constipation is to be avoided if possible; very frequently, however, it is a necessary evil. The principles in the administration of drugs are, first, to avoid their employment as much as possible; second, to use them, if at all, in small quantities regularly day by day, not allowing the patient to become constipated and then giving a purgative dose, but seeing that a passage from the bowels is obtained each day; third, to change the drug or the combination of drugs at short intervals, so as to prevent the intestinal tract from becoming accustomed to any one remedy. Enemata, glycerin or gluten suppositories, and similar contrivances, by acting upon the rectum and lower colon, produce fæcal discharges; but it is evident that they are much less effective than are laxatives in emptying the upper portion of the colon. Further, if continually used they produce an obtuseness of the rectum which is unfortunate for the patient: hence their employment in chronic constipation should be limited to an occasional use as substitutes for laxatives, or for the purpose of obtaining a stool when the laxative has failed to act.

Laxative drugs may be divided into the saline and the vegetable laxatives. Among the saline laxatives must be placed the various natural mineral waters, too numerous for mention, which are so fashionable, but which are probably little better than artificial combinations. These combinations may vary indefinitely; formulas 2 and 3 may be used, or, especially when there is a tendency to hepatic torpor, formula 19. On the other hand, a single saline, such as Rochelle, Epsom, or Glauber's salt, or magnesium citrate, may be administered by itself. The saline should always be given immediately upon getting out of bed in the morning, and should be taken in half a pint of water, hot or cold, according to the condition of the individual patient.

Among the vegetable laxatives may be mentioned extract of cascara sagrada, solid or fluid, or in the form of an elixir; compound liquorice powder; the so-called A.B.S. pill (aloin, gr. $\frac{1}{6}$; strychnine, gr. $\frac{1}{60}$; extract of belladonna, gr. $\frac{1}{12}$); alcoholic extract of colocynth, given preferably in combination with extract of belladonna; and preparations of senna, of resina podophylli, or of rhubarb. An alkaloid which has not been much used, but from which we have seen extraordinary results in chronic constipation, is eserine: it acts simply as a stimulant of the muscular coat of the bowels, and is especially valuable in elderly and other people in whom the intestinal muscular fibres are failing in power; by its use the amount of laxative required may often be very greatly reduced. The ordinary dose is from one-fortieth to one-thirtieth of a grain, though one-twentieth may be given with impunity. The objection to all laxative pills is to be found in the difficulty of varying the dose. Liquid preparations have the advantage over pills that the dose can be more readily changed and graded. An old combination whose use has given us more satisfaction than almost any other laxative is formula 20.

Ordinarily it is desirable to give vegetable laxatives at night, because they require some hours for their action. When, however, constipation is obstinate, the best results are sometimes achieved by administering the vegetable laxative either after each meal or after the mid-day and evening meals. Cascara sagrada especially acts well when administered in this way. It is also better to give eserine rather by such method than in a single very large dose at bedtime.

Much difficulty is sometimes encountered in the removal of *impacted fæces.* It is essential in all procedures to avoid as far as possible the production of irritation: hence great gentleness should be used and irritant drastic cathartics should be avoided. The mass should be attacked simultaneously from above and from below. It is better to give small doses of the laxative, repeated at short intervals, such drugs being selected as will especially cause free watery exudation and softening of the mass. Probably in the majority of cases the best results are to be obtained by giving calomel (one-quarter to one-half grain) and a saline (two drachms of Epsom or Glauber's salt, or two ounces of solution of magnesium citrate), so alternated that the patient shall take one or the other every two hours. Not more than eight grains of calomel in all should be administered. Combinations of vegetable drugs may also be used, and sometimes it is allowable to employ in such combination croton oil in doses of one-sixth of a drop. More generally useful is the old-fashioned "black draught" of senna and Epsom salt. When a very hard fæcal mass can be felt in or above the rectum, it may be advisable, and is sometimes necessary, to remove it with the finger, aided by a spoon or other appropriate instrument.

Injections are to be freely used, and, when it is possible to get them above the fæcal mass, or well into it, are often very efficient. In order to increase their softening power, they should be given as hot as can be borne. A warm thick mucilage of flaxseed or pure linseed oil is often comforting, and by lubricating the parts lessens the pain of delivery. In very severe cases it may be necessary from time to time to give the patient rest during the process of removal, so that the parts may recover from irritation.

CHAPTER IV.

DISEASES OF THE LIVER, GALL-BLADDER, AND BILE-DUCTS.

DISEASES OF THE LIVER.

MALFORMATION.

CONGENITAL and acquired deformities of the liver are to be recognized. The former, the lobulated liver, is usually the result of syphilis, and when it produces symptoms they are those of a fibrous hepatitis.

Acquired deformities of the liver result from prolonged pressure applied either to the waist, as from the corsets of women or the belts of men, or to the liver directly, by tumors or by the ribs in curvature of the spine. The extreme result of such pressure is a localized atrophy of the liver, with the production of a fibrous band, in which blood-vessels, lymphatics, and bile-ducts are so obstructed that their peripheral branches become dilated. This band may serve as a sort of hinge, permitting such undue mobility of the separated portion of the liver that the latter is sometimes mistaken for an abdominal tumor. This error is especially apt to occur in those rare cases in which a portion of intestine overlies the atrophied part of the liver. It is possible that a sensation of pressure or weight in the region of the liver may result, and that attacks of passive congestion of the dependent portion may produce pain, vomiting, and weakness. Jaundice rarely occurs.

MALPOSITION.

Displacement of the liver may exist at birth or be acquired later in life. Hernia of this organ and its transposition to the left side are congenital. Acquired displacements are the result of pressure from below, as from an abdominal tumor, ascites, or meteorism, in which case the liver lies abnormally high. It lies unusually low when pressure is applied from above, as from air or fluid in the pleural cavity or beneath the diaphragm, from an emphysematous lung, from deformity of the thorax, or from an intra-thoracic tumor. Such displacements are of more importance in calling attention to disease elsewhere than in causing disturbance in the function of the liver.

A *wandering* liver is sometimes found, especially in middle-aged women who have borne children. It is probable that there are an elongated suspensory ligament and a lax abdominal wall. Muscular strain and tight lacing act as favoring causes. If productive of symptoms, it causes a sensation of weight, increased on exertion, and of pain, at times severe, referred either to the liver or to the right shoulder, or to both, and is sometimes accompanied with jaundice. Such patients are often neurasthenic or hysterical. In extreme cases the lower edge of the liver may lie in the right iliac fossa. The finger-tips may be pushed between the costal cartilages and the upper border of the liver. The outline of the anterior edge of the liver is easily traced, and the liver may be returned to its place. Such characteristics are usually sufficient to differentiate the wandering liver from ovarian, uterine, or renal tumors, or from cancer of the omentum.

Treves has shown that this variety of displacement may be relieved by surgical treatment.

FATTY LIVER. FATTY INFILTRATION OF THE LIVER.

Fat may be present in the cells of the liver as a result of degeneration of the protoplasm or because of its accumulation in the normal protoplasm. The former condition is considered in the article on acute parenchymatous hepatitis. The accumulation of fat, resulting in the fatty liver, is of clinical importance in explaining the occurrence of enlargement of the liver, sometimes considerable, without significant symptoms.

The liver is one of the storehouses of fat, which may remain within its cells for a longer or shorter time. The removal of fat from the liver takes place by its oxidation in the blood and its elimination with the bile. A fatty liver is likely to be found in persons eating excessively of fats, sugars, and starches. Sedentary habits, chronic alcoholism, extreme anæmia, wasting diseases, chronic diarrhœa, rickets, and malaria check the oxidation of fat, and thus favor its retention.

There are no symptoms characteristic of a fatty liver. The associated disturbances, as loss of appetite, retching, nausea, vomiting, constipation, or diarrhœa, though often attributed to obstruction of the portal capillaries by the surrounding liver-cells filled with fat, have no necessary dependence upon the latter condition. Such conditions as ascites and enlargement of the spleen are absent. Jaundice also is lacking. A sensation of fulness and weight, perhaps of epigastric pain, may be complained of.

The anterior edge of the liver may lie as low as the navel, but is often palpated with difficulty from the usual presence of a large quantity of subcutaneous and subperitoneal fat. The fatty liver is also soft.

If the physical examination indicates enlargement of the liver, its fatty nature is to be determined by a knowledge of the etiology of this condition. Hypertrophic cirrhosis is excluded by the absence of jaundice and by the failing resistance of the enlarged liver. An amyloid liver is hard, and the patient is cachectic, dropsical, and has albuminuria. The enlarged liver in leukæmia is dense, and the examination of the blood makes certain the diagnosis of this affection. The enlargement due to cancer is usually resistant and often irregular and sensitive; jaundice is frequent, and cachexia is rapidly progressive.

TREATMENT.—The treatment of fatty infiltration of the liver is that of obesity. (See page 59.) Alcoholic drinks and tobacco should be absolutely forbidden, and a light diet strictly enforced.

CONGESTION OF THE LIVER.

This term, though frequently employed as explanatory of certain digestive disturbances, is often given exaggerated importance. Active and passive congestions are to be distinguished, the former occurring under physiological as well as pathological conditions, the latter the result of disease outside the liver.

ACTIVE CONGESTION OF THE LIVER.

Active congestion of the liver, due to the entrance of an increased quantity of portal blood, occurs after meals, and should be regarded as pathological if a person is a glutton or accustomed to highly seasoned food, alcoholic excess, and abstinence from physical exercise. Infection and traumatism are usually regarded also as causes. The congestion due to arrested catamenia, to ovarian or uterine irritation, or to suppressed hemorrhoidal flow is considered to be a vaso-motor disturbance. The symptoms usually attributed to congestion of the liver are those which may be due to a gastro-duodenal catarrh, and are accompanied by a sensation of weight and discomfort in the region of the liver, with an increase in the size of this organ.

TREATMENT.—The treatment of acute congestion of the liver varies with the cause. If the latter be an infection, the treatment is that of the infection. Hepatic congestion, due to habitual over-eating, to alcoholic excess, or to other similar cause, is so universally associated with gastro-intestinal catarrh that the treatment is largely that of this disorder ; ordinarily one-eighth to one-fourth grain of calomel may be given every two hours until free purgation is produced ; or, if this latter do not occur in a reasonable time, salines may be administered. In sthenic cases the diet should be reduced temporarily to a very low point, but when. as in alcoholics, the hepatic congestion is associated with general vital depression, animal broths or other easily digested nutritious food may be required.

PASSIVE CONGESTION OF THE LIVER.

ETIOLOGY.—Persistent obstruction to the outflow of blood through the hepatic vein produces passive congestion of the liver. Such obstruction is most frequently caused by uncompensated valvular disease of the heart, or by disease or degeneration of the myocardium. Next in frequency are the causes of obstruction to the flow of blood through the lungs, as asthma, bronchitis, fibrous pneumonia, emphysema, atelectasis, and chronic pleurisy. Thoracic aneurism, mediastinal tumors, pleuritic effusion, or a deformed spine may obstruct the outflow of blood from the liver. Obstruction of the inferior vena cava by aneurisms and tumors may produce a like result. Obstruction of the hepatic vein may be due to growths from its wall or to constriction from a periphlebitis.

Limited portions of the liver may become passively congested by the obstruction or obliteration of branches of the hepatic vein by tumors, thrombi, or tight lacing.

MORBID ANATOMY.—At first the liver is symmetrically enlarged, purple in color, and diminished in consistency. The surface is smooth and shining. On section of the organ abundant blood escapes, and the dark-purple centres of the lobules are sharply differentiated from the paler peripheral portions. In the later stages the peculiar appearances have given rise to the terms *nutmeg atrophy* and *red atrophy*. The liver is diminished in size, is of a reddish-brown color, and is increased in consistency. The capsule is wrinkled and opaque. On section the surface is largely of a dark reddish-brown color corresponding to the central regions of the lobules, which are separated by gray or yellow lines or spots in the region of the portal vein. Pigment-granules in abundance and an increase of fibrous tissue are found on microscopical examination. The spleen and pancreas are denser and darker than normal, and the radicles of the portal vein in the stomach and the intestine are dilated.

SYMPTOMS.—In the stage of enlargement there is a sensation of fulness and weight in the right hypochondrium; there may be shortness of breath and pain on slight exertion, accompanied by a palpable resistance in the right hypochondrium and by epigastric pulsation, and the anterior border of the liver may be found below the navel. Considerable changes in the apparent size of the liver at times rapidly follow active exercise. The heart-sounds may be readily heard on auscultation over the liver where it is superficial, provided the abdominal wall is lax. Percussion is usually less valuable than palpation in determining the lowermost outlines of the enlarged liver, from the ready transmission of intestinal resonance through its anterior edge.

In the later stages of passive congestion digestive disturbances, as loss of appetite, nausea, vomiting, belching, and epigastric pain, become conspicuous. Jaundice may be associated, but is usually slight, and in cardiac cases the combination of blue, from venous congestion, and of yellow, from jaundice, may produce a greenish tint of the skin. The urine and fæces are usually not indicative of obstruction to the outflow of bile. Dropsy, both ascites and anasarca, may eventually occur. The physical examination of the atrophied liver shows a considerable diminution in the area of hepatic dulness.

TREATMENT.—The radical treatment of passive congestion of the liver is the removal of its cause. In most cases this is impossible, but it is often of the greatest importance to stimulate hepatic action. Hence the value of mercurials in chronic heart disease. Further, the hepatic congestion is frequently accompanied by congestion of the mucous membrane of the stomach and intestines: hence saline purgatives, such as sodium phosphate, which unload the portal circulation and stimulate biliary secretion, are often of great service.

There are so many cases of deranged hepatic function associated with congestion and a tendency to secretion of thick bile, and often to catarrhal inflammation of the ducts, that especial notice of their therapeutic management seems required. This condition is often enormously benefited by the treatment at Carlsbad, Vichy, and other saline and alkaline springs, a treatment which consists essentially in regulation of the diet and exercise and in the administration of large quantities of alkaline and saline waters. We believe that if the patient can be controlled at home, all that could be accomplished at Carlsbad can be reached by a parallel home treatment.

In a large proportion of cases it is primarily necessary to reduce greatly the amount as well as to alter the character of the food taken. Sweets and all indigestible food should be forbidden. Even more deleterious are rich foods: fats and substances cooked in them or dressings containing them should be excluded. Fish may be used freely, meats moderately. In no case should more food be taken than is just sufficient to sustain the weight. Complete abstinence from alcoholic drinks is essential. Regular exercise, carried to the point of physical tire but not to that of physical exhaustion, not interrupted by weather or untoward circumstances, and steadily and progressively increased as the patient's strength increases, forms an essential part of the management of the case. If the patient's strength suffices, the exercise should be sufficiently severe to produce free perspiration, and should be followed by a rub-down. In cases of robust men, physical training at the hands of a professional trainer is often very useful. When the strength is so reduced that active exercise cannot be taken, general massage should be used.

On first rising in the morning the patient should take from two drachms upward of a mixture of sodium phosphate, sodium sulphate, and potassium iodide in eight ounces of hot water. (See formula 19.) A drachm of sodium phosphate, twenty grains of sodium bicarbonate, and ten grains of potassium bicarbonate should be administered in a half-pint of hot water one hour before the mid-day and the evening meal. These doses of salines are to be increased or decreased according to their effect, the object being to produce distinctly profuse but not too weakening watery passages.

The occasional alternation of mercurial treatment for a short time with the saline and alkaline medication is often very advantageous. The mercurial should be combined with ipecacuanha and euonymin, whilst in some cases the purgatively more active resin of podophyllum may be added. In many cases the giving of a pill of calomel and ipecacuanha once a week is very beneficial. Nitrohydrochloric acid is frequently very serviceable: it may be exhibited during the saline treatment, but not near the time of the administration of mercurials. It is essential that the acid be freshly made, of a distinct reddish color, and be administered after meals in doses of from four to eight drops, well diluted in sweetened water, and taken with proper precautions for the protection of the teeth. The nitrohydrochloric acid should always be diluted at the time of its taking, since by rearrangement of its constituents diluted nitrohydrochloric acid in a little time becomes a mixture of dilute nitric and dilute hydrochloric acid. Nitrohydrochloric acid has been used in India to a considerable extent in the form of baths, but the advantages do not seem to equal the inconvenience. In many cases of chronic hepatic congestion the local application of nitrohydrochloric acid (one part to from twenty to thirty) by means of a saturated cloth covered with oiled muslin is very advantageous. The strength of the solution should be sufficient to produce a local sense of warmth, with prickling, but not to cause much irritation even after some hours of contact. The application usually provokes local sweating, acts, perhaps, as a counter-irritant, and probably yields products to absorption.

Ammonium chloride is a valuable remedy, which may be substituted for the alkaline mixture heretofore recommended before meals, or may be added to the alkaline treatment. From twenty to forty grains should be given in a tumbler of water two hours before eating, or two hours after meals if the patient is taking the alkaline mixture.

In continuing cases of hepatic congestion vegetable cholagogues are often serviceable from time to time. We have known good effects obtained by the use of a mixture of the fluid extracts of sanguinaria and leptandrin, each two parts, with one part of the fluid extract of podophyllum ; dose, from five to ten drops after meals, according to the effect upon the bowels. Extract of taraxacum has not seemed to us efficient.

PERIHEPATITIS.

Inflammation of the peritoneal capsule of the liver may be either acute or chronic, the former of extreme clinical importance, and, as subphrenic abscess, made especially conspicuous by Leyden, Mason, and others, the latter of but little significance. Either may be part of a general peritonitis, or of a peritonitis limited to the immediate vicinity of the liver.

ACUTE SUPPURATIVE PERIHEPATITIS.

DEFINITION.—A suppurative inflammation of the peritoneal capsule of the liver, resulting in the accumulation of pus between the diaphragm and the liver, the more common variety of the *subphrenic abscess*, or in the presence of air or gas and pus in the same region, *subphrenic pyopneumothorax.*

ETIOLOGY.—Acute suppurative perihepatitis may be due to direct violence, especially a penetrating wound. It more commonly results from a perforating ulcer of the stomach or duodenum, abscess or echinococcus of the liver, suppurative inflammation of the biliary passages, especially of the gall-bladder, appendicitis, pancreatitis, or abscess of the lung, spleen, or kidney. Remoter causes are to be found in inflammation of the uterus and tubes, perforation of the œsophagus, cancer of the stomach and œsophagus, and chronic tuberculosis of neighboring parts.

MORBID ANATOMY.—The peritoneum covering the liver and the corresponding surface of the diaphragm is thickened, opaque, without lustre, and covered with a fibrinous false membrane. The general peritoneal cavity is separated from that portion overlying the liver by fibrinous adhesions either to the left or to the right of the suspensory ligament, more frequently to the latter, according to the cause of the perihepatitis. The abscess may hold a quart or more of pus alone, or the pus may be mixed with air or gas, especially when perforation of the stomach or duodenum is the cause. When bile is mixed with the pus the latter is likely to be of a yellow ochre color and to contain bilirubin crystals. The pus may be fattily degenerated and crystals of fat acids be found.

SYMPTOMS.—Suppurative perihepatitis being secondary to diseased conditions elsewhere, its onset may be sudden or gradual, the former being particularly the case when perforation of the stomach or duodenum is the cause. The rapidly progressing cases are those in which the symptoms suggest a circumscribed peritonitis in the vicinity of the liver. Severe pain, often of sudden onset and increased on prolonged inspiration, and tenderness, are present in the epigastrium or the right hypochondrium. There is a continuous fever, sometimes preceded by a chill. Loss of appetite, nausea, and vomiting usually occur, and there may be slight jaundice. There is increased frequency of respiration. The physical signs closely resemble those resulting from pleurisy, which disease is sometimes associated with perihepatitis. They vary in degree according to the quantity of exudation or gas present. Distention of the right hypochondrium and epigastrium and immobility of the corresponding intercostal spaces are conspicuous. The degree of dulness on percussion varies in like manner, and dulness may be present as high as the fourth rib, varying with change of position. The lower line of hepatic dulness may be found on a level with the navel, where the anterior edge of the liver is then to be felt. There is an absence of respiratory sounds and vocal fremitus in the region of dulness, whereas the respiratory murmur in the upper part of the chest is exaggerated. The presence of air or gas beneath the diaphragm produces similar physical signs, with the exception that a tympanitic region overlies the dull area.

The course of suppurative perihepatitis may be prolonged over a period of months, and is then characterized by an irregular range of temperature, with progressive emaciation and debility. The pus may be absorbed, or be discharged into the pleural cavity, lung, stomach, or intestine, or through the abdominal wall, or even below Poupart's ligament.

Obstruction of the hepatic or portal veins or of the inferior vena cava may result from thrombosis or periphlebitis, with the production of congestive atrophy of the liver, ascites, or œdema of the lower extremities. The extra-hepatic bile-ducts may be compressed by the cicatricial tissue, and persistent jaundice follow.

DIAGNOSIS.—Etiology is of especial importance in the diagnosis. The rational and physical signs may be insufficient to determine whether the seat of the exudation is above or below the diaphragm. Absence of cough and of expectoration and slight displacement of the heart are in favor of perihepatitis. Bulging of the hypochondrium and extreme depression of the liver are unlikely to occur in pleurisy. The diagnosis is eventually to be made by exploratory puncture in the seventh or eighth interspace in the axillary line. According to Pfuhl, fluid below the diaphragm escapes more freely during inspiration, the reverse being the case in pleurisy. The presence of bile-pigment would favor the subphrenic seat of the exudation.

PROGNOSIS.—Acute perihepatitis, when fibrinous in character, may terminate favorably in a short time. Recovery from the suppurative variety may also take place by absorption or spontaneous evacuation of the pus. The latter is always a grave affection, and often fatal when representing a peritonitis from perforation. A considerable diminution in the mortality is likely to result from surgical treatment.

TREATMENT.—The early treatment of an acute perihepatitis should be that of a local peritonitis. After the formation of pus, surgical evacuation and drainage are strongly indicated.

CHRONIC PERIHEPATITIS.

Although chronic perihepatitis is of but little clinical importance, it is of relatively frequent occurrence. When circumscribed it represents the result of prolonged pressure in certain trades or from articles of dress, or it may be due to localized growths of cancer or syphilis or to the extension of a pleurisy. The affected peritoneal capsule of the liver is thickened and opaque. Fibrous adhesions may unite the liver to the diaphragm, stomach, colon, or abdominal wall, and the contraction of the fibrous tissue may cause atrophy of the liver and narrowing or obliteration of its ducts and vessels. When extreme, as a sequel of suppurative perihepatitis, it may be of serious importance from its mechanical effects upon the liver and its vessels and ducts.

ACUTE YELLOW ATROPHY OF THE LIVER. ACUTE PARENCHYMATOUS HEPATITIS.

ETIOLOGY.—This affection is of rare occurrence, and is more common among women, especially in the latter half of pregnancy, than among men. It usually occurs in adults, though it may be present at any period of life. Intense mental excitement and alcoholic excesses are mentioned as causes, and it has been found in the course of acute infectious diseases, as typhoid and relapsing fevers, diphtheria, pyæmia, and septicæmia. The importance of infection in etiology is suggested also by the frequent discovery of bacteria in the diseased liver and by the occurrence of a number of cases in a given locality within a short time. Phosphorus poisoning produces a similar alteration of the cells of the liver, and it may occur as a secondary condition in the course of severe jaundice or of fibrous hepatitis.

MORBID ANATOMY.—The liver is more or less enlarged at the outset, and in phosphorus poisoning may remain so until death. As a rule, it eventually becomes decidedly diminished in size and flattened. It is of dirty-yellow color and firm consistency, though flaccid. On section the color may be uniformly yellow and opaque, and the lobular regions indistinct, or there may be alternate patches of red and yellow. Crystals of leucin and tyrosin may form white specks after prolonged exposure of the cut surface to the air. Microscopical examination shows extensive and extreme fatty degeneration of the liver-cells, the fat having been absorbed from the red portions of the liver. The interstitial tissue is slightly infiltrated with leukocytes, and clumps of cells are to be found, which are regarded as new-formed bile-ducts or bands of liver-cells. Crystals of leucin, tyrosin, and bilirubin are also to be seen.

The spleen is hyperplastic. There is fatty degeneration of the epithelium of the kidneys and of the gastric glands, of the heart, and sometimes of the voluntary muscles. Small hemorrhages are present throughout the body, and the tissues are stained yellow. The pleural and pericardial cavities may contain an excess of fluid.

SYMPTOMS.—The characteristic symptoms of acute yellow atrophy are usually preceded by loss of appetite, nausea, vomiting, belching, irregular stools, tender epigastrium, headache, prostration, and slight jaundice. The symptoms then suddenly become severe. There is constant vomiting, eventually of a bloody fluid. There are intense headache, restlessness, delirium, convulsions, and coma. Nasal, gastro-intestinal, urinary, and cutaneous hemorrhages may occur, and abortion with excessive flowing is likely to take place in pregnant women. The temperature is not especially elevated until shortly before death, when it may reach 104° F., although it may be subnormal at this time. The pulse, slow at the outset, becomes rapid and feeble. The area of hepatic dulness rapidly diminishes, and may wholly disappear, although Gerhardt reports a case in which there was no change in dulness despite the atrophy of the liver, in consequence of adhesions between the liver and the abdominal wall. According to Riess, there is tenderness in the right hypochondrium even when the patient is comatose.

The urine is diminished in quantity, and its secretion may be suppressed. It is bile-stained, acid, sp. gr. 1012 to 1030, moderately albuminous, and contains bile-pigment, bile-acids, and hyaline and fatty casts.

Urea is greatly diminished or absent. Leucin and tyrosin are usually present, and should be sought for, if necessary, in the urine treated with acetic acid and evaporated.

DIAGNOSIS.—Acute yellow atrophy is to be suspected on the sudden onset of restlessness, delirium, and convulsions in a case of apparent simple jaundice. The great diminution or the absence of urea, the presence of leucin and tyrosin in the urine, perhaps of bacteria in the blood, and the diminution in the size of the liver, confirm the diagnosis. Phosphorus poisoning cannot be distinguished solely by the symptoms. The absence of leucin and tyrosin from the urine in poisoning, and the other differences which have been alleged to be diagnostic by various writers, have been proved not to be constant, and cannot be depended upon. The enlargement of the liver is more persistent in the poisoning, but without a history poisoning cannot be more than suspected unless phosphorus be recognized chemically in the contents of the stomach or intestines, or in the urine in a lower degree of oxidation than phosphoric acid.

The symptoms of parenchymatous hepatitis occurring in obstructive jaundice and fibrous hepatitis are to be differentiated from those due to acute yellow atrophy by the rapid progress of the latter.

PROGNOSIS.—Although recovery from the severer forms of parenchymatous hepatitis has been reported, the prognosis of this disease is almost invariably fatal, death usually occurring in the course of two or three days after the onset of the severer symptoms, and within a fortnight from the beginning of the attack. Exceptionally the disease has extended over a period of two months.

TREATMENT.—There is no known specific treatment. Symptoms are to be met as they arise.

SUPPURATIVE HEPATITIS. ABSCESS OF THE LIVER.

ETIOLOGY.—Abscesses of the liver are due to the entrance into this organ of pyogenic bacteria or amœbæ coli, rarely of actinomyces or coccidia, and sometimes of a chemical agent. The irritant may enter directly by means of a wound or by means of the blood-vessels or the bile-ducts.

Traumatic abscesses are due to traumatism and infection, and Dabney has shown that abscesses of the liver rarely arise from disease of the bones or of parts of the body other than those intimately connected with the liver. Embolic and thrombotic abscesses follow the admission of the pyogenic irritant by means of the blood-vessels, and infectious emboli are brought from the inflamed radicles of the portal vein in the parts of the intestine affected in appendicitis, dysentery, and piles; but typhoid ulcers rarely serve as a cause of hepatic abscess. Emboli may also be transferred from abscess of the spleen and from the inflamed umbilical vein of the new-born child. Infectious embolism of the hepatic artery, however, rarely causes hepatic abscess except in ulcerative endocarditis and in pulmonary gangrene. Osler and Ross have suggested that a bland embolus carried from an aneurism of the hepatic artery may produce abscess of the liver by the presence of pathogenic agents in that part of the liver to which the embolus is carried. An infectious embolus from a body vein may enter the hepatic vein by regurgitation and act as a cause of abscess.

Abscesses of the liver of vascular origin may result as well from thrombosis as from embolism, the infecting thrombus being directly continued from the inflammatory or ulcerative process in the radicles of the portal vein or from the umbilical vein into the liver.

The irritant invades the liver through the bile-ducts in cases of suppurative cholangitis continued through the common bile-duct from the intestine. Abscesses are then more likely to result if gall-stones, parasites, or foreign bodies are present in the bile-ducts.

It is probable that abscesses occurring in the tropics are due to the causes above mentioned, and their frequency may be accounted for by the prevalence of dysentery in the tropics, especially since Kartulis, Councilman, and others have shown the relation of the amœba coli to dysentery and abscess of the liver. Furthermore, Kiener and Kelsch have shown that dysentery was present with hepatic abscess in eighty-five per cent. of three hundred and fourteen cases.

MORBID ANATOMY.—The liver is usually enlarged symmetrically, and the outside may show no appearances suggestive of pus. On the other hand, the presence of opaque, yellowish-white, rounded patches covered with false membrane, and yielding to the touch, indicates the existence of subjacent abscesses. The latter are single or multiple.

Single abscesses generally result from the confluence of multiple abscesses. According to Waring's figures, the abscess is limited to the right lobe in two-thirds of the cases, and usually lies near the convexity. It may contain several quarts of pus. Its cavity may be crossed by bands and cords, the remains of the partitions between smaller abscesses; in acute cases the wall is shreddy, not sharply defined. The wall of the chronic abscess is grayish white, dense, and circumscribed. The contents are an opaque, viscid fluid, of a yellow or reddish color, containing leukocytes, fat-drops, granular material, and crystals of bilirubin. The presence of hooklets, amœbæ, actinomyces, or coccidia indicates the cause of the abscess concerned.

Multiple abscesses may be found throughout the liver, are often in groups, and as many as fifty have been observed. They may be as large as walnuts, sharply defined, irregularly rounded, or lobulated. The walls and contents resemble those of the solitary abscess. A thrombotic or embolic origin is indicated by the presence of arborescent patches of necrosis and of puriform thrombi in the branches of the portal vein. Abscesses originating from the bile-ducts are associated with dilated ducts containing a green pus.

As the abscess reaches the surface of the liver, adhesions are formed between this organ and adjacent structures, as the diaphragm, the stomach or the intestine, the renal pelvis, or the abdominal wall. Perforation may occur and the pus be evacuated, escaping into the peritoneal, pleural, or pericardial cavity, and perhaps finding an outlet at some point remote from the liver.

Solitary and small abscesses are at times transformed into cysts with viscid contents in which cholesterin is often found, or the pus, becoming inspissated, forms a cheese-like mass or a calcified nodule enclosed within a dense fibrous capsule.

SYMPTOMS.—There may be no symptoms calling immediate attention to abscess of the liver, its presence being first suggested by the escape of pus from some other organ, or being made evident at a post-mortem examination. Even suggestive symptoms of suppurative hepatitis may be wholly obscured by those of the disease to which it owes its origin. The patient loses flesh and strength, is likely to be troubled with nausea or vomiting, and complains of epigastric distress. Jaundice is often absent, or slight, and, if considerable, may be due to pressure of the abscess upon the larger bile-ducts. Late in the course of the disease the patient at times becomes mildly delirious, and still later coma often supervenes. Pain in the region of the liver is usually present either as an early or as a late symptom. It occurs as the suppuration nears the surface, and is referred perhaps to the seat of the abscess or to the right shoulder. The latter localization is to be explained by the transmission of irritation from the branches of the phrenic nerve in the capsule of the liver and in the suspensory ligament to the fourth cervical nerve, which also receives branches from the shoulder. The sensation of pain is thus capable of being referred through the central nervous system to a point remote from its origin.

The presence of suppuration is especially indicated by the prolonged elevation of temperature, although this may be so slight in cases of chronic abscess as not to be especially significant. More frequently exacerbations and remissions of temperature are present, with a rise perhaps as high as 105° F. The elevation of temperature, if continuous, with evening exacerbations, often suggests typhoid fever or tuberculosis, and if associated with chills and sweating, especially when these are at regular intervals, gives rise to the thought of malaria. As the disease progresses, a sudden fall of temperature often results from the evacuation of an abscess, and a prolonged lowering of the temperature not infrequently occurs some time before death. The frequency of the pulse and respiration is in accord with the variations of temperature. The pulse is weak, and the respiration often rapid and painful, and accompanied by a dry cough when the surface of the diaphragm is inflamed. If the abscess breaks into the lung, its contents, usually of a reddish color, according to Osler resembling anchovy sauce, have given evidence, on microscopical examination, of the presence of amœbæ. The action of the bowels is irregular. The stools are not likely to become clay-colored, but contain abundant pus if the abscess empties into the bowel. The source of this pus is made apparent by the discovery of hooklets, amœbæ, or other evidence of its parasitic origin. The urine is not especially noteworthy, except in those cases where the abscess empties into the urinary tract.

Enlargement of the liver, which is usually present, is made evident both by percussion and by palpation, and often becomes apparent on inspection, especially when the patient is in the upright position. The enlargement is irregular if the abscess projects above the surface, and may be indicated by a sharply defined area of thoracic dulness, convex upward when the projection is from the upper part of the right lobe. The size of the liver is in the main in proportion to the quantity of pus present. It is sometimes sufficiently enlarged to cause ascites from pressure on the portal vein, or anasarca from pressure on the inferior vena cava. The upper border of hepatic dulness may extend to the second rib in front and to the spine of the scapula behind, and the anterior edge of the liver be found near the crest of the ilium. Crepitation or fluctuation is sometimes recognized on palpation, and is indicative either of a localized peritonitis or of the seat of the abscess. The liver is tender to the touch when the anterior surface is inflamed, and the abdominal wall then becomes tense when palpation is attempted. The spleen is moderately enlarged.

The symptoms of abscess of the liver may continue for a period of years,—five in the case reported by Ewald,—or a fatal issue may be reached in the course of a few weeks. The average duration of fatal cases is from six weeks to three months. The smaller abscesses tend to coalesce, thus forming the large abscess, which extends towards the surface of the liver, with the eventual spontaneous evacuation of the pus. Immediate relief to the symptoms is then likely to occur; if, however, the drainage is defective and the destructive process continues, with or without the complications of hemorrhage, embolism, peritonitis, or septicæmia, permanent fistulæ may become established, with perhaps the eventual occurrence of amyloid disease.

DIAGNOSIS.—Time is an important factor in the diagnosis of suppurative hepatitis, which is based upon the association of protracted continuous or intermittent fever, painful, perhaps tender, enlargement of the liver, leukocytosis, and a thorough appreciation of the etiology of suppurative hepatitis. The most important element in the diagnosis is the aspirator; but the exploratory puncture may not reach the abscess, or the pus may be too thick to flow through the needle. If an abscess is suspected, repeated punctures should be made, if necessary, the places of election being tender or yielding spots in the enlarged liver below the costal cartilages, the seventh right intercostal space in the axillary line, and at this level in front or behind if dulness extends farther up. The necessity for aspiration is greater than its danger, although there may be profuse hemorrhage while the needle remains in the liver.

Malaria is to be differentiated by the more typical recurrence of the chills, the considerable enlargement of the spleen, the successful treatment with quinine, and the absence of the parasites of malaria. Pulmonary tuberculosis, which simulates hepatic abscess by cough and hectic, is to be excluded by the results of physical examination. Empyema may be suggested by the fever and physical signs, but in hepatic abscess dulness is higher in front than behind, while in empyema the retracted lung lies along the spine and at the upper part of the thorax. Incarcerated gall-stones in the large bile-ducts at times are mistaken for suppurative hepatitis, but in gall-stones the pain is usually more severe and the jaundice more extreme and constant, whilst the elevation of temperature is often slight and recurs with the attacks of pain. The echinococcus cyst may present the physical characteristics of a large abscess, but its growth is slow, afebrile, as a rule, and without disturbing symptoms, except those of mechanical origin.

PROGNOSIS.—Large abscesses or many small abscesses of the liver are always dangerous, the mortality in cases not treated surgically being as high as eighty per cent. The possibility of recovery from small single abscesses by absorption or calcification has already been mentioned. Recovery from more extensive suppuration is possible only by the efficient evacuation of the pus. It is stated that one-half of the cases of evacuation of the abscess through the lungs or gastro-intestinal canal recover, and the mortality may be as low as thirty per cent. in cases treated surgically.

TREATMENT.—Septic abscesses of the liver are usually not amenable to any treatment, nor is there any known method of checking the formation of the primary abscess; the medical treatment must, therefore, be symptomatic and palliative. The diet should be largely or altogether liquid or semi-solid, unirritating, but nutritious and supporting.

The surgical treatment consists in complete evacuation of the pus and drainage of the cavity, for aspiration is of little value save as a means of diagnosis. The decision of the time at which the cavity shall be opened is often a point of great nicety: on the one hand, there is danger of the rupture of the abscess into the peritoneum or some other vital part, and also of exhaustion of the patient; on the other hand, there is the reasonable expectation that the formation of adhesions between the liver and the abdominal wall will greatly favor safe evacuation. When an abscess has discharged through an internal organ, such as a lung or the intestine, the case should usually be left to nature, unless increasing hectic fever and failure of strength point strongly towards approaching death, in which case the effect of a counter-opening may be tried.

FIBROUS HEPATITIS. CHRONIC INTERSTITIAL HEPATITIS. CIRRHOSIS.

Etiology.—Fibrous hepatitis is a disease more frequent in man than in woman. Although usually occurring in the adult, Howard and Hatfield have collected a considerable number of cases among children, and it may be present in the fœtus. It is generally considered to be the result of the continued presence in the liver of an irritant brought by the blood-vessels, especially the portal vein, or entering through the bile-ducts or directly from the surface. In nearly two-thirds of the cases the irritant is alcohol, especially that obtained from the fermentation of grains and potatoes. An unknown predisposing cause on the part of the individual is also probable, since many persons addicted to the excessive use of alcohol are free from this disease. The undue use of strong wines and beer sometimes gives rise to fibrous hepatitis. It is asserted that spices, coffee, ptomaines, phosphorus, arsenic, and antimony may act as causes, —a view based rather upon experiments than upon clinical evidence. Acute infectious diseases, as malaria, typhoid fever, scarlatina, cholera, and dysentery, are also regarded as causes. If they are to be admitted as such, their importance must be slight, owing to the rarity of fibrous hepatitis in comparison with the frequency of these diseases. Chronic infectious diseases, especially syphilis, whether congenital or acquired, produce cirrhosis, and miliary tuberculosis, rickets, and gout are maintained to be of etiological importance. Welch, in his observations on the pigmented liver of coal-miners, calls attention to the possible significance of a mechanical irritant, as coal-dust. Botkin maintains that fibrous hepatitis may result from obliteration of the portal vein, and Eichhorst favors the etiological importance of old age in producing an arterio-sclerosis with resulting atrophy, as in the kidney. The nutmeg atrophy from continued obstruction in the course of the hepatic vein causes a slight degree of fibrous hepatitis.

The importance of the bile-ducts in the production of fibrous hepatitis is seen in cases of chronic cholangitis. This affection in turn may be the result of gall-stones, tumors, or tuberculosis, or of congenital obstruction, stenosis, or obliteration of the bile-ducts.

Fibrous hepatitis proceeds from the surface of the liver in consequence of a perihepatitis when a chronic inflammation of the capsule exists, either limited or part of a chronic peritonitis.

Morbid Anatomy.—Chronic fibrous hepatitis is characterized by an increase of the connective tissue of the liver, usually associated with a destruction of liver-cells, and resulting in a shrinkage of the organ. Sometimes the liver-cells are not destroyed, the fibrous tissue does not shrink, but an enlargement of the liver results. Two varieties of fibrous hepatitis are thus to be recognized,—the one hypertrophic, the other atrophic, cirrhosis. The former is manifested by a symmetrically enlarged liver, weighing perhaps eight pounds, in which a new formation of connective tissue is more directly connected with the bile-ducts, and the liver-cells are either unaltered or fattily infiltrated.

In atrophic cirrhosis the liver, at first increased in size, may become so shrunken as to weigh but a pound. The atrophy is asymmetrical, and produces various degrees of deformity. Granules and nodules from the size of a pin's head upward project from the surface, and according to the predominance of larger or smaller projections the terms "granular liver," "hobnailed liver," and "lobulated liver" are applied. The capsule is thickened and opaque. The color varies from gray to a tawny yellow (*κιῤῥός*), whence the term "cirrhotic." The yellow shades of color are dependent upon the presence of fat and biliary coloring matter. The consistency of the liver is increased, and is often compared to that of leather. The growth of fibrous tissue proceeds rather from the vicinity of the portal vessels than from the neighborhood of the bile-ducts, and its shrinkage causes rather narrowing and obliteration of the former than obstruction of the latter. These may be affected to a certain degree, and clusters of cells indicative of new-formed bile-ducts may be seen.

The spleen is usually enlarged, and may become thrice the normal size. Its enlargement is chiefly the result of the portal obstruction, and is the greater the later the stage of the disease. The enlargement may be in part due to the cause of the cirrhosis, since the former is sometimes found before any considerable degree of ascites is present. If the capsule is thickened or has undergone marked atrophy, decided enlargement is prevented. The peritoneum is thickened and opaque, sometimes granular, and the mucous membrane of the stomach and the intestines is swollen and its blood-vessels dilated.

Owing to the obstruction to the flow of portal blood through the liver, the radicles of the portal vein are distended, and their anastomoses with the peripheral veins of the body dilated. The latter are to be found at the junction of the œsophagus and the stomach, along the course of the large intestine, in the lower part of the rectum, and in the retroperitoneal plexus of veins in front of the spine. Dilatation of the anastomosing branches in the suspensory and round ligaments also takes place, and is sometimes continued to the cutaneous veins around the navel, forming the caput Medusæ. Branches of the internal mammary and epigastric veins may also become dilated and tortuous.

SYMPTOMS.—There are no symptoms characteristic of the early stages of cirrhosis of the liver. As the disease advances and the destruction of the liver-cells and the obstruction of the portal circulation take place, disturbances of function appear, and sometimes develop with great rapidity. The obstructed portal circulation produces a chronic gastro-intestinal catarrh, which is manifested by loss of appetite, belching, nausea and vomiting, flatulence, and irregular action of the bowels. After a while hemorrhages are likely to occur, either as nosebleed or as bleeding piles, or, in consequence of the portal congestion, the blood may regurgitate from the œsophagus, be vomited from the stomach, or escape with the stools. The attacks of hemorrhage are often repeated, or a large quantity of blood is lost in a single attack, and in consequence persistent anæmia is the frequent result. Such hemorrhage may be the first symptom to excite suspicion of the existence of cirrhosis, and its occurrence may afford temporary relief to the digestive disturbances dependent upon portal stagnation. Cutaneous hemorrhages, which usually take place late in the disease, are dependent rather upon degenerative changes in the walls of the blood-vessels than upon mechanical obstruction.

Ascites eventually develops, and may be the first symptom to attract the patient's serious attention. It is due to the obstruction of the portal circulation, is usually slow in development, unless thrombosis of the portal vein occurs, and may result in the presence of several gallons of fluid. The characteristics of such fluid will be found in the article on ascites. The larger its quantity the greater the discomfort, chiefly manifested by the increased intra-abdominal pressure, which may vary from hour to hour or from day to day in consequence of the absorption of fluid, the action of medicines, the escape of gas from the stomach or intestines, or the occurrence of hemorrhage. If the fluid is removed by tapping, it usually reaccumulates within a few weeks, the more rapidly the more frequent the withdrawal. Jaundice is not an essential symptom of cirrhosis of the liver, although slight and perhaps recurrent attacks may occur. These are due rather to causes outside the liver, especially catarrh of the common bile-duct, than to pressure upon the bile-ducts within the liver.

The progressive destruction of the liver-cells results in more extreme disturbances of nutrition. Emaciation becomes conspicuous, and the patient is weak and irritable. The breathing is rapid and labored, partly in consequence of ascites. The heart is weakened, and the pulse correspondingly feeble. Œdema of the skin, especially of the feet and legs, the scrotum, and the dependent portions of the abdomen, occurs, as the result partly of the enfeebled heart-action and partly of the portal obstruction. Eventually hydrothorax and œdema of the lungs are likely to take place. Finally, profound disturbance of the nervous system, as delirium, convulsions, and coma, immediately precedes death.

Evidence of emaciation is seen especially in the face, arms, and hands, which strikingly contrast with the bloated portions of the abdomen and legs. The skin is pale, of a dirty-gray color; at times it shows a yellowish tint from complicating jaundice. The tongue is usually dry and coated. The abdominal distention, unless extreme, varies with the position of the patient. The navel protrudes. The veins at the dependent portions and perhaps near the navel are conspicuous. (See also Ascites.) The physical examination of the liver usually becomes possible only when the ascites is relieved by tapping. The outlines of the shrunken liver then may be determined, and its granules or lobules often be felt owing to the lax condition of the abdominal wall and the excessive mobility of the liver when not adherent. The enlarged spleen is also more readily recognized after the fluid is removed, although it is often possible to determine its outlines by percussion and palpation if the patient can lie on the right side.

DIAGNOSIS.—In the early stages of cirrhosis the diagnosis is impossible. When hemorrhage from the stomach or bowels is the first important symptom, fibrous hepatitis is to be suspected if there is an alcoholic history and there are no symptoms pointing to ulcer of the stomach or bowels. A similar history with or without hemorrhage but with the development of ascites also makes probable cirrhosis. The recognition by physical signs of a granular or nodular atrophied liver establishes the diagnosis. Previous to the withdrawal of fluid from the abdomen it is often impossible to recognize fibrous hepatitis, since a correct explanation of the cause of ascites has frequently been made only after a laparotomy or on the post-mortem table. In brief, the existence of cirrhosis of the liver is most likely to be made when the liver can be physically examined.

PROGNOSIS.—Fibrous hepatitis when capable of recognition is likely to prove soon fatal, death taking place within a few months or a year. On the other hand, patients have lived for years after the occurrence of hemorrhage and ascites, and considerable degrees of cirrhosis of the liver have been found after death in persons who had been supposed to be free from liver disease. A guarded prognosis is therefore to be made until the duration and severity of the symptoms, and the ability of the patient to preserve flesh and strength, have been thoroughly ascertained.

TREATMENT.—In most cases of cirrhosis of the liver the primary indication is the withdrawal of alcohol and all stimulating or irritating highly seasoned foods; in the case of tavern-keepers and bartenders who were unable to or would not cease using alcohol, we have known the substitution of hard cider for all other drinks apparently to protract life for a great length of time. Although in the advanced stages of the disease no medical treatment can effect anything, in the beginning it is the duty of the practitioner by the use of the remedies already spoken of under the head of chronic hepatitis to endeavor to bring about hepatic resolution. During the stage of contraction the treatment must be purely symptomatic. The patient's strength should be sustained by nutritious food, and the accidents of the disease appropriately met. (See Ascites.) Hemorrhage from the stomach due to cirrhosis of the liver is usually uncontrollable by treatment, which should consist in withdrawal of food and the administration of opium and styptics (especially Monsel's solution), as in hæmatemesis from gastric ulcer. (See page 842.)

HYPERTROPHIC CIRRHOSIS.

As has been stated, the liver may become persistently enlarged from an increase of its fibrous tissue intimately connected with the bile-ducts, but not differing in its histological characteristics from that found in atrophic cirrhosis. Hanot and Charcot maintained that this variety of cirrhosis originated from the bile-ducts, and it has been asserted that a bacterial catarrh of these ducts may be of importance in etiology.

This affection is of sufficient frequency to be regarded as of clinical importance. Jaundice is the most conspicuous symptom. It may be either slight or severe, and is likely to be permanent. The absence of clay-colored stools indicates that there is no obstruction to the entrance of bile into the intestine. The disturbance of digestion is slight, and frequently for a long time the nutrition is unaffected. Loss of flesh and strength may finally occur, or there may be a rapid rise in temperature and an increase in the degree of jaundice, followed by delirium, convulsions, coma, and death in the course of a fortnight. There is no ascites, and hemorrhages are infrequent. Persistent enlargement of the liver is the chief abnormality, except the jaundice, to be found on physical examination, and the anterior border may lie as low as the navel. The surface is smooth, the density of the organ is increased, and there is no tenderness on palpation. Enlargement of the spleen is present. Hanot and Meunier have recently called attention to the presence of a leukocytosis at various stages of the disease, ranging from twelve thousand to twenty-one thousand white corpuscles in the cubic millimetre. Importance is attached to these observations, since leukocytosis is absent in alcoholic cirrhosis and is present to the above extent in cancer alone of the various affections of the liver. This condition is regarded as an argument in favor of the infectious nature of the disease.

Diagnosis.—Persistent jaundice, hepatic and splenic enlargement, absence of ascites, and gastro-intestinal hemorrhages enable a diagnosis of hypertrophic cirrhosis to be made. The enlargement of the liver may be simulated by fatty infiltration, amyloid disease, or cancer. The first is excluded by the presence of continued jaundice; the second, not only by this evidence, but also by pallor, debility, albuminuria, and dropsy, and by a lacking etiology. Time may be necessary for the elimination of cancer of the liver, which produces more rapid disturbance of nutrition and debility and is unlikely to be accompanied by enlargement of the spleen. The uncertainty of the diagnosis has led to an exploratory laparotomy. The disease has continued for a period of seven years; and it is not known that its progress has ever been prevented or delayed.

Treatment.—There is no known effective treatment of this disease. Symptoms should be met upon general principles; potassium iodide (one grain) and corrosive sublimate (one-sixtieth of a grain) may be given continuously, largely to relieve the patient's mind.

AMYLOID LIVER.

The presence of amyloid material in the liver is connected with the destruction of liver-cells, and is productive of corresponding disturbances in the function of the liver. Its symptoms are less conspicuous than are its physical signs in calling attention to disease of the liver. The immediate cause of amyloid disease in the liver, as elsewhere, is uncertain. Of practical importance from its bearing on diagnosis is its association with chronic suppuration, chronic tuberculosis, especially of the lungs, intestine, kidneys, bones, and joints, and the later stages of syphilis. An amyloid liver is sometimes found in cancer, rickets, malaria, gout, leukæmia, pseudo-leukæmia, and chronic nephritis. From the fact that amyloid degeneration usually affects various important organs of the body simultaneously, the evidence indicative of this affection of the liver depends upon the physical examination of this organ. Pallor, emaciation, debility, and dropsy are present. There are loss of appetite, belching, vomiting, and diarrhœa. The urine is likely to be of low specific gravity, pale, with abundant albumin. The liver, although neither painful nor tender, may be so enormously enlarged as to produce a sensation of fulness and weight, and afford a percussion dulness extending from the third rib to the crest of the ilium. The surface is smooth, the density increased. The spleen may be enlarged, but from amyloid disease, not from obstruction to the passage of its blood into the liver. A diagnosis of amyloid liver is probable only when the organ has reached a considerable size, at which time death is likely to occur within the course of a few months. It is possible that incipient amyloid degeneration of the liver may remain stationary.

Treatment.—When it can be done, the suppurative or other disease which may cause amyloid degeneration of the liver should be removed. There is no known remedy which has any direct or indirect influence upon the degenerative process itself.

CANCER OF THE LIVER.

This term will be employed to designate the malignant tumors of the liver in contradistinction to the benignant tumors of this organ. The latter are the localized hypertrophy, myxoma, fibroma, lipoma, glioma, angioma, benignant adenoma, and retention cysts. The former include sarcoma, malignant adenoma, and cancer, to be differentiated only by histological examination.

Etiology.—Cancer of the liver has been found in about three per cent. of a large number of cases of death from various causes. It is more frequent in women than in men, usually occurring late in middle life, but may be found in the earliest years. Heredity, traumatism, and gall-stones are generally considered of etiological importance. The more common occurrence in women has been in part explained by the greater frequency of gall-stones in this sex.

MORBID ANATOMY.—Primary and secondary cancer of the liver are to be discriminated, the latter being the variety more frequently encountered. Primary cancer presumably arises from the liver-cells and from the smaller bile-ducts. Secondary cancer commonly owes its origin to primary cancer of the alimentary canal, especially of the stomach, rectum, colon, or œsophagus. Cancer of the gall-bladder or of the common duct may be primary, or the hepatic cancer may be secondary to that of the pancreas, uterus, ovaries, or mammary gland, but more commonly it arises in those organs in which the radicles of the portal vein begin.

The growth of the cancer in the liver follows the distribution of Glisson's capsule, or is uniformly infiltrated, or is present in the form of nodules. These are single or many, even more than one hundred, and may vary in size from that of a pin's head upward. The liver, especially the right lobe, may attain an enormous size, and the organ has weighed as much as twenty-five pounds. As a rule, the more excessive the enlargement the greater the loss of liver-cells. The enlargement is likely to be symmetrical when Glisson's capsule is conspicuously involved or the infiltrating variety is present, and may be exceedingly irregular when nodular cancer is the variety concerned. If nodules project above the surface they are rounded or flattened, and often umbilicated from central fatty degeneration and absorption. The larger masses are frequently surrounded by smaller growths in close proximity and tending to become fused with the former. The section of the liver varies in appearance according to the method of invasion of the cancer, the extent it has reached, the degenerations it has undergone, and the disturbances it has produced in the blood-vessels and bile-ducts. The infiltrating forms of cancer, whether in Glisson's capsule or spreading diffusely, may follow closely the structural details of the normal liver, and the appearances may resemble those of fibrous hepatitis. The nodules are usually rounded, more gray or white when small, more red or yellow when large, but are sometimes of a translucent gray (hyaline or colloid) appearance. The red color is due to injection or hemorrhage, the yellow color to fat or necrosis. The dark gray or black color indicates a melanotic cancer or sarcoma. The consistency of the nodules is soft (medullary or encephaloid) or hard (fibrous or scirrhous). Finally, cavities containing a thin, yellow fluid may be present within the nodule as a result of softening.

The blood-vessels of the liver may be obstructed or obliterated by the compression of the cancerous growths, which may also perforate their walls and grow along their interior. Obstruction results to the flow of blood through the corresponding parts of the liver, which become engorged with blood, and perhaps necrotic. Compression of the bile-ducts by the nodules produces dilatation and jaundice, either limited to portions of the liver or affecting the various tissues of the body, according to the seat of the obstruction.

Extension of the disease to the lymph-glands in the portal fissure, with possible obliteration of the portal vein, obstruction of the common bile-duct, or perforation of the gall-bladder, may occur. The peritoneum, especially of the omentum and Douglas's fossa, is likely to be invaded, and the disease may extend to the spleen, pancreas, and kidneys, and to the lungs.

SYMPTOMS.—The disturbances associated with cancer of the liver, as a rule, are complicated with those due to cancer of the organ in which the disease is primary. The symptoms attributable to the affection of the liver are usually dependent upon the extent of the disease in this organ, and, for practical reasons, no distinction is drawn between the results of primary and those of secondary disease of the liver. Loss of appetite, nausea, vomiting, and constipation are usually present, but do not indicate necessarily that the liver is diseased. Emaciation and debility eventually occur, sometimes with great rapidity. Disease of the liver is more directly suggested by pain and jaundice. The former, varying in degree, is almost constant, and may be limited to the right hypochondrium, or radiate in all directions from this region. Jaundice is present in at least half the cases, varies in degree, and is sometimes extreme. The abdomen becomes distended with fluid in about as many cases. The fluid is either due to obstruction to the flow of blood through the portal vein or is the result of cancerous peritonitis. Cutaneous œdema is likely to appear towards the end of life. Hemorrhages in the skin, or from the nose, stomach, and bowels, at times take place, and itching and hiccough may be troublesome. The pulse becomes weak, and is slow when jaundice is present. The temperature is usually normal, but is likely to be febrile if the disease runs a rapid course associated with suppuration, and the fever may be continuous or intermittent. A leukocytosis even to the extent of forty thousand leukocytes in the cubic millimetre may be present. The urine is diminished in quantity, high-colored, frequently with a trace of albumin, and bile-pigment may be present even when there is no discoloration of the skin. Indican is increased, and melanin or melanogen may be present in melanotic cancer or sarcoma. The presence of melanin is indicated by a dark, almost black, color of the urine when passed, whereas melanogen is made evident by the urine becoming black after a time or when heated with nitric acid. The fæces may or may not be clay-colored, even when jaundice is present. The liver is usually, sometimes visibly, enlarged, and the area of hepatic dulness may extend from the third rib to the crest of the ilium. The edge of the liver is generally readily felt, and cancerous nodules, if present, may be palpated and sometimes seen through the abdominal wall. At times they are so soft in consistency as to suggest the presence of fluid. The enlarged liver descends with the diaphragm, and the left lobe may so transmit the aortic impulse as to simulate aneurism. The spleen, as a rule, is not enlarged.

Diagnosis.—Cancer of the liver is to be diagnosticated by progressive loss of flesh and strength, pain and tenderness in the right hypochondrium, and enlargement, especially nodular, of the liver. The presence of leukocytosis, jaundice, ascites, and peritoneal nodules, the last apparent perhaps only on rectal examination, offers additional favorable evidence. The enlarged nodular liver produced by an echinococcus is not accompanied with the cachexia of cancer, and exploratory puncture of the nodule reveals a clear fluid, but not blood. Other nodular enlargements of the liver from hyperplasia or fibrous hepatitis lack the cachexia of cancer, and are of slower growth. If the liver is symmetrically enlarged, hypertrophic cirrhosis might be excluded by the failing splenic enlargement and the long period of jaundice without emaciation and debility. Amyloid enlargement of the liver is slower in progress, jaundice is infrequent, albuminuria and dropsy are more constant and severe, and the etiology may be apparent. If the liver is not enlarged, and the symptoms are suggestive of cancer, it may be impossible to discriminate between cancer of neighboring organs and that of the liver, especially if the former are adherent to the liver.

Prognosis.—Cancer of the liver is universally fatal, usually within a year after its recognition. The disease may progress so rapidly, with loss of flesh and strength, that death takes place in the course of a few weeks, perhaps from cancerous peritonitis, or suddenly from intraperitoneal hemorrhage from the rupture of a vascular nodule. If the duration of the disease extends over a longer period, it is likely to terminate fatally by progressive weakness, with eventual pulmonary œdema.

Treatment.—The only treatment of cancer of the liver is palliative.

DISEASES OF THE GALL-BLADDER AND BILE-DUCTS.

Under this term are to be considered the pathological processes involving the gall-bladder and bile-ducts, which, although they are often simultaneously affected, may be independently diseased.

JAUNDICE. ICTERUS.

Definition.—A pathological yellow discoloration of the skin and many of the tissues and fluids of the body, usually, if not invariably, due to bile-pigment, and occurring in many diseases and under a variety of conditions.

Etiology.—Mainly through the researches of Stadelmann it is now generally admitted that all cases of jaundice are due to the obstruction of the outflow of bile from the liver and the absorption of its pigment through the lymphatics of the liver, for there is no absorption of bile when the common bile-duct and the thoracic duct are tied. The assumption of a hæmatogenous jaundice in which the pigment is supposed to be set free in the blood without the mediation of the liver lacks support, since it is shown that the agents which set free the hæmoglobin also produce obstruction of the small intra-hepatic bile-ducts, thus favoring the absorption of bile, and the presence of bile-acids in the urine in such cases shows that absorption of bile has taken place. The idea of a jaundice from the suppression of the secretion of bile is effectually disposed of, especially by the experiments of Stein, which show that jaundice does not occur in birds and vertebrates from whom the liver has been removed.

Obstruction to the outflow of bile being the immediate cause of jaundice, it is convenient to consider that such obstruction may affect the common and hepatic bile-ducts or the intra-hepatic bile-ducts. Obstruction of the former may be produced by external causes, as constriction from scars or compression from tumors, whether neoplastic, aneurismal, parasitic (echinococcus), or fæcal. The internal causes producing obstruction are inflammation, stricture, tumors, or foreign bodies.

The external causes of obstruction of the intra-hepatic ducts are the various inflammations of the liver and the tumors and parasites of this organ. Passive congestion and fatty infiltration may be added. The internal causes are catarrhal and suppurative inflammation, calculi, concretions, and inspissated bile.

MORBID ANATOMY.—The skin may be of a pale yellow or deep yellowish-brown color, suggesting that of bronze, and shades of green and blue may be present. The darker colors represent a more prolonged and more complete obstruction. The variation in color is likewise dependent upon the quantity of normal pigment, the thickness of the epidermis, and the quantity and quality of the blood in the part stained. The teeth, cartilage, nerve-tissue, tears, saliva, and mucus are not discolored. The other tissues and fluids of the body, especially the urine, sweat, and milk, contain bile-pigment. The fibrinous sputa of acute pneumonia and acute inflammatory exudations are pigmented. The liver and kidneys may present a dark olive-green color, and their cells be diffusely stained or contain pigmented granules or scales. The latter may be present in the canal of the tubules. If the obstruction lies at the duodenal end of the common duct, where it is most frequently found, as a thickened mucous membrane or a plug of mucus due to catarrh, the bile-ducts are distended with dark viscid bile, and, if the obstruction is of a more permanent character, as a tumor or stricture, the ducts are dilated as well. If the larger bile-ducts are unobstructed, a microscopic examination of the liver may show elongated and branching plugs of inspissated bile within capillary bile-ducts. When the larger bile-ducts are inflamed, their walls are likely to be thickened and ulcerated and their contents a thin, gruel-like fluid.

SYMPTOMS.—The disturbances from obstruction to the passage of bile into the duodenum depend upon the absorption of bile-pigment and bile-acids and the absence of bile from the intestinal contents. Within three or four days after the obstruction has taken place—and the earlier the more sudden and complete the obstruction—the skin and visible mucous membranes become yellow. This color is usually first observed in the conjunctivæ, which are strongly contrasted with the white sclerotic coat.

Discoloration of the urine may be noticed even earlier than that of the skin, and as it increases in intensity assumes a dark brown resembling that of porter. The urine readily foams when shaken, and the froth has a yellow color. The quantity, reaction, and specific gravity are normal, and there is neither albumin nor sugar. On microscopical examination hyaline casts are often found, and the detached epithelium of the urinary tract is stained yellow. The urine may be similarly discolored after the use of rhubarb, senna, and santonin; but then it does not foam when shaken, and the color becomes red when caustic potash is added. The detection of bilirubin in the urine offers positive evidence of the presence of bile-pigment. This is usually accomplished by the use of Gmelin's test, which may lack a positive result in stale urine or in that of a patient with fever. An equal quantity of ordinary nitric acid is allowed to flow down the side of a wineglass containing a few drachms of urine, or a few drops of separated urine and acid are caused to unite in a thin layer on a white dish. Rosenbach filters the urine, and applies a drop of the acid to the surface of the stained filter-paper. If bilirubin is present it is oxidized when it comes in contact with the acid, with a resulting play of colors from green through blue, violet, and red to yellow, in which the presence of green is characteristic. A very convenient and easily applied test is that of Maréchal as modified by Rosin. Tincture of iodine is diluted with ten parts of alcohol. A thin layer of this diluted tincture is allowed to flow along the side of the wineglass upon the surface of the contained urine, which may be diluted if deeply pigmented. The presence of bilirubin is shown by the formation of a green ring at the line of apposition of the two fluids. The search for bile-acids is rendered unnecessary as a means of recognizing the especial variety of jaundice, since it is probable that all jaundice is of obstructive origin, and it has been found that bile-acids may be present in the urine of healthy persons. They may be rapidly decomposed in the blood, perhaps occurring only in traces in the urine. The search for them is laborious and without practical value. They are usually found by Pettenkofer's test as modified by Strassburger.

The effect of the bile-acids is especially manifested on the nervous system. Headache and dizziness are frequent. The patient is often irritable, despondent, dull and stupid, or restless and wakeful. Vision may become less distinct as darkness approaches, *hemeralopia*, or it may then become stronger, *nyctalopia*. *Xanthopsia*, the yellow appearance of objects, is of rare occurrence, and hence is not to be attributed to bile-pigment.

The absence of bile from the intestines interferes with the absorption of fat, favors putrefaction, and causes the fæces to become clay-colored. The unabsorbed fat crystallizes into acicular clusters which are to be found in the fæces, and are chemically, according to Oesterlen, a magnesian soap. The increased putrefaction of the intestinal contents is indicated by flatulence and the extremely offensive odor of the escaping gas. The formation of the latter may become so abundant as to cause tympany, colic, and constipation. The stools become clay-colored, since they are deprived of urobilin, their normal coloring matter, which arises from the transformation in the intestine of bilirubin. It is to be remembered that the fæces may be pale from a milk diet when there is no obstruction to the entrance of bile, and dark-colored from the presence of iron or bismuth even when bile is absent.

A bitter or disagreeable taste in the mouth, loss of appetite, nausea, a faint, "all gone" feeling before and a sensation of epigastric pressure after eating, are often present, and usually precede the occurrence of the jaundice. The tongue may be coated, the breath fetid, the area of hepatic dulness increased, the gall-bladder distended, and both liver and gall-bladder tender and painful.

Itching of the skin, one of the most distressing symptoms of jaundice, as a rule, eventually occurs, and is due either to bile-pigment in the skin or to the absorption of bile-acids. It is most severe in the palms and soles and between the fingers and toes, but may be found everywhere, and is especially disagreeable at night and when the patient is in bed. In consequence of scratching, papules, pustules, ulcers, and crusts are formed. Boils and carbuncles may occur, and urticaria, herpes, and xanthelasma may appear. Cutaneous as well as internal hemorrhages are frequent. A slow pulse is the rule, due, according to Legg, to the effect of bile-acids on the cardiac ganglia. The beat may be twenty per minute, but usually ranges between forty and sixty. Respiration and temperature are normal in the absence of complicating inflammations. The occurrence of fever in cases of jaundice is characterized by a lower temperature and slower pulse than are present in fever without jaundice.

DIAGNOSIS.—The discoloration of the skin and urine and the presence of bile-pigment in the latter establish the diagnosis of jaundice. The mere discoloration of the skin is insufficient for this purpose, since it may be simulated in the colored races and in Addison's disease, and if the jaundiced patient is first seen with artificial light the yellow color may be invisible. In Addison's disease the sclerotic is white, the pigment is most abundant in the head, hands, and flexures of the body, and the discoloration has existed for a long time.

In determining the seat of the obstruction the examination of the fæces and gall-bladder is of especial importance. The paler the fæces, and the more acute and intense the jaundice, the more likely are the extra-hepatic ducts to be obstructed. If the obstruction is near the duodenum, the fæces are colorless and the gall-bladder may be distended. If the hepatic duct is obstructed, although the fæces are colorless, the gall-bladder is not distended. The concurrence of jaundice and bile-stained fæces indicates an incomplete obstruction of the extra-hepatic bile-ducts, or that the cause of the obstruction lies within the liver. Persistence of the jaundice, with the absence of pain and acute symptoms, would favor the latter seat of the obstruction.

The duration of the jaundice is of importance in the diagnosis of the cause. Acute jaundice lasts several weeks; chronic jaundice extends over several months or years. Acute jaundice, if without complication, is probably catarrhal, but if associated with prolonged fever the catarrh has probably affected the smaller bile-ducts. If accompanied by attacks of pain in the region of the gall-bladder or by biliary colic, the jaundice is probably due to gall-stones. Chronic jaundice without conspicuous pain is likely to be due to fibrous hepatitis, to cancer of the liver, or to chronic passive congestion of this organ; ascites with enlarged spleen or symmetrical enlargement of the liver suggests the first, enlargement of the liver with deformity the second, and evident mitral stenosis the third of these conditions.

PROGNOSIS.—Acute catarrhal jaundice usually terminates favorably within the course of six weeks, yet fatal acute yellow atrophy of the liver may be preceded by a fortnight of apparently simple catarrhal jaundice. Acute jaundice from gall-stones, as a rule, rapidly subsides with the cessation of the attack of biliary colic. The prognosis of acute febrile jaundice is uncertain during the persistence of the fever, in consequence of the gravity of the complications which may arise. Chronic jaundice, especially when increasing in intensity, is of serious if not of grave importance, particularly if without fever and pain; then persistence with cachexia is suggestive of malignant disease of the liver; if the persistence is without cachexia, hypertrophic cirrhosis is probable.

TREATMENT.—As jaundice is only a symptom, its radical treatment is that of the disease which produces it. The various treatments of these diseases will be found under their respective headings; that of catarrhal jaundice is given in the article on inflammation of the gall-ducts. (See page 938.) When in any case it is not possible to determine the cause of a jaundice, a moderate careful treatment in accordance with the principles enunciated in the articles on catarrhal jaundice and on chronic hepatic congestion should be carried out.

INFLAMMATION OF THE GALL-DUCTS, CHOLANGITIS, AND OF THE GALL-BLADDER, CHOLECYSTITIS.

ETIOLOGY.—Inflammation of the bile-ducts and gall-bladder usually results from the continuous extension of a catarrh of the duodenum along the common duct into the hepatic and cystic ducts and into the gall-bladder. Duodenal catarrh is generally continued from the stomach, where it is most frequently caused by the presence of some irritant, as food, drink, medicine, or poison. The persistence and severity of the catarrh of the bile-ducts and gall-bladder largely depend upon the presence in them of some mechanical or microbial irritant, as gall-stones, verminous parasites, or bacteria. Inflammation of the biliary tract may also occur in the course of infectious diseases, as pneumonia, typhoid fever, malaria, and erysipelas, and the reported epidemics of simple catarrhal jaundice are presumably due to the action of some unknown infectious agent upon the gall-ducts. It is a frequent accompaniment of passive congestion, acute and chronic inflammation, and cancer, of the liver.

MORBID ANATOMY.—The inflammation is most frequently limited to the intestinal end of the common bile-duct (*choledochitis*), but it may be continued along this duct to the hepatic ducts and into the liver, or through the cystic duct into the gall-bladder. On the contrary, the inflammation may be limited to the cystic duct or to the gall-bladder, or may occur simultaneously in both. The anatomical varieties of inflammation of especial importance are the catarrhal and the suppurative. *Catarrhal choledochitis,* the most frequent cause of acute jaundice, is manifested by swelling and injection of the mucous membrane of the intestinal part of the duct, the outlet of which is filled with an opaque, viscid material containing abundant epithelial cells. Nearer the liver the mucous membrane of the duct may show little or no alteration, or appearances may be found similar to those just mentioned. Catarrhal inflammation of the cystic duct and of the gall-bladder is characterized by similar appearances. If the cystic duct becomes obstructed, dilatation of the gall-bladder results, and the latter tends to assume a pear shape. sometimes containing a quart or more of pale, watery, or slimy fluid. The mucous membrane becomes thin, smooth, and shining. This condition is called *dropsy of the gall-bladder.* If the obstruction persists, the gall-bladder shrinks very greatly, and its walls are thickened and calcified.

In *suppurative inflammation* of the biliary tract the mucous membrane is thickened, injected, and contains hemorrhages. In severe cases it may be ulcerated or necrotic and patches of fibrinous exudation may be present. Pus or muco-pus, but little stained with bile, is found in the dilated ducts, even in the intra-hepatic branches. When the dilated gall-bladder is filled with a purulent fluid the term *empyema of the gall-bladder* is applied. The extension of a suppurative cholangitis to the intra-hepatic branches of the bile-ducts tends to produce multiple, disseminated, small abscesses of the liver. If these coalesce, larger abscesses are formed, and, when superficial, more or less extensive localized peritonitis pursuing the course of a suppurative perihepatitis occurs over them. Suppurative inflammation of the gall-bladder and extra-hepatic ducts may extend to surrounding parts, frequently producing a localized peritonitis, and sometimes causing inflammation of the portal vein. Perforation may occur, resulting in the establishment of fistulous communications between the biliary and the alimentary, respiratory, or urinary tract, or the abscess may be evacuated through the abdominal wall.

The severer forms of inflammation may result in the production of cicatricial tissue, the contraction of which at times produces narrowing and distortion of the biliary tract and obstruction of the portal vein.

SYMPTOMS.—Inflammation of the bile-ducts, perhaps extended to the gall-bladder, is first made evident by the occurrence of jaundice, usually slight at the outset, but rapidly increasing in intensity. This symptom is the well-known *catarrhal jaundice.* For several days a period of malaise or some of the symptoms of gastro-duodenal catarrh, as failing appetite, nausea or vomiting, belching, epigastric discomfort, and irregular stools, are likely to precede the occurrence of the jaundice. The temperature is but little elevated. The liver is slightly enlarged and somewhat sensitive, and the patient presents the general and local disturbances considered in detail in the section on jaundice.

Catarrhal cholangitis may extend into the minutest gall-ducts of the liver and become persistent. The extra-hepatic ducts then show no alteration. The important symptom is continuous jaundice, and the course of this affection is that of hypertrophic cirrhosis.

When catarrhal inflammation of the gall-bladder exists independently of catarrh of the common bile-duct, it is usually associated with obstruction of the cystic duct; jaundice is not likely to occur; pain and tenderness in the region of the gall-bladder are present. The pain may be sudden and severe, associated with vomiting, and in the course of twenty-four hours the progress of the disease has so simulated acute intestinal obstruction as to lead to a laparotomy. Physical examination in the sensitive region discloses the presence of a tender, more or less rounded and elongated, elastic tumor, dull on percussion, intimately connected with the liver, and changing position with the movements of the diaphragm. Exceptionally, as mentioned by Eichhorst, an overlying coil of intestine may lie between the liver and the distended gall-bladder.

Suppurative cholangitis and cholecystitis are usually preceded by the milder symptoms of catarrhal inflammation. The change in its character is indicated by chills and fever. The former, usually irregular, are sometimes suggestive of malaria. The range of temperature is like that described in connection with abscess of the liver, and may persist for months. The disease becomes a septicæmia, and its course is modified as the resulting abscesses remain confined or find an outlet through a hollow organ or the skin or into the peritoneal cavity.

DIAGNOSIS.—The determination of a catarrhal jaundice establishes the diagnosis of catarrh of the common bile-duct. The extension of the inflammation towards the liver is rendered probable by increasing severity of the symptoms, especially by a higher range of temperature. Continuance of the jaundice after the disappearance of the acute disturbances and the relief of the obstruction at the duodenal end of the duct suggests that the intra-hepatic bile-ducts are affected, and the diagnosis eventually to be made is likely to be that of hypertrophic cirrhosis. Catarrhal cholecystitis alone, if persistent, forms an abdominal tumor which may become so large as to be confounded with a pelvic tumor. The history of the case, the seat of the early pain, and the bimanual examination of the pelvic contents will suffice to exclude a pelvic tumor. A movable kidney is more deeply seated, is less tender, is more readily displaced, and can be pushed into place. The enlarged gall-bladder may be mistaken for cancer of the liver. The latter is to be excluded by the frequent deformity of the liver and the usual prolonged cachexia and frequent jaundice. An echinococcus cyst in the region of the gall-bladder is rather spherical than pear-shaped, although the aspirator may be needed to discriminate between the two. Suppurative cholangitis or cholecystitis is to be diagnosticated when the chills and higher range of temperature, the general symptoms of septicæmia, and the local indications of peritonitis or abscess of the liver appear.

PROGNOSIS.—Catarrhal choledochitis, simple catarrhal jaundice, usually causes but little disturbance, and runs its course in a few weeks, jaundice being the last symptom to disappear. The longer the persistence of the latter, the more likely the occurrence of permanent and irremediable changes in the liver resulting in hypertrophic cirrhosis.

The immediate attack of catarrhal cholecystitis is likely to end favorably. The disease tends to recur, however. If cystic dropsy of the gall-bladder occurs, an abdominal tumor is formed which usually proves harmless. Suppurative inflammation of the biliary tract is always a serious affection, from the tendency it has to cause peritonitis, abscess of the liver, and pylephlebitis.

TREATMENT.—In catarrh of the bile-ducts and the catarrhal jaundice to which it gives rise it is rarely necessary or advisable to put the patient to bed, though of course violent exercise must be avoided. In many cases there is a coincident gastric or duodenal catarrh. Moreover, the digestive powers are distinctly impaired by the absence of bile. The diet should therefore always be light, though nutritious. Further, sweets, fatty matters, and starchy foods, which are more or less contra-indicated by gastro-intestinal catarrh, are especially to be forbidden in catarrhal jaundice, because the products of their fermentation (especially liable to occur in the intestines when bile is not present) are irritant to the liver.

As an excess of nitrogenous food is also very capable of doing injury in catarrhal jaundice, and as the digestive power is often so weakened that spinach and other green vegetables cannot be readily assimilated, it is evident that great care is necessary in feeding the patient. In many cases a diet of skimmed milk, or, if the patient prefer it, of buttermilk, koumiss, matzoon, or some similar preparation of milk, may be rigidly enforced. When meats are allowed they should be very light in character, and the quantity as well as the quality of food should be carefully restricted.

In the beginning of an attack of biliary catarrh an attempt should be made by the free use of calomel, followed by salines, to remove the existing hepatic congestion; this in many cases may be followed or accompanied by the administration of silver nitrate, for the purpose of affecting the gastric and duodenal mucous membrane. Both mercurials and alkalies are especially indicated, from the fact that they not only have a tendency to increase the biliary secretion and to make it more fluid, but also are antiphlogistics, with a pronounced tendency to render less adhesive the mucous secretions from the biliary ducts. An ounce of potassium citrate may be given daily for a short time, or thirty grains of sodium bicarbonate with twenty grains of potassium bicarbonate may be exhibited every two hours in dilute solution. Mild counter-irritation is sometimes of service, but should be preferably made by nitrohydrochloric acid. (See page 914.)

Suppurative inflammation of the gall-ducts is not amenable to treatment: it usually gives rise to multiple abscesses of the liver of pyæmic nature. The best that can be done is to meet symptoms as they arise, and to evacuate any local collections of pus that may present themselves.

CHOLELITHIASIS.

DEFINITION.—The disturbances produced by gall-stones.

ETIOLOGY.—Gall-stones occur in about seven per cent. of all autopsies, and are usually found in elderly adults, but sometimes in early childhood. Although present after death, according to Schröder, in twenty-five per cent. of all persons above sixty years of age, the disturbances resulting from them are more frequent between the ages of thirty and fifty. The gall-stones are formed within the hepatic ducts and in the gall-bladder, and are chiefly composed of cholesterin, bile-pigment, and lime, which are generally considered to be precipitated from the bile. Naunyn maintains that the cholesterin is formed from the fattily degenerated epithelium of the biliary tract, and that stagnation of the bile favors the formation of the calculus. Catarrh of the bile-ducts is also of probable importance as favoring the degeneration of epithelium and precipitation from the bile. Of late the frequent association of bacteria with gall-stones has suggested that the former may be of etiological importance. Gall-stones are found three or four times as often in women as in men, and especially in women who have borne children or from whom abdominal tumors have been removed. In more than half of a considerable number of cases of gall-stones in women there was likewise a corset liver, and movable kidney and liver, also of etiological importance, are more frequent in females. Nutmeg liver, adhesions of the gall-bladder, and fibrous degeneration of its wall may aid in the formation of gall-stones by preventing the passage of bile. Persons of sedentary habits, and rather the fat than the lean, are more prone to gall-stones. According to Musser, gall-stones are present in sixty-nine per cent. of cases of primary cancer of the gall-bladder, an affection likely to be accompanied by catarrhal inflammation and biliary stagnation. Foreign bodies, clotted blood, seeds, parasites, bacteria, mercury, and a needle have been found in the centre of a gall-stone.

MORBID ANATOMY.—Single or many gall-stones are to be found in the biliary tract, and when they are exceedingly minute the gall-bladder may contain enormous numbers. They vary in size from that of a pin's head upward, and may become as large as a hen's egg. They are round, ovoid, or elongated, and when multiple are likely to be faceted. The facets are usually regarded as the result of pressure, but may be present when the calculi do not fill the gall-bladder. According to Orth, the facets result from the deposition of the constituents of the gall-stone on those surfaces which do not lie in contact. The apposed surfaces of elongated calculi may be hollowed and rounded like the ends of bones in joints. Gall-stones vary in color from the white or yellowish-white of cholesterin to the dark chocolate color of pigment-lime. The calculus on section may appear homogeneous or have a deeply pigmented nucleus. Some calculi are formed almost entirely of cholesterin, which is dissolved by alcoholized ether, on the evaporation of which crystals of cholesterin appear. *Pigment-lime*, as a rule bilirubin and lime, is usually present in greater or less quantity, and is sometimes the chief constituent of the calculus. The addition of a dilute solution of potash to washed fragments of the calculus produces a deep yellow color, forming prismatic rings when treated with impure nitric acid. Usually both cholesterin and pigment-lime are present. Traces of iron and copper at times may be found. The cholesterin calculi are firm, white, crystalline, cutting like wax; pigment-lime calculi are dark in color, homogeneous, and brittle.

Although gall-stones are most frequently found in the gall-bladder, they are to be seen at any part of the biliary tract. They may lie in a dilated canal, which may be tortuous and bent, or be present in one which has a lateral dilatation or diverticulum. The mucous membrane either shows no alterations, or presents the characteristics of mild or severe inflammation. The resulting disturbances appear to be largely dependent upon the association of bacteria, which are always present at the duodenal end of the common duct. Dilatation of the gall-ducts and stagnation of the bile favor the extension of the bacteria towards the liver, and, although normal bile is free from bacteria, the latter may grow in dilute bile. The typhoid and the colon bacillus, Friedländer's bacillus, the staphylococcus, streptococcus, and pneumococcus, have been found in the inflammatory products associated with gall-stones. The inflammation may extend to adjacent parts, as the peritoneum or the connective tissue of the portal fissure, with the production of adhesions and abscesses, and the eventual establishment of an opening between the biliary tract and the stomach, duodenum, transverse colon, pelvis of the right kidney, ureter, abdominal wall, or bronchi, and the gall-stones may escape from the body through such openings. The suppurative inflammation may be continued into the liver, either directly by means of the bile-ducts or indirectly by the production of pylephlebitis, thus producing abscesses, although Dabney has shown that less than ten per cent. of the abscesses of the liver are attributable to gall-stones. When chronic inflammation of the biliary tract results from the presence of gall-stones, scars, fibrous adhesions within and outside of the ducts and gall-bladder, thickenings, indurations, twists, cysts, diverticula, or fibrous hepatitis may result.

SYMPTOMS.—Gall-stones are often present, and, when small, may even pass through the common bile-duct without giving rise to symptoms. Since the calculi, however, vary in size and are often numerous, the symptoms due to their passage may frequently recur within a short time, or months or years of freedom may ensue, to be ended by fresh paroxysms. The disturbances which are due to gall-stones are, first, those resulting from the expulsion, impaction, or incarceration of the stone, and, second, those dependent upon subsequent inflammatory conditions. The patient who suffers from the severe symptoms of gall-stones may have had antecedent digestive disturbances, slight jaundice, or sensitiveness in the region of the liver, the so-called bilious attacks. The symptom which usually first suggests the presence of the gall-stone is biliary colic, which, however, is no absolute sign of an attempt at the expulsion of the gall-stone, since it may be the result of an inflamed gall-bladder, or be recurrent and no gall-stone be present, and there may be no colic when gall-stones large enough to produce intestinal obstruction have escaped into the bowel.

Biliary colic, according to Kraus, oftenest takes place two or three hours after eating, especially at night, and is perhaps excited by unsuitable food or drink. The pain is usually sudden and severe, and rapidly increases in intensity. It is generally referred to a limited region to the right of the median line a short distance below the ensiform cartilage, or to the vicinity of the gall-bladder, and may radiate in various directions; at times it is referred to the right shoulder and arm. It is cutting, stabbing, tearing, or twisting, and is not relieved by pressure. It may suddenly cease, or it may last for hours, days, or weeks, with occasional intervals of freedom. When the pain is especially severe the patient is restless and has an anxious expression, the skin is cold and moist, perhaps cyanotic, and hiccough may be present. Vomiting soon follows, at first of the contents of the stomach, then of bile, if the common duct is not obstructed, and, rarely, of gall-stones. As a rule, after the contents of the stomach have been expelled repeated retching follows.

The attack of colic is in more than one-half of the cases followed in the course of a few hours by chills or rigors and an elevated temperature. The temperature may be as high as 104° or 105° F., and the fever usually quickly subsides with the cessation of the colic, but is of long duration if inflammation of the biliary tract ensues. It may be continuous, remittent, or intermittent, in the latter case resembling malaria, and then to be regarded as a septic fever the result of an infection of the patient. The pulse is often slow, but it may be frequent, weak, and irregular.

Jaundice is present in about one-half of the cases, beginning within a few hours after the attack of pain. Although probably an obstructive jaundice, it does not necessarily indicate that the gall-stone lies in the common duct, since it may be present when the gall-stones are in the gall-bladder. Unless inflammation of the biliary tract ensues or the gall-stones become impacted or incarcerated, jaundice usually disappears in the course of a week or two after the attack of pain. When the common duct is obstructed, either by the calculus or by swelling of the mucous membrane, the bowels are constipated and the stools are colorless and offensive. They may be alternately colored and without color when the duodenal opening of the common duct is only temporarily closed.

The abdomen is not distended. The painful region is usually sensitive, and palpation is resisted by the tense rectus muscle. The liver is likely to be somewhat enlarged, and the anterior edge may be found on a level with the navel. The swelling of the liver, like the jaundice, soon subsides with the disappearance of the colic, but may persist and increase with the evidence of inflammation of the biliary tract. The region of the gall-bladder may be tender, and this organ is palpable in one-third of the cases, but it may be contracted and inappreciable. Crepitation of the distended gall-bladder has been observed when the gall-bladder contained many small calculi, and a friction-sound has been heard over its inflamed surface. Persistent pain and tenderness in the region of the gall-bladder after disappearance of the colic are suggestive of the occurrence of inflammation.

The spleen may be found enlarged, though its swelling is slight and inconstant unless prolonged inflammation of the biliary tract occurs.

The attempt at the expulsion of the stone may be successful, with a speedy disappearance of all the symptoms and the elimination of the calculus. The latter, as a rule, is possible only in the case of stones not larger than peas. The attempt may be unsuccessful, the calculus remaining, although the symptoms disappear. The retained gall-stones are either incarcerated or impacted. If incarcerated in the gall-bladder they may produce no further disturbance, or renewed attempts at expulsion take place, or inflammation of the gall-bladder, perhaps infectious, occurs, or the gall-bladder becomes contracted, forming a capsule, perhaps calcified, around the gall-stone. If incarcerated in the common duct, renewed efforts at expulsion are frequent, resembling those already described, ceasing only with the passage of the stone or with the death of the patient, which may be directly attributable to the presence of the calculus, as from abscess of the liver, chronic hepatitis, or peritonitis.

If the gall-stone is impacted in the cystic duct, the immediate result is a painful distention of the gall-bladder, due to retention of secretion, unless a complicating infection gives rise to the presence of pus. If the stone is impacted in the common duct, the pain eventually ceases or becomes slight, jaundice persists and increases, the bile-ducts continue to dilate, and the gall-stone is either expelled or is encapsulated in a circumscribed lateral dilatation, a diverticulum. The passage of bile then normally takes place, the jaundice disappears, and the patient recovers health and strength. If the impacted calculus is neither expelled nor incarcerated, jaundice persists, and in the course of a year death results from cholæmia, ending in delirium, convulsions, and coma. The principal danger from the impacted or incarcerated calculus is that it favors the occurrence of an infectious inflammation of the biliary tract, especially when impaction is in the common duct. Netter has shown that ligature of the duodenal end of the common bile-duct results in infectious inflammation of the liver, while ligature of the duct near the liver causes no infection of this organ. It is possible that the fever following the painful efforts at expulsion of the gall-stone may represent a mild infection. The repeated chills, and the intermittent type or prolonged continuance of the fever, offer positive evidence of the existence of the infection. It is further characterized by hepatic and splenic enlargement, perhaps by evidence of a meningitis, an endocarditis, or a nephritis. The various infectious inflammations of the biliary tract and their possibilities have already been described, and it may be added that abscesses may be found in remote parts of the body as well as in the biliary tract in consequence of the existing infection.

DIAGNOSIS.—The presence of gall-stones is considered probable when the sudden severe attack of localized pain is followed by vomiting, perhaps by jaundice, and is associated with enlargement of the liver. The severe pain and vomiting of acute gastritis are directly attributable to improper food, and the pain from ulcer of the stomach immediately follows eating. The early pain from pancreatic hemorrhage and acute pancreatitis at times closely resembles biliary colic, especially as there may be associated jaundice, but in the pancreatic affections there is greater collapse, and the epigastric tenderness is more severe and extends to the left. The pain is also more easily relieved, and in the course of a few days tender spots from fat-necrosis may appear in the abdomen. It may be impossible to exclude a pancreatic calculus, the passage of which has produced symptoms not differing from those resulting from the passage of gall-stones. The sudden severe pain may suggest peritonitis from perforation, but in the latter the abdomen rapidly becomes rigid and tender and is swollen and tympanitic. The pain of appendicitis may also be sudden and severe, but is usually seated in the right iliac fossa, and tenderness exists in the region of the appendix. Renal colic may simulate biliary colic, but in the former the pain extends along the course of the ureter, is perhaps referred to the penis or the testicle, and blood, sand, or gravel may be found in the urine. The gastric crises of locomotor ataxia may simulate biliary colic, but are not attended with persistent jaundice or fever, and are especially to be recognized by the presence of other symptoms of locomotor ataxia. Lead colic is to be excluded by the history of the case, the absence of jaundice or hepatic tenderness or enlargement, the obstinate constipation, the retraction of the belly, and the peculiar tenseness of the pulse. Small concretions may pass with but little pain, yet jaundice follow, and the attack be considered as one of catarrhal jaundice. In the latter affection the symptoms at the outset are less sudden, and the jaundice persists for a longer time. The probable diagnosis of gall-stones is made certain when the calculus is obtained. The fæces, if necessary, for several days after the pain has ceased should be freely diluted with water and so thoroughly stirred as to contain no fragment larger than a pea. The liquefied fæces should be then poured through a fine sieve, when the concretion may be obtained. A calculus may have passed through the common duct with characteristic symptoms and yet not be discovered by this examination, since Naunyn has shown that gall-stones may be decomposed in the intestine.

Repeated typical attacks of biliary colic may occur and no calculus be found, even when the biliary tract is directly explored after a laparotomy. The possibility of recurrent attacks of biliary colic even with jaundice, especially in nervous persons, perhaps alternating with neuralgic attacks elsewhere, and not due to gall-stones, should be recognized, particularly when the question of surgical treatment is entertained. The diagnosis of gall-stone, the failure to find the calculus, and the persistence of the biliary colic suggest that the gall-stone lies in the gall-bladder, the cystic duct, or the common bile-duct. Its seat in the gall-bladder is to be inferred from the persistence of the fever and the localized tenderness, especially if jaundice is associated. A number of calculi may be present and the gall-bladder be not distended. If the calculus lie in the cystic duct, a distended gall-bladder and slight or absent jaundice give important evidence of its seat. Increasing jaundice, persisting attacks of pain, conspicuous enlargement of the liver, and a gall-bladder of normal size are in favor of the presence of calculi in the common duct. The significance of the calculus in the production of the acute and chronic inflammations of the bile-passages of the liver is indicated by the history of early attacks of biliary colic. In like manner antecedent attacks of biliary colic may lead to the recognition of the gall-stone as the cause of an acute obstruction.

PROGNOSIS.—The immediate attack of biliary colic is rarely fatal unless serious disease exists elsewhere, as in the heart or the brain, when fatal collapse or cerebral hemorrhage may occur. Rupture or perforation of the biliary tract has also taken place, with a fatal peritonitis. As a rule, repeated attacks of colic occur at various intervals without especial danger to life unless complicated with the more severe results of an infectious inflammation. The prognosis is then somewhat dependent upon the search for a calculus. If a faceted calculus is found or the search for a stone proves negative, other attacks may be expected. A small, round calculus not larger than a pea is often the only calculus present. Persistent jaundice of calculous origin may be relieved after it has existed for months, even for three years, as reported by Osler, and the patient recover. More frequently death from cholæmia in the course of a year, the production of a hypertrophic cirrhosis proving fatal in the course of years, or the possible development of cancer of the biliary tract, is to be feared. That gall-stones may serve as a cause of cancer of the gall-bladder is suggested not only by their frequent concurrence, but also, as stated by Gumprecht, by the facts that attacks of biliary colic may for a long time precede the symptoms attributable to cancer, and that cancer has been found to develop from ulcers and scars of the gall-bladder. The latter are frequent results of gall-stones.

TREATMENT.—During an attack of hepatic colic the pain is so severe as to demand immediate relief, which is best afforded by a hypodermic injection of morphine (one-eighth to one-third of a grain) with atropine (one two-hundredth to one-eightieth of a grain). Opium should not be given by the mouth, or, except in rare cases, by the rectum, absorption being too slow and uncertain. The hypodermic injection may be repeated at intervals of half an hour, care being taken that the whole amount given shall not be distinctly toxic, lest the relief from pain consequent upon expulsion of the stone should allow a sudden narcosis. The opium should be aided by the general hot bath and by the continuous application of moist heat to the hepatic region. In rare cases the local use of cold is preferred by the patient and should be practised. When the pain becomes unendurable, ether, or even chloroform, may be used to mild anæsthesia, which by lessening spasm favors the passage of the stone. When the paroxysms are frequent and severe, saline purgatives are often of service.

The objects of the continuous treatment of a case of gall-stones are—to prevent, as far as may be, inflammation of the gall-ducts and congestion of the liver; to aid, if possible, in the solution of the gall-stones; and especially to prevent the formation of new concretions. Outside of the body gall-stones can readily be dissolved by certain medicinal substances; but it is doubtful whether these solvents when given by the mouth have any effect. The continued use of them by the profession, and the fact that especially during the so-called Carlsbad cure gall-stones are frequently passed in considerable numbers without pain, makes, however, their trial imperative.

It must further be remembered that bile itself has the power of dissolving some of the substances of which hepatic calculi are made, so that the solution of gall-stones would seem to be aided by remedies which increase the amount and fluidity of the bile. The method practised by Bartholow of injecting these solvents directly into the gall-bladder has not found favor, and we have never used it, because it has seemed to us dangerous. Probably the most famous of the direct solvents is the Durande remedy, which consists of three parts of ether and two of turpentine. Some practitioners prefer chloroform or ether by itself. These drugs should always be given in capsules and upon an empty stomach, so that they may reach the liver in as concentrated form as possible. The dose should be from ten to twenty minims four to six times a day, according as the stomach will bear. Olive oil, six to eight ounces a day, has been much used, and occasionally seems to do good; being a harmless remedy, it is certainly worth a trial in individual cases. As bile is the natural solvent of its solid constituents, probably no one of the artificial solvents is as effective as is the restoration or even exaggeration of the function of the liver.

To aid in the mechanical expulsion of the gall-stone digital manipulation has been commended by some authorities, but its capabilities for harm are far greater than those for good. In cases with great distention of the gall-bladder aspiration has been practised; it usually produces no evil effect, but is liable to be followed by an escape of bile and consequent severe or even fatal peritonitis. Very rarely, if ever, can any good be accomplished by it.

As the formation of gall-stones is largely the result of improper functional activity or of loss of functional activity in the liver, and as this hepatic torpor is usually due to congestion of the liver or to catarrhal inflammation of the gall-ducts, it is evident that such treatment of cholelithiasis as is directed to the prevention of the formation of concretions is practically the same as that of chronic hepatic congestion, and is closely allied to that of biliary catarrh. (See articles on these subjects.)

Formerly surgical procedures were justified only when the gall-stone was believed to be hopelessly impacted. The results of recent operations have, however, been so uniformly good in uncomplicated cases that at present the physician is not justified in waiting for more than three or four serious attacks of bilious colic, after he is satisfied that a gall-stone has lodged in the common duct. Care, of course, must be exercised not to mistake attacks of biliary colic produced by the successive formation of gall-stones for evidence of the persistent existence of a single stone. Too long waiting not only endangers the structure of the liver and the health of the patient, but makes the operation itself much more difficult and less apt to be a success. Destruction of the gall-bladder, apparent obliteration of the gall-duct by overlying exudation, and other anatomical changes, may render it impossible for the surgeon to locate the diseased part. The operations which are performed are *cholecystotomy*, or incision of the gall-bladder, *choledochotomy*, or incision into the common bile-duct, and *cholecystenterostomy*, or union of the intestine with the gall-bladder. Of these operations the first is the simplest and the most frequently successful, since the cystic duct is often dilated and the stone may be removed without opening the common duct, which is deep and difficult to close with sutures so as to avoid leakage; the second appears to be the best when the stone is in the common duct and cannot be removed through the gall-bladder, although some surgeons prefer cholecystenterostomy with the use of Murphy's button. According to the statistics of Murphy, up to April, 1895, in thirty-eight cases of cholecystenterostomy there had been but one death. The union should be made either between the duodenum and the duct or between the colon and the duct, as experience has shown that great digestive disturbance follows the union with the lower portions of the small intestine. The union between the colon and the gall-bladder is much the more easily brought about, but it seems physiologically probable that the absence of bile from the small intestine must work evil to digestion. The chief objection to the operation seems to be the danger of the final closure of the artificial fistula between the gall-duct and the intestine: choledochotomy is free from such objection, is comparatively easy of performance, and answers in the great majority of cases. If during the operation the patient's condition becomes such as to make imperative the immediate finishing of the operation, no hesitation should be felt in leaving connection between the cut duct and the air with a drainage-tube. Almost invariably the biliary fistula thus connected finally closes, if all stones have been removed. In any case of operation a careful, not too violent, attempt should be made to crush the stone with the fingers before opening the duct (cholelithotripsy): if success attends the effort the gall-bladder should not be opened: if during the subsequent passage of fragments there is much pain, hypodermic injections of morphine should be given.

TUMORS OF THE BILIARY TRACT.

Tumors of the biliary tract may be situated in the bile-passages within or without the liver. The former have already been referred to in connection with tumors of the liver; the latter deserve independent consideration. Such tumors are either benignant or malignant. The former are the rare fibroma and myxoma of the gall-bladder, and are of no practical importance; the latter is cancer, both primary and secondary, relatively rare in the extra-hepatic gall-ducts, although not infrequent in the gall-bladder. Musser has recently collected one hundred cases of primary cancer of the gall-bladder and eighteen of primary cancer of the gall-ducts.

CANCER OF THE GALL-DUCT.

Primary cancer of the biliary tract usually occurs late in life, in women four times as often as in men, and is generally associated with gall-stones, although it is a question which is the earlier in development. Cancer of the bile-duct usually arises at the duodenal end of the common duct. Sometimes it may be found near the junction of the cystic and the hepatic duct.

Morbid Anatomy.—The alterations produced by cancer of the gall-duct are often so slight that the growth may be easily overlooked, especially since the secondary growths elsewhere, particularly in the liver, are frequently extensive. It may represent merely a circumscribed, either nodular or oblong, thickening of the mucous membrane and submucous tissue near the duodenal papilla. It tends rather towards stenosis than towards ulceration, and its cancerous nature is often questionable until a microscopic examination has been made.

Symptoms.—As a result of the stenosis, complete obstruction occurs to the outflow of bile, and persistent and intense jaundice results. Bacterial invasion of the bile-ducts may take place, and a continued or an interrupted fever follow. More frequently death occurs in the course of three or four months, from cholæmia with profound disturbance of the nervous system. It is probably in consequence of such early death that ulceration of the neoplasm and extension to the liver or elsewhere, with emaciation and debility, are often absent.

Diagnosis.—The condition is one of rapidly progressing, intense, chronic jaundice, the physical examination giving no evidence of the cause as in hypertrophic cirrhosis. The negative character of the evidence, and the fact, as stated by Naunyn, that about one-half of the cases of chronic jaundice are due to cancer of the biliary tract, are the factors of avail in making the diagnosis.

Treatment.—The only medical treatment of cancer of the biliary tract is palliative. Laxatives and narcotics should be used as necessary. Life may sometimes be prolonged and relief obtained by a cholecystenterostomy.

CANCER OF THE GALL-BLADDER.

Morbid Anatomy.—Starting as a circumscribed thickening projecting from the inner wall of the gall-bladder, the disease progresses both in depth and in circumference until the entire gall-bladder may become infiltrated. This viscus is often increased or diminished in size, the former perhaps due to accumulation of fluid as well as to the growth of the cancer. The wall is frequently nodulated as well as thickened, the inner surface extensively ulcerated, perhaps shreddy or papillary, the cavity containing more or less fluid, usually opaque gray or yellow, not necessarily bile-stained, in which may be shreds of tissue. In the large majority of cases one or more gall-stones are present. As a rule, the liver is also cancerous, and the alterations of this organ may be so extreme, especially when the changes in the gall-bladder are slight, that the origin of the disease in the latter organ is overlooked. Extension to the liver may take place directly from the gall-bladder or indirectly by means of the blood-vessels or lymphatics. Adhesions and eventually fistulous communications may be formed between the gall-bladder and the colon or the duodenum.

SYMPTOMS.—As might be anticipated, the symptoms of cancer of the gall-bladder in the majority of cases are identical with those due to gall-stones, since the latter are present in the majority of cases of cancer of the gall-bladder. The attacks of biliary colic or more or less persistent localized discomfort and febrile disturbances occur in the one as in the other. Jaundice is less frequent, except late in the disease, when gall-stones lie in the gall-bladder. If the latter is enlarged, the duodenum may be compressed, and vomiting, perhaps of blood, take place. As the disease progresses, loss of flesh and strength becomes conspicuous, and ascites, perhaps dropsy, may occur, death usually taking place within a few months after the malignant nature of the disease is apparent. If the gall-bladder is enlarged, its outlines may be appreciated on physical examination, and nodules may be felt if they are present. On the contrary, the cancerous gall-bladder often is shrivelled and inappreciable to the touch. Nodules are likely to be found elsewhere, as in the liver or the peritoneum. Aspiration of the enlarged gall-bladder may reveal the thickened wall, and permit the escape of an abundant albuminous fluid in which blood, bile, or granular detritus may be present.

DIAGNOSIS.—The uncertainty of the diagnosis is evident from the fact that unsuspected cancer of the gall-bladder has frequently first been recognized after the abdomen has been opened by the surgeon or at the autopsy. Its presence may be inferred when, in the course of more or less severe biliary colic, with or without jaundice, a rapidly progressing cachexia occurs. If complicated with fluid in the peritoneal cavity there is but little enlargement of the spleen, since the liquid is due probably to cancerous peritonitis. Primary cancer of the gall-bladder may be considered probable when cancer of the liver is to be recognized and there is no evidence of antecedent cancer in other parts of the body.

TREATMENT.—The medical treatment of cancer of the bladder is purely palliative; if the diagnosis be reached sufficiently early, surgical excision is justifiable.

CHAPTER V.

DISEASES OF THE PANCREAS AND OF THE PERITONEUM.

DISEASES OF THE PANCREAS.

HEMORRHAGE.

ETIOLOGY.—Hemorrhage into the pancreas not infrequently occurs to a limited extent under a variety of conditions. Among these are passive congestion of its veins, acute infectious diseases, and the hemorrhagic diatheses. Extensive hemorrhage, sometimes designated pancreatic apoplexy, is of occasional occurrence, and is at times the result of direct injury ; more often there is no obvious cause. It is found in adults usually after the middle period of life, rather in the fat than in the lean, and has often been observed in persons of alcoholic habits.

MORBID ANATOMY.—The hemorrhage commonly takes place into distinct portions of the gland, and the infiltrated regions are of various size. The affected part is swollen, firm, and of a dark-purple color, though sometimes it is of normal size and of soft consistency. The hemorrhage frequently extends beyond the limits of the gland, and the blood then may be found in the fat-tissue around the pancreas, within the omentum and mesentery, and occasionally in the fat-tissue over the kidney. Evidences of bleeding at some earlier period are at times to be found as reddish-yellow spots due to the presence of crystals and granules of hæmatoidin.

SYMPTOMS.—Sudden abdominal pain, usually severe, though sometimes trivial, and immediate collapse, are the only constant symptoms. The pain is at times referred to the epigastrium, but often is not sharply localized. The diagnosis is generally made after death.

PROGNOSIS.—Severe forms of pancreatic hemorrhage usually prove fatal within twenty-four hours. That recovery may occur is indicated by the discovery of blood-pigment in the pancreas when death has resulted from disease of other organs, and is demonstrated by recovery from a laparotomy at which this lesion has been seen. Usually, if the patient survives the immediate effects of the hemorrhage, inflammation of the pancreas, with its several possibilities, occurs.

TREATMENT.—The treatment of pancreatic hemorrhage consists in the meeting of symptoms of collapse at the time of bleeding and in the administration of opiates as required for the relief of pain. Morphine, from one-eighth to one-fourth of a grain, atropine, from the two-hundredth to the one-hundred-and-fiftieth of a grain, strychnine, from one-thirtieth to one-twentieth of a grain, and digitalis, from five to ten minims, are to be used hypodermically. Alcoholic stimulants and small doses of nitroglycerin are to be given by the mouth.

ACUTE PANCREATITIS.

ETIOLOGY.—Since acute pancreatitis at times represents the result of pancreatic hemorrhage, its etiology is in part that of the hemorrhage, and a traumatic cause has repeatedly been observed. Next in importance is gastro-duodenal catarrh, especially when recurrent. Acute pancreatitis oftenest occurs in males above the age of fifty years, and in fat persons, especially in those who use alcohol freely.

MORBID ANATOMY.—Three anatomical varieties of acute inflammation of the pancreas are to be found,—namely, the hemorrhagic, the gangrenous, and the suppurative ; although the transition between the symptoms and lesions of the hemorrhagic and gangrenous varieties is so gradual as to make it probable that these are rather stages of a single process than independent affections.

In *hemorrhagic pancreatitis* the gland is enlarged, generally throughout, but sometimes at one end, and the head, when affected, may be half the size of the fist. Larger or smaller extravasations of blood of a dark-red, almost black, color are present, and the pancreatic duct may contain a thick, bloody fluid. As in rapidly fatal cases of pancreatic hemorrhage without inflammation, bleeding is frequent into the fat-tissue in the vicinity of the pancreas, especially in the mesentery, mesocolon, and omentum, and in the region of the left kidney.

Gangrenous pancreatitis at times is represented by a circumscribed necrosis of the gland, which elsewhere presents the appearances of hemorrhagic pancreatitis. When the necrosis is more extensive the entire gland may be transformed into a dark-gray, spongy mass loosely attached to the abdominal wall. The adjacent peritoneum, especially that of the omental bursa, and the serous coat of the neighboring coils of intestine, are covered with a fibrinous exudation which in places forms adhesions between the apposed surfaces. The cavity of the omental bursa usually contains a sero-purulent exudation in greater or less quantity, *omental bursitis*, but evidences of a general peritonitis are rarely found.

Multiple large and small opaque white spots are often to be found within the pancreas and in the subperitoneal fat-tissue, especially in the vicinity of the gland, and occasionally in other portions of the abdomen. These spots are characteristic of a necrosis of the fat-tissue, *disseminated fat-necrosis*, and often lie immediately beneath the peritoneum, the surface of which is then coated with fibrin. The necrotic lobules of omental and mesenteric fat and of that in the region of the kidney and colon are surrounded by a zone of purulent infiltration which leads to the detachment of the necrotic portions, perhaps accompanied by hemorrhage. If the fat-necrosis is in the vicinity of the omental bursa, the masses of fat-tissue are often discharged into this sac and swim in the liquid exudation therein contained. A fistulous communication may be established between the cavity of the omental bursa and the interior of the stomach or the duodenum. The importance of inflammation of the pancreas, especially of hemorrhagic pancreatitis, in the production of the fat-necrosis is based not only upon their usual association, but also upon the experiments of Langerhans, Whitney, and Hildebrand, who have succeeded in producing experimentally multiple disseminated necrosis of the fat-tissue either by injection of the minced pancreas into the subcutaneous tissue or by direct injury to the pancreas and its ducts and blood-vessels.

In *suppurative pancreatitis* also the gland is enlarged, but contains single or many abscesses of various size. When these reach the surface the inflammation is extended through the parapancreatic tissue to the neighboring peritoneum, at times resulting in a large abscess of the lesser omental cavity, which may be discharged into the stomach or the duodenum.

Thrombosis of the splenic vein is not infrequent, and the thrombus may be in a state of puriform softening. Abscesses of the liver are at times to be found, especially in suppurative pancreatitis. Pleurisy and pericarditis are occasional results of the extension through the diaphragm of the inflammation from the peritoneum.

SYMPTOMS.—In the hemorrhagic and gangrenous varieties of pancreatitis the initial disturbances are commonly sudden and severe. Abdominal pain is usually the first symptom, and is either persistent or paroxysmal; it is generally referred to the epigastrium, and is sometimes localized in the region of the pancreas. It is quickly followed by vomiting, which may persist even after the contents of the stomach have been thoroughly removed, in which case the vomitus is slimy, dark green, or black, and may contain liquid or clotted blood. Chilly sensations often are present, and the patient may be in a state of prostration or collapse for several hours. As a rule, fever soon supervenes, and the temperature, although at first only slightly elevated, eventually may be as high as 104° F. Hiccough, slight jaundice, and albuminuria have been observed, and mild delirium, especially in the later stages of the disease, not infrequently occurs.

In suppurative pancreatitis the initial symptoms are usually neither so acute nor so severe, and there may be little or no pain. The fever pursues a varying course, and chills not infrequently occur at irregular intervals, followed by sudden changes of several degrees in the temperature.

During the further progress of acute pancreatitis, whether hemorrhagic, gangrenous, or suppurative, the upper half of the abdomen becomes swollen, tense, and tympanitic. The region of the pancreas and sometimes that of the spleen are tender on palpation, and a deep-seated resistance may be found near the head of the pancreas, as observed by Elliot. The occurrence of a complicating fat-necrosis may be announced by the appearance of painful and tender spots in various parts of the abdomen. The distention of the abdomen continues to increase either locally, perhaps only in the left half, or throughout; in the latter case the entire abdomen at times is extremely swollen, and is tympanitic except in the flanks. The abdominal pain becomes general, vomiting persists, diarrhœa is frequent, and there is rapid and progressive emaciation. During the third or fourth week perforation of the circumscribed peritoneal abscess, the omental bursitis, not infrequently takes place. It is made evident by severe lancinating pains, by copious dejections in which the sloughing pancreas has been found, and by a rapid diminution in the size of the abdomen.

The course of suppurative pancreatitis may be continued over a period of months, in which case ascites and anasarca have been observed. The skin has become bronzed, and sugar has been found in the urine.

DIAGNOSIS.—Sudden abdominal pain, especially when referred to the upper abdomen, followed immediately by vomiting and great prostration and later by moderate fever and circumscribed resistance in the epigastrium, perhaps in the vicinity of the head of the pancreas, especially when occurring in a well-nourished person beyond middle life, should suggest acute pancreatitis. Confirmatory evidence is offered by the presence of disseminated points of abdominal tenderness, which are suggestive of incipient fat-necrosis. The differential diagnosis lies between the action of an irritant poison, peritonitis from perforation of the stomach or the duodenum, a calculus in the common bile-duct, and acute intestinal obstruction. The history of the case and the examination of the vomitus permit the exclusion of an irritant poison. Perforation of an ulcer of the stomach is usually preceded by repeated attacks of characteristic pain and by the presence of blood in the vomitus or in the dejections. Biliary colic from the passage of a gall-stone is not at once accompanied by symptoms of collapse, the relief from pain and distress is often immediate and complete, and jaundice, if present, is usually early and considerable.

The affection oftenest mistaken for acute pancreatitis is acute intestinal obstruction. Its onset is less severe, however, and the epigastrium is rarely the seat of the primary localization of the pain and distention. In the later stage of acute pancreatitis the tumor due to the frequently resulting omental bursitis may be mistaken for cyst of the pancreas. The latter is to be excluded by the existence of fever and by the results of the examination of the aspirated fluid.

PROGNOSIS.—Acute pancreatitis is a disease of extreme gravity, death usually taking place from shock in the course of a few days after the onset of the hemorrhagic variety. When gangrenous pancreatitis is concerned, the patient is likely to die in the course of four to eight weeks from septicæmia. In suppurative pancreatitis the patient may die in the course of a few months from septicæmia, or may live for a year and then die from progressive exhaustion or from diabetes. The prognosis is not absolutely hopeless, however, since evidences of antecedent acute pancreatitis have been found repeatedly at post-mortem examinations. In addition, Osler, Körte, and Thayer have reported cases in which the presence of hemorrhagic pancreatitis was assured by laparotomy and recovery followed. The possibility of recovery from gangrenous pancreatitis, even at a late stage of the disease, is placed beyond doubt by the observation of Trafoyer. His patient lived seventeen years after the pancreas was discharged from the bowels.

TREATMENT.—The treatment of pancreatitis is practically that of a localized peritonitis. If in the beginning there are hemorrhage and collapse, stimulants must be used with an activity proportionate to the degree of collapse. Morphine hypodermically is often required for the relief of pain. Surgical treatment is demanded when gangrene or suppuration is believed to be imminent, free drainage being necessary, and the operation is the more likely to be successful if the peritonitis is confined, as is usually the case, to the lesser omental cavity.

CHRONIC PANCREATITIS.

ETIOLOGY.—Chronic inflammation of the pancreas may be the result of a long-continued suppurative pancreatitis. More commonly, however, it is due probably to a chronic inflammation of the pancreatic duct resulting from persistent or recurrent gastro-duodenal catarrh, especially in persons of alcoholic habits. Conditions which give rise to obstruction of the pancreatic duct, as calculi or tumors, are also a source of chronic pancreatitis. This affection may be limited to a portion of the pancreas in connection with ulcer of the stomach or of the duodenum, with caries of the spine, and with neighboring tumors or aneurism. Chronic pancreatitis has been found in infants, in whom it is regarded as the result of congenital syphilis.

MORBID ANATOMY.—The pancreas is usually atrophied either throughout or in limited portions, though rarely it is enlarged, and then has been mistaken for cancer. The consistency of the diseased part is increased, and may resemble that of cartilage. The surface of the gland is smooth or irregular, and the appearance of the section varies according to the amount of fibrous tissue present and the associated changes. Reddish-gray or grayish-white bands of fibrous tissue traverse the cut surface and enclose or replace the more or less atrophied lobules. Fatty degeneration of the gland-cells causes a yellow mottled appearance, and white specks may be seen, due to the deposition of calcium salts and crystals of fat acids. The duct of Wirsung is either unaltered in appearance or is tortuous, dilated, and often sacculated. The fibrous tissue in the vicinity of the pancreas is frequently thickened and indurated.

SYMPTOMS.—In chronic pancreatitis the symptoms of a chronic catarrhal gastritis are present, lasting indefinitely, and frequently accompanied with diarrhœa. Attention is especially to be directed to the pancreas as a source of the symptoms when there is deep-seated epigastric pain, either mild or severe, and particularly when it occurs in paroxysms associated with restlessness, anxiety, and faintness. Tenderness and perhaps resistance on pressure in the region of the pancreas have been observed. Jaundice occasionally exists, and is sometimes persistent from constriction of the common bile-duct by the head of the pancreas. The stools are at times colorless, even in the absence of jaundice, and may contain fat. There is usually progressive loss of flesh and strength.

Glycosuria has repeatedly been found in chronic pancreatitis, and Lancereaux maintained that there was a diabetes due to serious disease of the pancreas. Williamson, in a collection of one hundred cases of pancreatic lesions in diabetes, found that there was more or less atrophy in thirty-nine, and extensive fibrous thickening in thirteen, while in the rest of the cases a variety of lesions was present. It is demonstrated by the experiments of Von Mering and Minkowski and De Dominicis that total extirpation of the pancreas is always followed by diabetes, while if one-eighteenth to one-twelfth of the gland is left the glycosuria is moderate, and if more than one-tenth remains there is no glycosuria. It is, therefore, probable that the occurrence of glycosuria in chronic fibrous pancreatitis depends upon the degree of destruction of the pancreas, and that the presence of glycosuria in this variety of pancreatitis indicates an extreme degree of destruction of this gland.

PROGNOSIS.—As there can be no restoration of the destroyed portions of the pancreas, the prognosis of chronic pancreatitis is serious: at the same time it is to be remembered that extensive atrophy of the pancreas may take place, and even a large part of the gland be discharged from the bowel, and yet the patient remain in fair health. The occurrence of permanent glycosuria with symptoms of chronic pancreatitis makes the prognosis hopeless.

TREATMENT.—The chief feature in the treatment of chronic pancreatitis is the regulation of diet. All food which requires pancreatic juice for its digestion should be reduced to a minimum: hence the necessity of restriction in the quantity of fats and starches. The use of carbonated waters is to be advised, since Becher has found that they increase the pancreatic secretion and its digestive power in dogs. Minced pancreas also should be tried, as in Abelmann's experiments the administration of pancreatin after extirpation of the pancreas promoted the digestion of fat.

PANCREATIC CALCULI.

ETIOLOGY.—Catarrhal inflammation and retention of secretion in the duct of Wirsung are probably of chief importance in the origin of calculi in the pancreas. The retention of secretion may be due to obstruction of the duct from some external cause, as chronic pancreatitis or tumor of the gland.

MORBID ANATOMY.—Few or many, even more than a hundred, calculi may be present, varying in size according to their number. Solitary calculi may be as large as walnuts, and the smallest concretions are mortar-like. The calculus is generally rounded or oblong, rough or smooth, and may be continued by lateral projections from the wall into the primary branches of the duct. It is of a grayish color, and easily disintegrated. The pancreatic duct and its branches are usually dilated, and atrophy and induration of the gland are frequently associated. Ulceration of the wall of the duct may be present, and even fistulous openings into the stomach, duodenum, or peritoneal cavity. Cancer of the pancreas is rarely an accompaniment.

SYMPTOMS.—Calculi often are found unexpectedly in the pancreas at a post-mortem examination, but in many cases there are antecedent disturbances due to a gastro-duodenal catarrh. The first symptom especially suggestive of the presence of a pancreatic calculus is pain, without especial tenderness, due probably to the displacement of the calculus. It manifests itself either as a sharply defined feeling of pressure or discomfort in the epigastrium or as an intense spasmodic pain continued along the left costal cartilages towards the spine and the left shoulder-blade. This pancreatic colic resembles biliary colic, and jaundice is occasionally associated. Indeed, it may be impossible for the patient himself to discriminate between pancreatic and biliary colic, as in the case reported by Minnich of a patient who passed at different times gall-stones and pancreatic calculi. When the calculi are incarcerated, dilatation of the duct of Wirsung and fibrous pancreatitis are the usual results, and in rare instances a cyst of the pancreas is formed. There is progressive loss of flesh and strength, diarrhœa is frequent, the stools often contain fat acids, undigested muscular fibres are abundant, and pancreatic concretions have been found in the dejections. Occasional or permanent glycosuria also may be present.

DIAGNOSIS.—The diagnosis is based upon the persistence or frequent occurrence of more or less severe attacks of deep-seated epigastric pain, radiating to the left and simulating biliary colic, though usually without jaundice. It is confirmed by the discovery of typical concretions in the stools. If the characteristics of pancreatic diabetes—namely, glycosuria, polyuria, polyphagia, polydipsia, and loss of flesh and strength—are preceded by frequent attacks of pancreatic colic, the presence of concretions and of chronic inflammation of the pancreas is rendered probable.

PROGNOSIS.—Pancreatic calculi are sometimes spontaneously evacuated, either from the intestine—perhaps because of the establishment of a fistulous communication between the pancreatic duct and the stomach or intestine—or through the abdominal wall, as seems probable in the case reported by Capparelli. Fatal peritonitis may follow perforation of the pancreas into the peritoneal cavity. Usually, however, the duration of life is unaffected by the calculi, or the prognosis becomes that of chronic pancreatitis or of pancreatic cyst.

Treatment.—Attacks of pancreatic colic are to be treated like biliary colic, by the local application of heat, the subcutaneous injection of morphine, and the inhalation of ether or chloroform. The treatment of the ultimate results of pancreatic calculi is that of chronic pancreatitis or of pancreatic cyst. The possibility of the successful removal by the surgeon of pancreatic calculi before permanent alterations in the pancreas have taken place should always be borne in mind.

CYST OF THE PANCREAS.

This term is intended usually to indicate a cavity formed by the dilatation of the duct of Wirsung, the wall being composed of fibrous tissue containing portions of the glandular structure of the pancreas, and the contents being a fluid presenting the characteristics of the pancreatic secretion.

Critical investigation, however, of many of the reported cases of pancreatic cyst makes it probable that this term has been applied often to collections of fluid near the pancreas but wholly outside of its boundary; and in rare instances multilocular cysts occur in the pancreas which have no apparent connection with the pancreatic duct or its branches.

Etiology.—Pancreatic cyst may be of congenital origin, for Richardson extirpated a probable cyst of the pancreas from an infant fourteen months of age. Usually, however, cysts of the pancreas are found in adults, and with equal frequency in each sex. The common cause is obstruction in the course of the pancreatic duct, either from inflammation of its wall or of the pancreas around the duct, or from the pressure of tumors, the impaction of calculi, or possibly, as in the case reported by Durante, the presence of a lumbricus in the duct. Although an important place in etiology is usually assigned to local injury, it is probable, as suggested by Lloyd, that, as a rule, the cyst which subsequently appears in the region of the pancreas is an omental bursitis,—that is, an encysted peritonitis in the lesser omental cavity. The injury is likely to produce a hemorrhagic pancreatitis, and the extension of the inflammation to the peritoneal covering of the pancreas is an efficient cause of the encysted peritonitis, which may closely simulate in its physical characteristics a cyst of the pancreas. Multiple cysts of the pancreas are usually due to obstruction of the smaller branches of the pancreatic duct, but Salzer and Hartmann have reported cases of cystic tumor of the pancreas which correspond apparently to the cystoma of the ovary, and some of them are of a malignant nature.

Morbid Anatomy.—Cysts of the pancreas are situated behind the stomach, being separated from it by the two layers of peritoneum which form the walls of the omental bursa, and which are sometimes fused. When small they may be found on either side of the median line, but when large they develop more to the left than to the right. The stomach is then pushed upward, although in rare instances it may lie upon or below the tumor. The transverse colon extends across the cyst or is displaced downward. The cysts are single or many, and are sometimes multilocular. They originate in any part of the gland, and vary in size from those scarcely visible to the naked eye to one as large as the pregnant uterus at full term and extending from the ensiform cartilage to the pubic symphysis. The larger cysts may result from the fusion of smaller cavities, and are of a spherical shape, the structure of the pancreas being lost in that of the cyst. At times the gland may resemble a bunch of grapes, from the presence of numerous and closely joined cysts. The inner surface of the wall of the cyst is smooth or trabeculated, and is lined with cylindrical epithelium. Papillary outgrowths at times project into the cavity, and openings in the wall communicating with smaller cysts are frequently to be seen. The duct of Wirsung, in certain cases, is to be followed to the interior of the cyst both from the head and from the tail of the pancreas, but frequently ends blindly when traced from the duodenum to the cyst. The contents of the cyst may be upward of four gallons in quantity. They are of a pale gray color, somewhat turbid, viscid or watery, of alkaline reaction, and of a specific gravity between 1010 and 1024. Microscopical examination shows leukocytes, fattily degenerated epithelium, fat-drops, and crystals of cholesterin and of fat acids. The fluid presents some or all of the physiological characteristics of the pancreatic secretion, but the older the cyst the less likely are the contents to show all of them. Blood is sometimes present.

The larger the cyst the more extensive is the atrophy of pancreatic tissue, but portions of the gland are to be found within or upon the wall. When rupture takes place the contents escape into the stomach, into the colon, or into the general peritoneal cavity or that of the omental bursa. In the latter case a large cystic tumor of this structure may be formed, the contents having the properties of pancreatic juice, and yet a large portion of the pancreas may remain but little altered.

SYMPTOMS.—There may be no antecedent symptoms, the cyst being discovered accidentally during convalescence in childbirth or from typhoid fever. Usually, however, there is epigastric pain, often persistent perhaps for months or years. It is not infrequently interrupted by paroxysms of pain which are without obvious cause or follow errors in diet, and which are sometimes so severe as to be associated with symptoms of collapse. The pain usually starts near the ensiform cartilage and extends downward or to the left, and may be continued into the left shoulder and perhaps into the left half of the face. In addition to the pain there is frequently vomiting, diarrhœa, or constipation, and the patient complains of fulness in the epigastrium, which is perhaps tender. Recurrent attacks of intestinal hemorrhage sometimes occur. The general nutrition may be well maintained, or there may be loss of flesh and strength.

The cyst is usually first noticed in the left half of the epigastrium, but may be discovered in the left lumbar region. Its growth is apt to be slow, and the cyst after remaining at a stand-still for years may suddenly increase to a large size within a few weeks. On palpation the tumor is smooth, rounded, resistant, and has a slight mobility. It frequently transmits the aortic pulsations. It is inelastic except when superficial, and then fluctuation is at times to be recognized. It is dull on percussion where not overlain by stomach or intestine. On auscultation a souffle from compression of the aorta is frequently transmitted.

When the cyst attains a considerable size the dull epigastric pain or sense of pressure is usually constant. The disturbance of digestion becomes persistent, and loss of flesh and strength is progressive. Fat and abundant undigested muscular fibres have been observed in the fæces, and sugar and albumin have been found in the urine.

Eventually mechanical disturbances are caused by the cyst even if the resulting atrophy of the pancreas does not interfere with digestion and assimilation. There are difficulty of breathing from interference with expansion of the lungs, ascites from pressure upon the portal vein, and anasarca of the lower half of the body from pressure upon the inferior vena cava. Intestinal obstruction has resulted from the pressure of the cyst upon the bowels.

DIAGNOSIS.—The presence of a cyst of the pancreas is to be inferred from the discovery in the epigastrium or left hypochondrium of a smooth, rounded tumor, slightly movable, especially in the vertical direction, and separated from the liver and spleen by a resonant area unless the tumor is of very large size. Its position behind the stomach and colon is made evident by inflation of these organs, and its cystic nature is readily determined by aspiration. The pancreatic nature of the cyst is rendered probable if the aspirated fluid emulsifies fat, saccharifies starch, and digests albumin and fibrin. Von Jaksch and Boas, however, maintain that the diastasic and emulsifying properties of the pancreatic juice may be present in other liquids, and that the peptonizing property is frequently absent from the contents of an unquestioned pancreatic cyst. The presence of blood in the aspirated fluid is of no diagnostic value, since it is inconstant. Even when blood is present in a fluid containing one or more of the properties of the pancreatic secretion and drawn from a cyst in the region of the pancreas, it does not follow that there is a pancreatic cyst. In such cases there is frequently the history of a local injury, and it is not unlikely that a laceration of the pancreas occurs, followed by an omental bursitis, and that pancreatic secretion becomes mixed with blood and peritonitic exudation.

The tumor may be confounded with aneurism of the aorta, dropsy of the gall-bladder, cystic tumors of the kidney, hydronephrosis of the left kidney, and large cysts of pelvic origin. Aneurism of the aorta is to be excluded by the absence of expansile pulsation and the disappearance of the transmitted aortic beat when the patient is placed on the hands and knees. The distended gall-bladder is in the right half of the abdomen, is intimately connected with the liver, and changes position with the movements of the diaphragm. Multilocular cystic kidneys are bilateral, and although hydronephrosis may be limited to the left kidney, it then forms an oblong tumor in the lumbar region, and is crossed by the colon rather in the vertical than in the transverse direction. Extremely large pancreatic cysts may be confounded with cysts of the ovary or the broad ligament, but the history of the case will show that the distention of the abdomen proceeds from above downward. Pelvic cysts lie immediately beneath the abdominal wall, and are to be felt on vaginal examination. The fluid from them is often gelatinous and does not present the physiological characteristics of pancreatic juice.

It may be impossible to discriminate absolutely between cysts of the pancreas and encysted fluid in the omental bursa or in the mesentery, such as result from omental bursitis, echinococcus, lymphangioma, or chylangioma. The characteristics of the aspirated fluid may permit the elimination of suppurative peritonitis, echinococcus cyst, and chylangioma, but are insufficient to exclude a serous peritonitis of the omental bursa, especially if blood is present in the exudation.

PROGNOSIS.—A cyst of the pancreas has been known to be present for twenty years and yet cause but slight disturbance. Digestion may be interfered with but little even when large cysts are present. The prognosis becomes grave in case diabetes is present.

TREATMENT.—Pancreatic cysts when producing permanent or serious discomfort are to be treated surgically. The result of the operation is usually favorable unless diabetes exists.

CANCER OF THE PANCREAS.

Although benign tumors are found in the pancreas, they are rare and of no clinical interest. The malignant growths are lymphoma, sarcoma, and cancer, which produce similar symptoms and are conveniently described as cancer.

ETIOLOGY.—In spite of the assertion that cancer of the pancreas occurs in about six per cent. of all cases of cancer, Mirallié has been able to collect but one hundred and thirteen cases of primary cancer of this viscus. Nearly two-thirds of them are in males, usually in the middle third of life, although pancreatic cancer has been found at birth.

MORBID ANATOMY.—The head of the pancreas is the part most frequently diseased. The growth may be circumscribed or be infiltrated throughout the gland. The tumor is usually rounded, at times as large as a child's head, and varies in color and consistency according to the quantity of fibrous tissue, the number of epithelioid cells, the degenerations they have undergone, the vascularity and hemorrhages, and the degree of jaundice present. The unaffected portion of the gland may be normal in appearance, or be atrophied from cystic dilatation of the duct of Wirsung. Pressure upon the common bile-duct is frequent. The disease often extends to the neighboring lymphatic glands, to the liver and spleen, and to the peritoneum. Fibrous adhesions are common between the diseased pancreas and the surrounding organs.

SYMPTOMS.—The existence of cancer of the pancreas may first be made known at the autopsy. As a rule, disturbances of digestion referable to the stomach or the duodenum precede for years the symptoms more directly attributable to the cancer. These are epigastric pain and jaundice. The pain not infrequently occurs in paroxysms, especially at night, and is very severe in at least one-half the cases, and then may be accompanied by symptoms of collapse. The pain radiates in various directions as a cœliac neuralgia, and when extending into the back has repeatedly been mistaken for lumbago. When jaundice is present it may follow an attack of pain resembling biliary colic, but it persists and is associated with enlargement of the liver and gall-bladder.

The characteristic feature of cancer of the pancreas is a tumor in the region of the gland; but this is discovered in less than one-half of the cases. It lies near the median line, above the navel, and is deep-seated. It is but slightly movable, and varies in outline, density, and sensitiveness. The pulsation of the aorta is usually transmitted. The tumor by pressing upon the portal vein may cause ascites, and then may first be discovered after withdrawal of the fluid from the abdominal cavity. Pressure on the inferior vena cava causes anasarca of the lower half of the body. Pressure upon the duodenum may induce dilatation of the stomach or lead to symptoms of intestinal obstruction. Cancer of the tail of the pancreas is a cause of hydronephrosis of the left kidney, from pressure upon the ureter.

The appetite may be unaffected or be even excessive. When there is vomiting, blood, free fat, and fatty acids may be found in the vomitus. Constipation or diarrhœa may occur. Blood is sometimes present in the stools, but liquid or solid fat or fat acids are rarely found in them. Most important as evidence of disturbed pancreatic digestion is the presence of abundant undigested muscular fibres in the dejections when there is no diarrhœa. The urine is sometimes increased in quantity, and may contain albumin; sugar is at times present, and the glycosuria may disappear shortly before death. In the later stages of the disease the general nutrition may be but slightly affected, and death occur suddenly from gastro-intestinal or intra-peritoneal hemorrhage. More commonly there is a progressive, rarely rapid, loss of flesh and strength, and death occurs either gradually, or suddenly from pulmonary embolism.

Diagnosis.—The recognition of cancer of the pancreas depends largely upon the discovery of a tumor in the region of the pancreas, accompanied by symptoms of obstruction of the pancreatic duct and the common bile-duct. The relation of the tumor to the stomach and the colon is to be determined by inflation of these organs. Evidence of obstruction of the common bile-duct is afforded by abundant undigested muscular fibres after a meat diet and when there is no diarrhœa; also by the absence of a dark greenish-brown color of the urine when a drachm of salol is taken in divided doses in the course of twenty-four hours, and by a diminished quantity of indican in the urine. Neither fat in the stools, lipuria, nor glycosuria is of value in the diagnosis of cancer of the pancreas.

Cancer of the pylorus is more freely movable, is associated usually with a dilated stomach, and is generally unaccompanied by jaundice. Cancer of the duodenum is not to be differentiated from cancer of the pancreas, and many of the reported cases of duodenal cancer are probably cases of cancer of the head of the pancreas. In cancer of the transverse colon inflation of the large intestine is difficult, symptoms of intestinal obstruction are present, and there is likely to be abundant indican in the urine. In cancer of the liver there is usually enlargement of this organ, and jaundice and ascites are frequently associated.

Prognosis.—Cancer of the pancreas usually progresses rapidly after the discovery of the tumor, death taking place, as a rule, within a year. Jaundice and ascites often precede death by a few weeks only.

Treatment.—The symptoms of cancer of the pancreas which especially require treatment are pain, to be relieved by morphine, and perhaps ascites, which, if a cause of mechanical discomfort, should be treated by abdominal puncture.

DISEASES OF THE PERITONEUM.

INTRA-PERITONEAL HEMORRHAGE. HÆMATOCELE. HÆMOPERITONEUM.

According as hemorrhage into the peritoneal cavity is free or circumscribed, so the terms hæmoperitoneum and hæmatocele are used. In the former the hemorrhage may reach any part of the peritoneal cavity; in the latter the progress of the bleeding is limited by adhesions.

Etiology.—Hæmoperitoneum is caused by penetrating wounds of the abdominal wall or viscera, whether due to knife, bullet, or surgical instrument; also by laceration of the liver, spleen, kidneys, or intestine. Hemorrhage into the peritoneal cavity may likewise result from ruptured aneurisms of the aorta and its larger abdominal branches, from omental and mesenteric aneurisms, and from the tearing of large and thin-walled blood-vessels in malignant disease of the liver, pancreas, and ovary. Although scurvy, purpura, hæmophilia, phosphorus poisoning, and certain infectious diseases may be productive of intra-peritoneal hemorrhage, the latter is so slight as not to be of practical importance.

Varicose veins of the ovary or of the broad ligament may rupture, either producing at once a hæmatocele or causing a hæmatoma within the broad ligament, whose subsequent tearing produces the hæmatocele. The escape of menstrual blood through the open end of a normal tube, or through the tube cut across in the removal of a pelvic tumor, or from a tube dilated with blood, *hæmosalpinx*, in consequence of obliteration of the fimbriated end, sometimes causes hæmatocele.

Rupture of the new-formed blood-vessels in the membrane resulting from a hemorrhagic pelvic peritonitis is still to be included among the causes of hæmatocele, although its importance has become lessened since the surgeons have shown the far greater etiological importance of ectopic gestation.

The most important as well as the most frequent of the remediable causes of hæmoperitoneum is ectopic pregnancy, which is also the most important cause of hæmatocele. The fœtus may be present in any part of the dilated Fallopian tube, usually in that beyond the uterus. The time at which the hemorrhage is to take place is chiefly dependent upon the situation and the age of the fœtus. The wall of the tube becomes weakened, usually during the first three months of pregnancy, although the immediate rupture of the tube may result from blows, falls, or strains, and may occur at or about a menstrual period or during sexual excitement.

Morbid Anatomy.—In fatal cases of hæmoperitoneum in consequence of the usual cause, the pelvis contains clotted blood which extends upward between the abdominal wall and the lowermost coils of intestine. The hæmatocele forms a rounded tumor which may be larger than a child's head, but more frequently is about the size of an orange. It usually lies behind the uterus and the broad ligament, although sometimes in front, and may extend into the abdominal cavity. The peripheral portions of the tumor are composed of old or fresh blood-clots entangled in fibrous adhesions and adherent to the thickened peritoneum. The centre contains fresh blood, liquid or clotted, with or without adhesions, and perhaps a fœtus or other products of gestation. In both hæmoperitoneum and hæmatocele a dilated and ruptured Fallopian tube is likely to be found.

Symptoms.—The disturbances resulting from intra-peritoneal hemorrhage vary in accordance with the amount of blood extravasated and the length of time during which hemorrhage has taken place: hence they may be those of a sudden and extreme loss of blood, or of protracted and slight, perhaps recurring, hemorrhage, or of the latter condition terminating in the former.

Pain is generally an early symptom, although, as a rule, of but little severity. Its importance consists in attracting attention to the place from which the hemorrhage proceeds, and it is especially significant in the possibly pregnant female, since it is ordinarily sufficiently frequent and severe to lead her to seek for relief. The hemorrhage may be so extreme that the patient in apparent health and without obvious cause becomes rapidly enfeebled and dizzy; her face is pale and her pulse rapid and weak. If the bleeding is more profuse, the patient is restless and anxious, with a hollow and husky voice, a cold and clammy skin, a pulse scarcely to be felt, and a prolonged and infrequent respiration.

Physical examination may give no evidence of the seat or extent of free hemorrhage into the peritoneal cavity. If dulness exists, it is such as lies within normal limits. Palpation or bimanual exploration may meet with no more resistance than that afforded by intestinal coils with liquid contents. If ectopic pregnancy is the cause, and the tube is ruptured, there may be no physical evidence of its pathological condition. When the hemorrhage is limited by adhesions, the symptoms and signs are those of hæmatocele. The patient is likely to have had antecedent pelvic peritonitis, whether mild or severe, and there is a history of more or less pelvic disturbance. If previously pregnant, a considerable interval is likely to have occurred since the birth of the last child. The patient has been exposed to impregnation, but the frequent occurrence of irregular, perhaps profuse, flowing, has opposed the idea of pregnancy. Nausea and fulness of the breasts may be present or absent. If occasional twinges of pain have preceded, they are likely to have been attributed to intestinal disturbance. A more profuse metrorrhagia may have been accompanied by the discharge of a membrane,—the exfoliated, hyperplastic, uterine mucous membrane.

With such antecedents the formation of the hæmatocele is favored, and its presence is rather indicated by pressure upon the neighboring parts than by symptoms of collapse or the occurrence of severe pain. Within twenty-four hours the hæmatocele may attain a size sufficient to produce frequency of micturition, irritability of the rectum, or pain and abnormal nervous sensations in the legs, according as the pressure is upon the bladder, the rectum, or the pelvic plexuses of nerves. Vaginal examination then indicates the presence of a tender, rounded, elastic tumor at one side, behind or in front of the uterus. The uterus is likely to become somewhat enlarged and soft, lying nearer the symphysis or the hollow of the sacrum according to the seat and size of the hæmatocele. Not infrequently the death of the fœtus is the result of the hemorrhage, which then ceases, the extravasated blood being often absorbed. The symptoms which ensue are those of a mild localized pelvic peritonitis, and consist in a slightly elevated range of temperature lasting a few days, hypogastric and vaginal tenderness persisting for some time, and moderate tympanitic distention of the abdomen. If the blood is absorbed, a diffuse induration in the vicinity of the uterus may remain in its place. If the fœtus continues to grow, the hemorrhages become more frequent and severe, the hæmatocele increases in size, and symptoms of progressive anæmia are associated with the febrile disturbance and localized pain.

The tendency is now towards a perforation of the hæmatocele into the rectum or elsewhere in the intestine, into the vagina, or, more rarely, into the bladder. Perforation into the rectum is preceded by frequent mucous discharges and tenesmus, and a soft and especially tender spot may be found on rectal examination of the surface of the tumor. The discharge of the contents of the hæmatocele into the rectum is likely to be followed by an immediate relief to the symptoms, which relief may become permanent with the disappearance of the hæmatocele. On the other hand, the admission of the rectal contents into the hæmatocele is likely to produce gangrene of the wall of the latter, with a fatal peritonitis. If perforation takes place into the intestine elsewhere than in the rectum, a similar fatal peritonitis may result or fistulæ be established between the hæmatocele and the ileum or cæcum and the rectum. Perforation into the vagina is less frequent than into the rectum, and is followed by like possibilities. Perforation into the bladder usually causes cystitis and pyelonephritis.

DIAGNOSIS.—The diagnosis of intra-peritoneal hemorrhage is based upon the occurrence of sudden, more or less severe collapse in an apparently healthy person, associated with pain and tenderness in the lower abdomen or pelvis. Although such symptoms suggest peritonitis from perforation, the rapid progress of the anæmia excludes this diagnosis. The source of the hemorrhage being concealed at the outset, the history of the case becomes important in calling attention to the gastric, intestinal, uterine, or urinary source of the hemorrhage. In severe gastric or intestinal hemorrhage, pain in the region of the stomach or duodenum or symptoms of typhoid fever or evidences of fibrous hepatitis are likely to have preceded, or blood is found in the vomit and stools. Blood in the urine or in the vagina would point towards the source of the hemorrhage in the genito-urinary apparatus. Hemorrhage from tumors of the liver, pancreas, or ovaries is likely to occur in the later stages of these affections and after characteristic symptoms have been manifested. Sudden collapse and pain may be due to the rupture or to the twisting of the pedicle of an ovarian cyst, especially the latter, in which case the symptoms of anæmia are very conspicuous. The physical examination will disclose the presence of a tumor of such size as to have been previously recognized or of such rapid and extreme development as to exclude the idea of hemorrhage alone.

In hæmoperitoneum there is usually no satisfactory physical evidence of diagnostic value in determining the presence of blood. On the contrary, if the hemorrhage is circumscribed, a tumor is to be found and its outlines are to be determined by bimanual examination. The tumor may be simulated by a hæmatoma of the broad ligament, but this is at the side of the uterus and lacks the severe symptoms of pain and collapse, although later these may follow the rupture of the hæmatoma, which produces either hæmoperitoneum or hæmatocele. A retroflexed pregnant uterus, uterine fibromyomata, small ovarian tumors, pregnancy, or retained menses in a rudimentary horn may simulate a hæmatocele. The nature of the tumor may be suspected if the possibility and signs of pregnancy are absent. The history of pregnancy and the catheter or sound in the bladder permit the diagnosis of retroflexion of the pregnant uterus to be made. Although a distended uterine horn may closely resemble the hæmatocele in history, symptoms, and signs, the tumor is usually lateral and the dilated crescentic os is a part of the tumor. A circumscribed perimetritis or a salpingitis may also resemble the hæmatocele in symptoms, seat, and physical characteristics, although the symptoms are less violent and less rapid and anæmia is absent. The use of the aspirator in case of need will show whether the tumor is composed of blood or of inflammatory exudation.

PROGNOSIS.—Small intra-peritoneal hemorrhages are of but little consequence, since blood is readily absorbed by the peritoneum. If the hemorrhages are sufficiently large, death occurs within a day or two, at times within a few hours. Small hæmatoceles are usually readily absorbed. Large hæmatoceles may also be absorbed, although months are necessary for this to take place. The prolonged illness attending absorption becomes grave if perforation and evacuation of the contents of the hæmatocele, with the risks of putrefaction and gangrene, occur. If the patient recovers, fibrous adhesions, obliterated tubes, and dislocated organs are likely to be left and cause dysmenorrhœa, sterility, and chronic invalidism.

TREATMENT.—There is no medical treatment for rapidly progressing hemorrhage into the abdominal cavity. If the hemorrhage is circumscribed, the patient should be made comfortable, the strength supported, and the absorption of the clot favored by rest and proper care. Serious recurring hemorrhages, threatening life, or producing large tumors, or progressing with symptoms of impending perforation or of septicæmia, require surgical treatment. Zweifel has found that the mortality of one hundred and forty-four cases of hæmatocele under medical treatment was sixteen and six-tenths per cent. Of sixty-six cases treated by puncture the mortality was fifteen and one-tenth per cent. Of thirty cases treated by incision ten per cent. died. A still lower mortality follows the treatment of hæmatocele by abdominal section, although the series of cases thus treated includes those of mild as well as of severe hemorrhage, the large majority of the cases needing no surgical treatment. Martin states that of two hundred and sixty-five cases of ectopic gestation treated expectantly thirty-six per cent. recovered and sixty-three per cent. died, while of five hundred and fifteen operated upon seventy-six per cent. recovered and twenty-three per cent. died.

HYDROPERITONEUM. ASCITES. ABDOMINAL DROPSY.

Accumulation of transuded fluid into the peritoneal cavity takes place under several conditions, and is the symptom of various diseases.

ETIOLOGY.—Two varieties of dropsy are usually recognized, the mechanical and the cachectic. The former is the result of obstruction to the venous outflow, causing increased transudation, or of obstruction to the lymph-current, preventing absorption. Cachectic ascites occurs when the peritoneal endothelium is abnormal and interferes both with venous transudation and with lymphatic absorption. The immediate mechanical causes of abdominal dropsy are general or local. Conspicuous among the former is obstruction to the passage of blood through the heart, as in uncompensated valvular disease and cardiac weakness, pulmonary emphysema, and fibrous pneumonia. The most important local cause is obstruction to the passage of blood through the liver, whether from fibrous hepatitis, malignant disease of the liver, thrombosis of the trunk of the portal vein, or pressure upon the latter by pancreatic tumors or enlarged lymphatic glands or its constriction by peritonitic thickenings and adhesions. The pressure of tumors upon the lymphatics and the hepatic vein or upon the inferior vena cava or the root of the mesentery is likewise a local cause of ascites. The immediate cause of cachectic ascites is the disturbed nutrition of the peritoneum, such as occurs in emaciating diseases, amyloid degeneration, nephritis, and chronic malaria.

Chronic fibrous hepatitis or cirrhosis of the liver is the most frequent cause of abdominal dropsy. The enlarged abdomen occurring in peritoneal tuberculosis and cancer is a manifestation rather of peritonitis than of dropsy.

Ascites takes place at all periods of life, and may even interfere with the birth of the child, although it is of more frequent occurrence in the adult.

MORBID ANATOMY.—The peritoneum may show no abnormal appearances, or it is slightly thickened and opaque, especially in patches. The subperitoneal fibrous tissue may likewise be thickened and indurated. Such thickenings are to be regarded rather as complications than as essentials. The ascitic fluid or dropsical effusion varies in quantity up to several gallons. It is commonly of a pale yellow color, with a slightly greenish tint, though it may become dark green from the presence of bile-pigment, or red or reddish brown from that of blood. It is usually clear, but may be opalescent, especially after its removal during life, in consequence of the precipitation of some probably albuminoid constituent. It is either alkaline or neutral, is of a watery or a viscid consistency, and has a specific gravity of 1010 to 1015. Its chemical characteristics are practically those of blood-serum. It contains from two to two and five-tenths per cent. of albumin, and the ready determination of the percentage of this constituent is of considerable importance. The greater the quantity of albumin contained in free fluid in the peritoneal cavity, the more likely is its origin to be inflammatory; the less the percentage of albumin, the more likely is the fluid to be ascitic. The lowest percentages are found in cachectic ascites, the highest percentages in cancerous peritonitis. The quantity of albumin present is approximately determined by the use of the urinometer, and the accompanying table, a modification of that of Runeberg, indicates the relation of the specific gravity to the percentage of albumin in free fluid from the abdominal cavity, and its etiology.

Specific Gravity.	Percentage of Albumin.	Cause.
1008	0.2	Hydræmic ascites.
1009	0.6	
1010	1.0	Portal obstruction.
1011	1.3	
1012	1.7	General venous obstruction.
1013	2.1	
1014	2.5	Peritonitis.
1015	2.8	
1016	3.2	
1017	3.6	Cancerous peritonitis.
1018	4.0	
1019	4.3	
1020	4.7	
1021	5.1	
1022	5.5	
1023	5.8	
1024	6.2	

When ascitic fluid is allowed to stand as above stated it may become faintly opalescent. A delicate clot of fibrin is likely to occur, and there is little or no appreciable sediment. Leukocytes, endothelium (sometimes in flakes), red and white blood-corpuscles, granular or fatty cells, and crystals of cholesterin may be found at the bottom of the vessel containing the fluid. If the ascites is due to general venous obstruction, the red blood-corpuscles are numerous. The term *chylous ascites* is applied when the ascitic fluid resembles milk, the appearance being due to the presence of molecular fat. It is found when the lacteals leak from wounds, ulcers, or rupture, or when filariæ are present in them. Obstruction of the thoracic duct by thrombosis, scars, induration of the mesenteric attachment to the intestine, and thrombosis of the subclavian vein where the thoracic duct enters, also are causes. In the case of the last-mentioned cause the left pleural cavity may likewise contain a milky fluid. If the milky appearance of the fluid is due to large and small fat-drops, the term *adipose ascites* is employed. Such fat results from the fatty degeneration of cells, and their presence in sufficient abundance is indicative of a chronic peritonitis or of a tubercular or cancerous peritonitis.

The resemblance to milk is further suggested by the formation of a creamy layer on the surface when the fluid is allowed to stand. A high specific gravity and large percentage of albumin favor the inflammatory or neoplastic origin of the fluid, and the sediment then may contain the cells of cancer, blood-corpuscles, endothelium, and fat.

SYMPTOMS.—The immediate effect of ascites is the production of pressure upon the abdominal wall and its contents. Such pressure usually takes place gradually, but may be of rapid progress. As the abdomen becomes distended there is a sensation of fulness in it which may at first be attributed to fat, but soon increases to one of weight and eventually of pressure. The patient moves about with difficulty. He may be obliged to remain upright or in the supine position. The displacement of the heart upward is likely to produce palpitation. The expansion of the lungs is prevented, and respiration becomes frequent and difficult. Vomiting and constipation often occur. The urine is scanty, high-colored, concentrated, and usually contains a brick-dust sediment. These physical characteristics, in addition to a trace of albumin and the presence of occasional red blood-corpuscles, are indicative of a chronic passive congestion of the kidneys. On physical examination the swelling of the abdomen corresponds to the duration and degree of the distention, and presents a striking picture if the patient is emaciated. In the dorsal position the abdomen is likely to bulge in the flanks. In the upright position it is prominent towards the front. When the distention is extreme the belly is rounded, and the navel may protrude or be obliterated. The skin is dry, smooth, shining, and scarred as in pregnancy. The veins in the abdominal wall, especially in the groins, the flanks, and over the lower costal cartilages, are distended and prominent, and, if cirrhosis is the cause, may radiate outward from the navel. The results of percussion vary in accordance with the position of the patient and the distention and mobility of the intestines. If the patient is on his back, and the stomach and intestines are distended with gas, the epigastrium is resonant and separated from the lower abdomen by a curved line of dulness concave upward. When the patient turns to the side, dulness is found over the dependent parts of the abdomen, and resonance in that flank which is uppermost.

If the intestines contain no gas, or are adherent to the abdominal wall, or the mesentery is so short, or the quantity of fluid so large, that they are prevented from floating to the surface, the dulness is no longer characteristic ; but the resonance of the intestines may be found in the lower abdomen or in the flanks, in either of which places it may remain even if the patient changes his position. Fluctuation is usually present, being especially apparent near the border-line between dulness and resonance, and the wave is often to be seen when the abdominal walls are moderately tense. If the latter are excessively fat or œdematous, a superficial wave may be transmitted, not due to fluctuation of the ascitic fluid, and should be controlled by the application of pressure between the palpating fingers by means of some solid object. There may be no fluctuation if the tension of the abdominal wall is either extreme or very slight.

Although the transudation of fluid is usually progressive and the ascites persists, there are frequent temporary variations in the quantity of fluid present. A period of relief to the symptoms of pressure may thus arise, to be soon followed by increased discomfort. The free escape of the contents of the stomach and bowels also produces temporary ease. More permanent freedom takes place from the absorption of the fluid in hepatic ascites, which may occur when there is distention of the smaller veins uniting the radicles of the portal vein with those of the peripheral venous system. More rarely the ascitic fluid has been discharged into the rectum or through the navel. Such relief is usually but temporary, since the cause of the ascites is likely to be permanent, and, as a rule, steadily increases in severity.

DIAGNOSIS.—The discovery that the enlargement of the abdomen is due to the presence of free fluid is essential for the diagnosis of ascites. It is also necessary to determine that this fluid is of non-inflammatory origin. The existence of fluid is decided by dulness on percussion and by the presence of fluctuation. That the fluid is free is indicated by a shifting of the dull fluctuating region with a change in the position of the body. Various conditions associated with the presence of more or less fluid have been mistaken for ascites. This is especially true in the case of fluid contained in large cavities, as the unilocular cystoma of the ovary, the parovarian cyst, the fibro-cyst of the uterus, dropsy of the amnion, the distended bladder, and the dilated stomach or intestine. It is also to be recognized that cysts and ascites may coexist. In such a case either the free fluid or the encysted fluid is likely largely to prevail. The sex of the patient will exclude some of these conditions. If the abdominal wall and that of the cyst are not especially tense, the displacement of the intestines is possible when the position of the patient is changed. It may be difficult to determine whether the fluid is free or encysted. Commonly in the dorsal position the upper line of dulness is concave downward in case of ascites and convex upward in case of cyst. On vaginal examination the uterus is more freely movable in ascites than in the case of a pelvic tumor, and the posterior wall of the vagina is pushed forward by the ascitic fluid, which usually fluctuates when the abdominal wall is palpated. The absence of elongation of the uterine canal, as shown by exploration with the sound, will ordinarily eliminate the uterine fibro-cyst. The uterine cavity is also lengthened when dropsy of the amnion is present, and a possibility of pregnancy, followed by recurring bloody or watery discharges from the vagina, may lead to the diagnosis of this condition. Pregnancy has also been mistaken for ascites, but the history of the case and the auscultation of the abdomen will suffice to make the diagnosis clear. The distended bladder is easily excluded by the passage of a catheter. For dilatation of the stomach to be mistaken for ascites the former must be extreme, the lower border of the stomach lying at the symphysis. The history of the case, the increase of the abdominal distention from above downward, and the use of the stomach-tube will serve for the diagnosis of dilated stomach. Litten states that extreme abdominal enlargement, dulness with change of position, and fluctuation were present in dilatation of the ileum in consequence of stricture. If there was no evidence in such a case of a possible cause of ascites, visible peristalsis, borborygmi, and perhaps aspiration, would aid in the diagnosis.

Free fluid in the distended abdomen may be of inflammatory origin, due to chronic peritonitis or to tubercular or cancerous peritonitis as well as to ascites. In the last there is no elevation of temperature. In the former pain and fever are more likely to occur. In tubercular or cancerous peritonitis tubercles or cancer may be found elsewhere, and the examination of the omentum, mesentery, and parietal peritoneum, including that of the pelvis, may disclose thickenings, perhaps nodular masses. The diagnosis of ascites is favored by the existence of symptoms of obstructive disease in the liver, heart or lungs, and kidneys.

The diagnosis is aided by the examination of the aspirated fluid. The characteristics of ascitic fluid have been already described. Typical ovarian fluid is yellow, somewhat opaque, viscid, of a specific gravity of 1018 to 1055, and contains a larger quantity of albumin. There is no formation of a clot on exposure to the air, and the sediment contains cylindrical or globular epithelium, often in a condition of hyaline or fatty degeneration. The fluid from the parovarian cyst is pale, watery, of a specific gravity of 1005, and has merely a trace of albumin. In the fluid from the uterine fibro-cyst clotted fibrin appears in considerable quantity after exposure to the air. Peritonitic fluid usually has a specific gravity above 1014, contains more than two and five-tenths per cent. of albumin, and may also contain a considerable quantity of fibrinogenous material. In cancerous peritonitis cells resembling endothelium may be found, but their cancerous nature is at times to be recognized, according to Quincke, by the presence of glycogen, which becomes brown on the addition of dilute solutions of iodine. The physical examination of the abdomen after the removal of the greater part of the fluid often makes the diagnosis clear by permitting the recognition of an atrophied liver and an enlarged spleen, or of an abdominal or a pelvic tumor.

PROGNOSIS.—Ascites is always a symptom of serious import, from the usually incurable nature of the disease in which it occurs. The greater the quantity of fluid, the more rapid its accumulation, and the earlier its return after being withdrawn, the worse the prognosis. On the other hand, recovery from dropsy has taken place. Small benign tumors have been the cause of ascites, and the removal of these has effected a cure. Life has been prolonged for years during which repeated removal of the fluid has taken place,—in the case reported by Lecanu more than eight hundred times. The necessity for the removal of the fluid has not rarely proved a new source of danger, since fatal intra-peritoneal hemorrhage has resulted from puncture of arteries in the abdominal wall, and a fatal peritonitis has been caused by the use of unclean trocars.

Treatment.—In ascites, as in other forms of dropsies, diuretics and sudorifics are often useful in getting rid of the dropsical effusion, but, owing to the excessive congestion of the kidneys due to the obstruction of the portal vein, diuretics very commonly fail to act satisfactorily, whilst purgatives are not only certain in their action, but by relieving the excessive congestion of the whole alimentary tract tend to improve digestion, provided they are not given in such form or dose as to cause irritation of the gastro-intestinal mucous membrane. In the selection of the purgative the choice lies between elaterium, compound jalap powder, and salines. Instead, however, of confining the patient to one of these purgatives, it is better to use all in turn. One-eighth grain each of elaterium and extract of belladonna may be given every six hours, or Epsom salt, two drachms, may be given in concentrated solution.

Whenever the collection of fluid in the abdomen interferes with the respiration, and cannot be controlled by purgatives, paracentesis abdominalis should be performed, although it is almost invariably followed by the rapid accumulation of the fluid. Antiseptic precaution should be absolute; the trocar should be disinfected immediately before use. Unless in cases of extreme weakness, the patient should be tapped in a sitting posture, the trocar being thrust into the abdomen half-way between the navel and the pubes and through the linea alba; hard pressure should be exerted upon the whole abdomen by a properly prepared many-tailed bandage applied before the beginning of the operation and continually drawn on during the flowing of the fluid. After the operation the puncture should be covered with a piece of rubber plaster, and over this antiseptic absorbent cotton, whilst the abdomen should be tightly bandaged so as to support the abdominal vessels, which have a tendency to become extremely relaxed upon the withdrawal of the support they have been receiving from the fluid about them. From six to twelve or even more quarts of fluid may be drawn at a single tapping. Trickling of the fluid through the aperture after the operation will usually do no harm if the liquid be absorbed immediately in the dressing and the skin be well protected by the free use of zinc ointment: rarely a circumvoluted suture is necessary. In feeble cases digitalis and strychnine should be given before the operation, to prevent possible syncope.

INFLAMMATION OF THE PERITONEUM. PERITONITIS.

It is especially important for the physician to discriminate between acute and chronic peritonitis, since the appropriate treatment of the former may prevent the chronic variety, and the early recognition of this form may lead to the adoption of measures productive of speedy cure and preventive of prolonged invalidism. Although there is often no sharply defined line of division between acute and chronic peritonitis, the one being continued into the other, there are cases which are chronic from the outset and which require separate consideration, since they are not merely the terminal stage of an acute process.

ACUTE PERITONITIS.

ETIOLOGY.—Peritonitis occurs equally often in either sex and at all times of life, even in the fœtus. A distinction is drawn between primary and secondary varieties of peritonitis according as the inflammation is limited to the peritoneum or is associated with or dependent upon disease of parts covered by the peritoneum. Primary peritonitis is also sometimes called idiopathic or spontaneous. It has been called rheumatic when a primary peritonitis has developed suddenly after exposure to cold, or when, as sometimes occurs, the peritonitis develops during the course of acute articular rheumatism.

In accordance with the prevailing view that inflammations are due to the action of irritants and that such irritants are often of bacterial nature or origin, a distinction is drawn between simple and septic or infectious varieties of peritonitis. Simple peritonitis represents the results of traumatism alone, as from falls, blows, wounds, and aseptic operations, the rupture of an ovarian cyst, or the twisting of the pedicle of a pedunculate, especially pelvic, tumor.

Septic peritonitis results from the association of the above causes, especially traumatism and surgical operations, as well as of those to be later mentioned, with the introduction of bacteria. Such bacteria may vary in kind. Some of them, as shown by experiment, when introduced into the healthy peritoneal cavity produce no disturbances; they are absorbed, their growth is checked, or they are destroyed. If the peritoneum is abnormal in consequence of traumatism or the presence of chemical agents, perhaps derived from the growth of bacteria, or if fæces and blood-clots are present in which the growth of bacteria is favored, or if the absorbing powers of the peritoneum are interfered with by pre-existing disease, as ascites, heart disease, and fibrous hepatitis, septic peritonitis follows the admission of the bacteria. These may directly enter the peritoneal cavity through open Fallopian tubes. They may also pass through the peritoneum from parts communicating with the surfaces of the body, as the gastro-intestinal canal, the biliary tract, the pancreas, the genito-urinary apparatus, the abdominal wall, and the lung by way of the diaphragm. The occurrence of a septic peritonitis without apparent local cause in diphtheria, erysipelas, acute articular rheumatism, pneumonia, and cerebro-spinal meningitis is attributable to the transfer of bacteria through the blood and lymph from remote parts of the body to the peritoneum.

An appreciation of the local causes of peritonitis is of the greatest importance, since the resulting inflammation is usually limited for a while to the place of its origin, although showing an early and sometimes immediate tendency to become general. Such local causes are traumatic or pathological processes in the gastro-intestinal and biliary tracts, the pancreas, spleen, and genito-urinary apparatus. Pathological processes in the blood-vessels and lymphatics of the abdomen and in neighboring parts, as the thoracic organs, the spine, and the pelvic bones, are also to be included. It is probable that the development of a peritonitis in the various infectious diseases is connected with a local cause which eludes recognition,—for example, embolism or hemorrhage.

Exclusive of injuries and operations, the most frequent local causes of peritonitis are to be found in affections of the gastro-intestinal tract, the genitals of the female, and the gall-bladder.

MORBID ANATOMY.—The lesions of acute peritonitis are usually circumscribed at the outset, but tend to become diffused. A distinction is thus drawn between a local and a general peritonitis, the lesions being the same, differing only in extent. At the outset the peritoneum is slightly opaque, its surface dull, its blood-vessels injected, and minute hemorrhages are present in the subperitoneal fibrous tissue. Soft gray or yellow clotted fibrin appears on the surface, forming false membranes varying in thickness and opacity, or adhesions as delicate threads, strings, or bands. A liquid exudation likewise appears, at first thin, watery, and slightly opaque, but later thick, yellow, and purulent. According to the predominance of one or another characteristic of the exudation the peritonitis is spoken of as fibrinous, serous, or purulent. Red blood-corpuscles are also present in the exudation, sometimes in such abundance as to produce a red color. This hemorrhagic peritonitis occurs chiefly in scorbutic or purpuric cases or in tubercular and cancerous peritonitis. When acute inflammation of the peritoneum results from the rupture of an ovarian cystoma, vascularized fibrous adhesions are eventually formed between the surfaces of the peritoneum and of the tumor. The gelatinous material secreted from the wall of the ruptured cyst may become traversed, enmeshed, and encapsulated by the adhesions, a mass thus being formed resembling a myxoma. To this variety of peritonitis Werth has applied the term *peritoneal pseudo-myxoma*. Bacteria, especially the streptococcus, the staphylococcus, the colon bacillus, and the pneumococcus, are found in the exudation and upon the inflamed peritoneum in septic peritonitis. The streptococcus is especially frequent in puerperal peritonitis. The quantity of liquid exudation which may be present varies from a few ounces to several quarts. When the exudation is both fibrinous and liquid, collections of the fluid are often found enclosed within a mesh-work of fibrin, in which the spaces may sometimes be of considerable size.

The stomach and intestines are distended with gas, often to an extreme degree; their walls are œdematous, and the peritoneum is easily torn and stripped from the muscular coat. In peritonitis from perforation of the stomach or intestine, gas is likely to be present in the peritoneal cavity, and the exudation is thin, opaque, of a greenish tint, and of a fæcal odor. Such an odor may also be present when there is no visible perforation of the intestine, especially if the exudation is circumscribed in the vicinity of the large intestine.

SYMPTOMS.—Abdominal pain is usually the first symptom of acute peritonitis. It is sometimes slight at the outset, gradually increasing in severity, when it is apt to be attributed to indigestion, or it may be sudden and intense, as in peritonitis from perforation; in puerperal cases it is not, as a rule, severe. It may be preceded by a brief period of slight malaise, or may follow a chill. The pain is cutting, piercing, tearing, or griping, and is likely to be aggravated on motion of the patient, even from coughing, vomiting, or defecation, perhaps from drawing a long breath. The dorsal decubitus is usually maintained. The knees are often drawn up, and the pressure of even the bedclothes may be burdensome.

The pain is often localized at first, or it may be present throughout the abdomen. It is frequently referred to the vicinity of the navel, to the epigastrium, or to other regions which may be remote from its place of origin. The source is more correctly determined by the seat of tenderness than by the painful region. The localized tenderness and pain soon become generalized, the suffering being greatest where the normal peritoneum is being invaded, and diminishing as the exudation is formed.

Vomiting is also an early symptom, perhaps the first, but usually quickly follows the incipient pain, and is likely to continue throughout the disease. It is so readily produced by food or drink, even in small quantities, that the patient often refuses them. At first the contents of the stomach are expelled, then bile is vomited, and in the course of a few days the contents of the small intestine, often having the odor and appearance of thin yellow fæces, regurgitate through the incompetent pylorus and are vomited. At times, and especially towards the end of life, the vomit is of a dark-brown color, perhaps flocculent, resembling partly digested blood. Nausea and belching also occur. Hiccough may take place when the peritoneal covering of the diaphragm is inflamed, and often proves a most distressing symptom by causing wakefulness and prolonging pain.

The abdominal walls in the region of incipient pain and tenderness are usually found tense, perhaps retracted. The abdomen soon becomes swollen, however, from the increasing formation and retention of gas within the bowel, its expulsion being prevented by a paresis or paralysis of the muscular coat. With the increasing distention of the bowel the abdomen grows larger, and may be enormous if the abdominal wall is thin and the muscles are stretched and flaccid, as in puerperal peritonitis. In men with powerful abdominal muscles the abdominal distention may be inconsiderable. If intestinal perforation is the cause of the peritonitis, gas in the peritoneal cavity usually aids in producing the abdominal enlargement. With the persistence of the peritonitis, and especially with freedom of action of the bowels, the abdomen is likely to become less distended.

Fever is next in importance to the previously mentioned symptoms of peritonitis. The temperature usually ranges from 100° to 103° F., the evening temperature being, as a rule, higher than that observed in the morning. If the patient is collapsed, the temperature is often subnormal, and it has been found as high as 110° F. shortly before death. The temperature may be lower than 100° F., yet the peritonitis be general, critical, or fatal. In circumscribed peritonitis the pulse may be but little altered in strength or frequency. The severer the peritonitis and the longer its duration, the weaker and more frequent the pulse, which often rises to 130 or 140 and is counted with difficulty or becomes imperceptible.

The respiration is rapid, superficial, and painful. In extreme cases the inspirations may be thirty or forty in a minute, such frequency being in part explained by the displacement upward of the diaphragm, with the resulting retraction of the lungs and dislocation of the heart, and in part by the weakening of the heart by the existing toxæmia. It is painful from the irritation of the nerves in the inflamed peritoneum by the respiratory movements of the diaphragm and of the anterior wall of the abdomen.

The tongue soon becomes covered with a thin, white coat. Later it is dry, brown, and cracked or fissured. There is no appetite. Both food and drink are taken only in small quantities, and are often quickly regurgitated, or the accumulated contents of the stomach, largely undigested, are vomited at intervals of hours. The bowels are usually constipated at the outset, although a few loose movements may take place, while numerous loose dejections may occur in the later stages, and are the rule in puerperal peritonitis. Micturition is frequent and painful when the peritoneal coat of the bladder is inflamed, although retention of urine often occurs in peritonitis, largely from the effect of the opiates administered. The urine is diminished in quantity, dark, acid, and of high specific gravity. It usually contains a trace of albumin, and indican is present in large quantity. The latter is best recognized, according to Jaffé, by adding two or three drops of a fresh concentrated solution of chlorinated lime to equal parts of urine and hydrochloric acid. The mixture when shaken becomes of a dark-blue or bluish-black color.

The mental condition of the patient is often unaffected throughout the disease, although there may be mild delirium or stupor. Symptoms of extreme collapse usually occur early in peritonitis from perforation.

The eyes become sunken, the nose pinched, the skin cold and moist, the voice husky, and the pulse rapid and weak. Such symptoms, less extreme and more gradual, occur later in the disease, when the exudation of a circumscribed peritonitis escapes into the general peritoneal cavity. The expression of the patient is one of suffering and anxiety, and the physical examination of the abdomen is dreaded through fear of an increase of the pain. At the outset the abdomen is not enlarged, and may be retracted, but with the accumulation of gas and exudation the abdomen may become so swollen as to project considerably above the thorax. The overlying skin is smooth and shining, and the outlines of coils of distended intestine are at times to be seen. The heart is displaced upward and outward, and the apex may be found in the fourth intercostal space, and the border of cardiac dulness at the third rib. The upper border of hepatic dulness at times is found at the third rib, and the area of hepatic dulness may be greatly diminished by a rotation of the liver on its transverse axis.

Palpation of the abdominal wall shows that its muscles are tense and resistant in the early stage of the peritonitis, but the resistance diminishes with the progress of the disease, and is least when an abundant liquid exudation is present. Fluctuation may then be found, especially at the dependent portion of the abdomen, and the presence of peritonitic fluid may be simulated if there is much liquid in the intestines, or if the subcutaneous tissue contains abundant fat or fluid. During the early stages of a fibrinous exudation friction sometimes is felt.

The distended abdomen is tympanitic until the exudation becomes considerable. Resonance is generally due to the presence of gas in the intestines, but also results from gas in the peritoneal cavity when peritonitis is caused by perforation of the stomach or intestines. Undue importance has been attached to the disappearance of hepatic dulness as a sign of such perforation. Resonance may replace hepatic dulness, however, when a portion of intestine distended with gas lies between the liver and the abdominal wall, also when the liver is pushed upward and rotated on its transverse axis in extreme enlargement of the abdomen from accumulation of gas in the intestines. Even if gas is present in the peritoneal cavity, it is prevented from accumulating between the liver and the diaphragm where there is obliteration of this space by adhesions. It is therefore important to know the pre-existing limitations of hepatic dulness before concluding that the existing condition is pathological. With the appearance of the liquid exudation, resonance is usually replaced by dulness, which begins in circumscribed peritonitis in that part of the abdomen where the inflammation originates. In general peritonitis the dulness is first found in the dependent portions of the abdomen. In the former the region of dulness remains fixed when the position of the body is changed, while in general peritonitis the dull area shifts as the fluid gravitates. A liquid exudation may not give rise to dulness on percussion if it is separated from the abdominal wall by coils of intestines distended with gas. On auscultation crepitation may be recognized where friction is to be felt. The former may also be heard when the latter is absent, and may be produced by the rubbing of opposed peritoneal surfaces in respiration or peristalsis. Gurgling may be heard with and without the stethoscope when the movements of the bowels are active.

The progress and results of a localized peritonitis are best considered in connection with the diseases which it complicates, and the clinical picture often is so characteristic that especial terms in nosology are applied when certain localities are affected, as subphrenic abscess, perityphlitis, pelvic abscess. If early death does not occur in the course of an acute general peritonitis, a long period of invalidism is likely to ensue. The fever persists, and may follow an irregular, atypical course. Lack of appetite and digestive disturbances are conspicuous. The abdominal enlargement lessens with the free escape of the intestinal contents. Pus may escape by the establishment of fistulæ opening externally, but the tendency is towards progressive wasting and enfeeblement. If the patient survives, the liquid and fibrinous exudations disappear, and are replaced by fibrous thickenings of the peritoneum, forming patches and plates or bands and cords, producing atrophy, disturbance of function, persistent suffering, perhaps death. The liver, spleen, and ovaries are especially liable to be atrophied. Severe disturbance of the function of the stomach may result from the thickening of the peritoneal coat, while chronic obstruction of the intestines may ensue from thickening and adhesions of their peritoneum. The formation of gall-stones and their incarceration in the gall-bladder are favored by the fibrous thickening of the peritoneum covering the latter, and chronic or recurring attacks of appendicitis frequently result from the persistence of the products of a peritonitis in the right iliac fossa. Similar thickenings and adhesions in the pelvis are often sources of organic and functional disease in women, being productive of sterility and ectopic gestation, and interfering with intra-uterine gestation and childbirth.

DIAGNOSIS.—The diagnosis of acute peritonitis rests upon the occurrence of abdominal pain and tenderness, soon followed by vomiting, fever, and abdominal distention, which is at first tympanitic, but later dull on percussion. The diagnosis is doubtful in the early stage alone, when colic and vomiting are the only significant symptoms. Gastro-intestinal colic is commonly relieved by pressure, and often has an obvious cause. Biliary colic is more sharply defined, is associated with less localized tenderness, and a distended gall-bladder may be felt. The usual absence of fever and the frequent eventual appearance of jaundice may be of avail. Renal colic is sharply confined to the region of the kidney or to the course of the ureter, and the examination of the urine is likely to indicate the nature of the pain by disclosing sand, gravel, concretions, or blood. Uterine colic is afebrile, intermittent, and generally associated with menstruation, pregnancy, or a tumor. The pain and tumor from salpingitis remain localized, and are unaccompanied by the vomiting and abdominal distention of general peritonitis, although fever may be present. In acute intestinal obstruction the pain, in its onset, character, and severity, may closely resemble that of acute peritonitis. If the large intestine is obstructed, there is no excess of indican in the urine, as in acute general peritonitis and in obstruction of the small intestine. It may be impossible, however, to make a satisfactory differential diagnosis, and an exploratory laparotomy has frequently been performed for this purpose. Most important for the diagnosis is the recognition of a probable cause for the peritonitis.

Rarely the pain may be so slight as to be insignificant, and the diagnosis of peritonitis then will depend upon the recognition of the exudation. It may be mentioned that a pregnant uterus, an ovarian tumor, an echinococcus cyst, and a full bladder have each been mistaken for peritonitic exudation. Auscultation, catheterization, and perhaps aspiration and examination of the fluid will serve to eliminate these possibilities.

Since most cases of general peritonitis are circumscribed at the outset, and since many cases of circumscribed peritonitis remain localized, and some are more likely to spread than others, it is important to determine if the peritonitis is spreading or likely to become general. It is, therefore, of the utmost practical importance to determine the cause of the peritonitis. For this purpose a thorough knowledge of the etiology of peritonitis, a complete history of the patient's antecedents and symptoms, and an accurate knowledge of the seat of original tenderness are essential.

The spreading of a peritonitis is indicated by the persistence or increase of the vomiting, tympany, elevated temperature, and rapid and weak pulse, in addition to enlargement of the tender and painful area and increase in the quantity of exudation, perhaps discoverable by a vaginal or rectal examination. Undue importance should not be attached to variations in temperature, since generalization of the peritonitis may occur with little or no elevation of temperature.

Prognosis.—Acute general peritonitis usually proves fatal within the first ten days, especially within the first week. If the patient's life is prolonged beyond this period, the disease tends to become chronic, and then often ends fatally in the course of weeks or months, the condition being essentially one of septicæmia.

The prognosis of the case in hand especially depends upon the cause of the peritonitis, the quality of the exudation, and the previous condition of the patient. In general the cessation of vomiting and the return of defecation are favorable signs. A lower temperature combined with a steady pulse and other favorable symptoms is encouraging, but a normal or subnormal temperature may be present in cases of the utmost gravity.

It is also to be remembered that, especially after the first week, patients in whom improvement is apparently taking place may suddenly die from cardiac paralysis. Peritonitis from perforation of the stomach and of the free portion of the intestine into the normal peritoneal cavity is rarely recovered from. Puerperal peritonitis and that following instrumental abortion are often fatal, especially when there is abundant exudation of a more purulent than serous or fibrinous character. If the exudation is thin, acrid, and of an offensive odor, the patient almost invariably dies. If acute peritonitis occurs in a person suffering from serious acute or chronic disease, the prognosis is correspondingly grave.

The prognosis of circumscribed peritonitis is generally favorable, and medical treatment may suffice for the cure. The prognosis of spreading peritonitis is uncertain until the employment of surgical measures, after which the outlook may remain doubtful for a short time, or may immediately improve, and recovery be almost certain.

TREATMENT.—In all forms and degrees of peritonitis it is essential that the patient be kept absolutely quiet on the back in bed, and that every precaution be used to prevent movements of the abdomen. If there be any difficulty whatever in passing urine, catheterization should be at once practised. In sthenic cases the food should be restricted to animal broths and milk (preferably predigested), so as to reduce as far as possible the fæcal residuum. In asthenic cases beef essence and concentrated broths may be used; but solid food should never be given.

External applications to the abdomen may be used in all forms of the disease. In most cases ice-poultices or ice-bags are both agreeable and effective, but sometimes hot applications are more grateful and should be used. Very frequently cold applications are better borne and do better in the early stages of the disease, whilst moist heat is to be preferred at the later period. Counter-irritants have very little control over a pronounced peritonitis; the only form which is effective is the blister, but to effect anything in a wide-spread, general peritonitis this must be so large that it interferes with other local applications and is of itself depressing to the vitality. On the other hand, in a circumscribed peritonitis, after the severity of the attack has been lessened by local blood-letting, blisters may be distinctly advantageous.

The active medical treatment of acute peritonitis varies extremely with the cause and the character of the attack. In sthenic medical peritonitis, beginning as it usually does in a circumscribed centre, the taking of blood from the arm, or, as I have always done it, by free leeching, will influence the disease very markedly. I have repeatedly seen it arrest an attack in which the symptoms were so violent and the cause so evident that there could be no doubt as to the nature of the disease. It is necessary to take enough blood to afford complete relief of pain or to affect the pulse. If leeches be used, not less than from seventy-five to one hundred American leeches or from fifteen to twenty foreign leeches should be applied, and often free after-bleeding from the leech-bites should be encouraged. In septic peritonitis or a peritonitis following perforation in typhoid fever such leeching would almost certainly kill the patient.

The question of the use of purgatives in peritonitis is vital, but difficult to answer. Their use or disuse should be in accordance with the nature of the peritonitis and the character of its cause. If the peritonitis be due to irritant food or other material in the intestinal tract, or if it be accompanied by the presence of fæcal masses in the intestines, there can be no doubt as to the value of saline purgatives. Free serous purgation, further, must greatly empty the intestinal vessels and probably relieve congestion and reduce œdema if it exists, and also lessen the chances of serous exudation. On the other hand, the increased peristalsis which the purgative produces must tend to increase peritoneal irritation, and the argument which has been brought forward that purgation does good by preventing adhesions seems to show, if it be founded on fact, that purgatives are dangerous remedies, because if they increase peristalsis enough to tear adhesions they must greatly irritate the inflamed membrane. It is evident that in a peritonitis depending on perforation purgation is strongly contra-indicated.

It is sometimes argued that purgatives do good in peritonitis by eliminating ptomaines and other products of the inflammation, but of this no proof seems ever to have been afforded. Many laparotomists very highly recommend salines after operation, as tending to check peritonitis; the concurrence of surgical opinion is not absolute, and certainly in medical peritonitis caution is necessary in their use. In many cases it may be judicious to give a single large dose of the saline, and after full action has been secured to withdraw the purgative and check peristalsis by opium.

The opiate treatment of medical peritonitis has been very largely practised, and has great value; the drug not only relieves pain and checks vomiting and peristalsis, but seems, in some way not at present understood, to allay inflammation and to prevent vital exhaustion. The amount of opium required is sometimes extraordinarily large. Dr. Alonzo Clark is said to have given successfully over seven hundred grains in two days. I do not believe such heroic treatment justifiable. I have never given over seventy-five grains of opium in a day, and have at least in one case seen a fatal narcosis produced. It is essential in the use of such doses that the drug be administered in such a way as to secure immediate absorption. No solid preparation of opium should be used. The deodorized tincture may be given by the mouth, but at least the alternate doses should be morphine sulphate (one-fourth to one-half grain) administered hypodermically. Enough opium must be given to produce continuous decided narcotism, but the patient must be carefully watched by a competent attendant, and the drug suspended whenever the narcotic symptoms become pronounced.

In sthenic peritonitis without tendency to the formation of pus I believe calomel to be a valuable remedy. It is, however, essential to avoid its cathartic effects, and it should therefore be given cautiously in small repeated doses (one-sixth of a grain every two hours) during the day. The slightest evidence of ptyalism should be the signal for its withdrawal.

In septic peritonitis, and in the peritonitis which follows perforation, medical treatment is of very little avail. The most that can be done is to administer opium freely and to support the general strength. When there has been perforation, all food should be withdrawn except teaspoonful doses of beef essence every half-hour.

In the management of peritonitis various symptoms must be judiciously met. For the allaying of the thirst, which is often excessive, small pieces of ice should be given; on no account should the patient be allowed to take a large drink of water. The vomiting is usually controlled by the opiate and the ice, but may require small quantities of effervescing draughts. In some cases of excessive vomiting a portion of the opium may be given by the rectum. When there is great tympany a rectal tube should be passed as high up as possible into the large intestine and allowed to remain. Some authorities recommend puncturing the distended intestine with a fine trocar; this I have never done. When collapse and cardiac failure occur, digitalis and strychnine may be given by the mouth, or, better, subcutaneously, and champagne or other alcoholic liquors are allowed. When, however, the vital powers so far fail in peritonitis, death usually occurs: on the other hand, the local irritant influence of alcoholic liquors upon the gastro-intestinal tract is capable of doing much ill, so that stimulants are rarely of real service.

The question of operative procedure in acute peritonitis is one of great importance. According to Richardson, in advanced general peritonitis laparotomy and irrigation are so uniformly fatal that it is safer to take the slim chances of recovery under purely medical care. Early in the disease it is, in my opinion, never justifiable to operate unless the cause is of such character as in itself to indicate surgical interference: of such nature would be a localized abscess, a removable tumor, or a gastric or an intestinal perforation.

In the treatment of the convalescence from peritonitis the greatest care must be exercised to prevent mechanical irritation from without or from within, so that all violent exercise must be strenuously interdicted, whilst constipation and irregularities of diet are carefully guarded against. It is often essential to put on the abdominal bandage. (H. C. W.)

From my point of view all acute peritonitis of any severity is both septic and suppurative to a greater or less extent; leeches, therefore, are harmful, and the cathartic effects of calomel are to be avoided.

The widely divergent opinions as to the use of purgatives in the treatment of acute peritonitis appear to be based largely upon the assumption that the diagnosis is plain and that this affection is always the same. The most expert diagnostician, however, cannot always recognize at the outset the nature of an abdominal affection of which pain, tympany, constipation, and fever are the symptoms. In the great majority of such cases the symptoms, especially in the male, when of obscure or unknown etiology, are due to a peritonitis of intestinal origin, the lesion being at or above the cæcum. The effect of laxatives is to cause the bowels to move, usually with the expulsion of more or less of the contents of the large intestine. It seems more rational to empty this part of the bowel, in case of need, from the nearest point, namely, from the anus, than to irritate the many feet of small intestine above the region of fæcal accumulation. Such irritation is unquestionably dangerous in peritonitis from perforation of the bowel and in strangulation of the intestine, conditions often not to be differentiated in the early stage from a harmless grouping of similar symptoms.

The medical treatment of acute peritonitis, therefore, should consist in the adoption of measures tending to localize the inflammation in the vicinity of its starting-point. Opium is to be given in such quantity only as will relieve pain. All laxatives by the mouth are to be avoided as long as there is any possibility of the alimentary canal being the source of the peritonitic symptoms. Evacuation of the bowels is demanded, not with the hope of aborting or of curing the disease, but for the sake of promoting the expulsion of the intestinal contents, the retention of which, especially when gaseous, is often a source of marked discomfort. If the localized peritonitis persists or the inflammation tends to become generalized despite the medical treatment, surgical measures alone offer a more promising outlook. (R. H. F.)

CHRONIC PERITONITIS.

The possibility of the termination of acute inflammation of the peritoneum in chronic peritonitis has been already mentioned. In such cases there is a gradual improvement in the symptoms suggestive of a prolonged convalescence from the acute attack, although permanent disturbances of the function of the alimentary canal, the liver, and the gall-bladder, and of the genito-urinary apparatus of the female, may remain as evidence of the previous disease. Such a chronic peritonitis, like the acute attack, is either circumscribed or diffused. An explanation of the origin of such chronic attacks is to be found in the semi-fluid, caseous, or calcified masses of inflammatory exudation due to an acute attack and encapsulated by thickened peritoneum or dense adhesions. The contents of such circumscribed portions of the peritoneal cavity may be discharged through communications established between them and the interior of the intestine or the bladder, or may escape externally through the abdominal wall. The course of the chronic peritonitis thus becomes complicated and protracted.

The rare occurrence of the chronic hemorrhagic peritonitis described by Friedreich and caused by repeated abdominal tappings in the treatment of ascites may be referred to. Layers of a thick granular membrane containing numerous nodules of extravasated blood were found adherent to the visceral and parietal peritoneum.

The productive manifestations of a chronic peritonitis may be manifested by fibrous thickenings and peritoneal adhesions found after death in those regions which are most frequently the sites of an acute peritonitis. In the history of the patients, however, there is frequently no recognition of any disturbances which would suggest the possible discovery of such lesions. This evidence is significant that attacks of peritonitis may be of sufficient duration to produce permanent alterations, yet be productive of such slight disturbance as to be disregarded.

CHRONIC SEROUS PERITONITIS. CHRONIC GRANULAR PERITONITIS.

Within a few years especial attention has been called to the occurrence of cases of chronic diffuse peritonitis in which large quantities of liquid exudation have been found. Such cases had been previously confounded with ascites or with tubercular, cancerous, or sarcomatous peritonitis.

Etiology.—That this affection has been called idiopathic or essential is sufficiently indicative of the recognized obscurity of its origin. Among the exciting or predisposing causes are enumerated injury, exposure to cold, profuse and protracted diarrhœa, measles, typhoid fever, syphilis, and vulvo-vaginal catarrh. What is of greater practical importance is the fact that girls are predominantly affected after the age of three years, and frequently at the beginning of puberty. It is probable, as suggested by Henoch, that many of the cases of supposed peritoneal tuberculosis with abundant liquid exudation successfully treated by laparotomy belong in this series.

Morbid Anatomy.—The anatomical changes are in general rather a matter of inference than of observation. Hirschberg, in a case dying from intercurrent disease, found the peritoneum normal except over a portion of the colon where it was irregularly thickened. At a laparotomy the peritoneum was seen by Henoch to be studded with small nodules resembling tubercles. These proved to be composed of fibrous or granular tissue, bacilli and giant-cells being absent. Other observers, Welch among the first, have noted similar lesions, and the term chronic granular peritonitis has been applied to this variety.

Symptoms.—Progressive distention of the abdomen, often becoming extreme, apparently from free fluid, is the characteristic symptom. It may be considerable and the patient present no other symptoms. On the other hand, it may be associated with loss of appetite, pallor, and wasting. Fever, pain, and tenderness are usually lacking, and the discomfort, perhaps slight, is attributed to the pressure and weight of the liquid exudation.

Months generally elapse before the abdominal distention becomes considerable, although at times a few weeks only are necessary. Occasionally temporary variations in the size of the abdomen may occur without any special modification of the general course of the disease. In certain cases a continued diarrhœa or persistent diuresis is associated with the absorption of the fluid.

The physical signs indicating the presence of free fluid are those already mentioned in connection with ascites. The fluid may be more serous, sero-fibrinous, or sero-purulent, and is richly albuminous. Its inflammatory origin is thus indicated, although fibrin or pus-corpuscles may be absent. At times, after removal of the fluid by tapping or absorption, dense rounded masses have been felt in the abdomen, and in certain instances have been found to be circumscribed thickenings of the peritoneum of the omentum, mesentery, or intestine.

DIAGNOSIS.—Since the characteristic sign is excessive free fluid in the abdominal cavity, and symptoms are often wanting, it becomes necessary to differentiate this affection from ascites and tubercular or cancerous peritonitis. The age and sex may prove of value in excluding ascites, as may the absence of digestive disturbances, jaundice, enlarged spleen, clay-colored stools, itching, and hemorrhage. The negative character of the symptoms, and the absence of abnormal signs on auscultation of the heart and lungs, and of albuminuria, blood, and casts on examination of the urine, are of importance in eliminating other sources of ascites. Especial importance is to be attached to the specific gravity of the aspirated fluid, which is likely to be above 1015, while that of ascites is below this point. Tubercular peritonitis is excluded with more difficulty. The latter affection sometimes progresses, for a while at all events, with but little constitutional disturbance. The macroscopic appearances of the lesions as found by Henoch and others may not differ from those present in tuberculosis. The enlargement of the abdomen in tubercular peritonitis, as a rule, progresses more rapidly, and is more likely to be associated with elevation of temperature, pain and tenderness, progressive emaciation, and loss of strength. The physical signs of tuberculosis elsewhere in the patient, or its presence in other members of the family, would favor the diagnosis rather of tubercular peritonitis than that of chronic granular or serous peritonitis. Cancerous peritonitis might be excluded by the youth of the patient and failing cachexia and tumors.

PROGNOSIS.—In the light of our present knowledge the prognosis is favorable, since recovery usually takes place unless prevented by intercurrent disease or unsuccessful surgical treatment. Months are generally required for the absorption of the fluid.

TREATMENT.—The diet in chronic peritonitis should be light, easily digested, and nutritious. Confinement to bed may or may not be necessary, but if possible an abundance of out-door life in a hammock or otherwise should be secured. Symptoms should be carefully combated as they arise: thus, laxatives may be useful if there be constipation, astringents if there be diarrhœa. The long-continued administration of minute doses of corrosive sublimate (one-fiftieth of a grain) or of potassium iodide (one to two grains), or, especially to children, of ferrous iodide, is justifiable. Great benefit has been asserted to be produced by various local applications, especially the solution of iodine in olive oil (seven to thirty grains to the ounce) and mercurial ointment. Pribram strongly recommends gentle friction once a day with soft soap (*vulgo*, "green soap") and water, followed by the continuous application of oil-silk or thin rubber cloth, and steadily maintained until the skin becomes hard and scaly. When there is excessive fluid, tapping has been largely practised; but it is advisable, if the symptoms continue notwithstanding treatment, to have laparotomy performed.

TUMORS OF THE PERITONEUM.

Tumors grow from the free surface of the peritoneum and from the subperitoneal tissue. Among the former are the sarcoma, endothelioma, and cancer, which belong to the malignant tumors. The benignant cystic, dermoid, and teratoid tumors of the ovary, although lying within the peritoneal cavity, do not originate from the peritoneum, while the malignant adenoma of the ovary, also lying within the peritoneal cavity, may after rupture of the cyst-wall become extended as a secondary growth to various portions of the free surface of the peritoneum. The tumors which proceed from the subperitoneal tissue, especially of the omentum and the mesentery, are often benignant, as the myxoma, fibroma, lipoma, hæmangioma, chylangioma, and entero-cysts, but may be malignant, as the sarcoma and cancer. The malignant tumors are more frequently primary in the abdominal viscera, their growth being continued into the subperitoneal tissue. Some of these tumors are of purely pathological interest; others, in virtue of their size and resulting mechanical disturbances, demand surgical treatment. Those which are of especial importance to the physician are the malignant tumors of the peritoneum, which are conveniently described as cancer, although the structure may prove to be that of a sarcoma, endothelioma, or malignant adenoma.

CANCER OF THE PERITONEUM.

ETIOLOGY.—Cancer of the peritoneum is sometimes primary, but usually secondary, proceeding from parts covered by peritoneum. In the latter case the primary growth is to be found in the alimentary canal, especially in the stomach, in the large intestine (cæcum, sigmoid flexure, and rectum), and in the œsophagus. Cancer of the peritoneum may be secondary to cancer of the ovary, uterus, kidney, pancreas, liver, gall-bladder, or suprarenal capsule. It may be secondary to primary disease in more remote parts of the body extended through the blood-vessels or the lymphatic apparatus, especially from the retroperitoneal glands. It usually occurs after middle life, and its etiology is as obscure as that of cancer elsewhere.

Morbid Anatomy.—All the varieties of cancer conveniently described as scirrhous, hard or fibrous, encephaloid, soft or medullary, pigmented or melanotic, colloid or hyaline, may be found, the last most frequently. The tumors may be directly continued to the peritoneum, or there may be several centres of growth,—so-called metastatic tumors. Single or multiple growths thus arise, the latter sometimes so minute as to resemble miliary tubercles. All tend to enlarge and to become fused, resulting in the presence of masses which are sometimes enormous. The omentum and the mesentery are especially liable to be the seats of large tumors. Nodules of considerable size may be found early in the progress of the disease in Douglas's fossa. Cancer of the peritoneum is frequently associated with evidences of ascites or of peritonitis. The ascitic fluid is sometimes milky, from the presence of abundant fattily degenerated cells,—*adipose ascites.* The peritonitic liquid exudation has a high specific gravity, above 1016, in virtue of the abundant albumin, and is often hemorrhagic. Fibrous thickenings and adhesions, the latter sometimes producing obliteration of portions of the peritoneal cavity, are often present.

Symptoms.—There may be no symptoms associated with cancer of the peritoneum, the condition being first recognized at an autopsy. If symptoms are present, they are usually attributable to the associated ascites or peritonitis, or are dependent upon disturbances in the function of the organ in which the disease arises. The ascitic symptoms are conspicuously mechanical, especially the disturbed respiration and circulation. The peritonitic symptoms also are partly mechanical, but in addition there are likely to be pain, usually moderate, and fever, as a rule slight and irregular. Colic, constipation, and meteorism are not infrequent results of obstruction to the function of the bowels by the growth of the cancer. Symptoms of sudden, severe, perhaps fatal, anæmia may occur in consequence of rupture of large and thin-walled blood-vessels in excessively vascular varieties of cancer, and an acute peritonitis, either circumscribed or diffused, in the latter case rapidly fatal, may result from perforation of the intestine infiltrated with cancer. As the disease progresses, pallor, wasting, and debility are likely to occur. On physical examination tumors are often felt, although there may be so much fluid as to prevent their recognition until the liquid has been removed. In repeated instances a growth of the cancer has taken place along the track of the trocar used in withdrawal of the fluid. Rectal or vaginal palpation may disclose a tumor in Douglas's fossa when external palpation indicates merely the presence of fluid. If tumors are found, they may be fixed or floating, superficial or deep-seated. When fluid is aspirated, it is likely to have a high specific gravity, and to contain large and irregularly shaped cells, in which fat, hyalin, or glycogen may be found. At times a gelatinous fluid may be aspirated in which the structural characteristics of a malignant tumor are present.

DIAGNOSIS.—The diagnosis is based usually upon the recognition of the presence of abdominal tumors, often movable, associated with liquid, and preceded by disturbances of function in some one of the abdominal organs. It is confirmed by the aspiration of cancerous tissue, or by the withdrawal of fluid the specific gravity and glycogenic cells of which (according to Quincke) indicate its malignant origin. The differential diagnosis usually is between ascites and tubercular peritonitis. The presence of tumors after withdrawal of the fluid may exclude the former, while the absence of a family and personal history of tuberculosis, with evidence of this disease elsewhere in the body, would aid in the exclusion of tubercular peritonitis.

PROGNOSIS.—Since peritoneal cancer is usually secondary, not only does it have the general mortality of cancer, but its presence also indicates that the later stages of this disease are at hand, and that the patient has but a few remaining months to live. An immediately fatal issue may follow intra-peritoneal hemorrhage or perforation of the intestine. Primary cancer of the peritoneum is usually of slow growth, although eventually fatal.

TREATMENT.—The medical treatment of cancerous tumors of the peritoneum has no other effect than to afford temporary relief of the symptoms.

SECTION VI.

DISEASES OF THE URINARY APPARATUS.

CHAPTER I.

DISEASES OF THE KIDNEYS.

ANOMALIES OF SHAPE AND POSITION.

ANOMALIES in the size, shape, number, and position of the kidneys exist as a result of developmental or pathological disturbances, and are usually not productive of symptoms unless associated with pathological conditions elsewhere. The most important of these anomalies are the fused kidney and the floating kidney.

The *fused kidney*, which often, though not necessarily, suggests the shape of a horseshoe, is usually displaced downward, lying near the sacral promontory, and may be found in the pelvis. A single kidney, usually the left, also may be displaced downward. Such dislocated kidneys may be mistaken for abdominal or pelvic tumors, have obstructed labor, and a dislocated fused kidney has been the immediate cause of death by pressing upon the inferior vena cava and producing thrombosis and embolism.

FLOATING KIDNEY. MOVABLE KIDNEY. WANDERING KIDNEY. NEPHROPTOSIS.

Although a slight degree of mobility of the kidney normally exists in connection with respiration, the resulting change of position is usually insufficient to permit the lower edge of the kidney to be felt. Not infrequently an otherwise normal kidney is freely movable even within wide limits and readily palpated.

ETIOLOGY.—The movable kidney is stated to be found from five to ten times more often in females than in males, and is especially frequent in adults from thirty to fifty years of age, although it may be found in old age and in childhood. Congenital predisposing causes, as a superabundance of peritoneum, lax perinephric tissue, and elongated renal blood-vessels, are probably of importance. Among the acquired causes are the emaciation of a previously fat person and sudden changes in the resistance of the abdominal wall, such as may occur after parturition or the removal of abdominal tumors. Pressure of tumors or of the pregnant uterus on the kidney, or increased weight of the organ, as from tumors or hydronephrosis, favor its displacement and mobility. It is maintained that repeated congestions of the kidney may occur during menstruation and thus promote the occurrence of the floating kidney. The use of corsets is often considered to be an important cause, and in considerable part explanatory of the greater frequency of this anomaly among women. It is probable, however, that dislocation of the kidney must exist before the organ can be made movable by the pressure of corsets. Traumatism and prolonged excessive muscular contractions are also usually included among the causes. Of late years, since the publication of Glénard's work on enteroptosis, it has been generally recognized that movable kidney is frequently associated with displacement of the stomach or the intestine, of the liver, and of the uterus, often in combination with nervous, digestive, and nutritive disturbances, especially in young persons of a chlorotic type, thus rendering still more conspicuous the agency of congenital conditions in the production of the floating kidney.

MORBID ANATOMY.—The post-mortem recognition of the floating kidney is extremely rare, due in all probability to the failure to seek for this condition. Rotch has found in an analysis of eight hundred and sixty-seven cases that in upward of eighty per cent. the right kidney was affected, while the left kidney or both kidneys were movable in about ten per cent. of the cases. Elongation of the blood-vessels of the kidney, a curved ureter, hydronephrosis or pyonephrosis, limited twists of the ureter and blood-vessels, and adhesions to the transverse colon or liver have been found associated. The possible displacement of other abdominal organs has already been mentioned.

SYMPTOMS.—Floating kidney often produces no symptoms. Frequently it is accompanied with disturbances not attributable to the mobility of the kidney, and when symptoms are referred to the kidney they are usually associated with abnormalities of function elsewhere. The patient may be aware of the existence of a floating kidney during years of health, but may not assign importance to it until he has become weakened from other causes.

The floating kidney may produce discomfort by causing a sensation of pressure or dragging and a feeling as of some moving object in the abdomen. Such symptoms may become apparent on change of position, especially when lying on one side or in stooping, when the floating kidney may become painful and tender. The pains may be fixed or shooting, and may be referred to other parts of the abdomen, as the back or the groins, or to other parts of the body, as the chest or the extremities. The patients are often neurasthenic, and in women hysterical symptoms, especially at the menstrual period, are likely to occur, while men are frequently hypochondriacal.

Disturbances of digestion, such as loss of appetite, nausea, vomiting, epigastric pressure or weight, flatulence, and constipation, are frequent. Jaundice sometimes occurs, rather as the result of a duodenal catarrh than as attributable to the pressure of the floating kidney on the common bile-duct. The digestive disturbances are those usually regarded as evidences of a nervous dyspepsia, although gastric catarrh, dilatation of the stomach, and, especially, prolapse of this organ, may exist. The dilatation of the stomach associated with floating kidney is often attributed to the direct pressure of the latter upon the pylorus or the duodenum. Landau attaches importance to the floating kidney in the production of intermittent or permanent hydronephrosis and pyonephrosis, and asserts that in such cases traumatism may prove an immediate excitant of these complications by producing a twist of the ureter. Dietl first called attention to attacks of sudden, intense abdominal pain, followed by tenderness, swelling of the abdomen, and symptoms of collapse, perhaps with vomiting, chills, and fever, during which he found the region of the movable kidney painful and extremely sensitive, while palpation and percussion indicated the presence of a tumor in this region. The urine became scanty and often contained blood. In the course of a week the distressing symptoms diminished in severity and the flow of urine became abundant. Dietl considered that these symptoms were due to a circumscribed peritonitis from an incarceration of the kidney in the peritoneum surrounding it, while Gilewsky regarded the condition as an acute hydronephrosis from compression or twisting of the ureter, and Landau attributed it to a disturbance of circulation in the floating kidney caused by obstruction of the renal vessels, especially the vein, in consequence of a displacement or twist of the floating kidney.

Although the position of the floating kidney is such that it is usually covered by more or less resonant intestine, it may exceptionally lie directly beneath the abdominal wall uncovered by intestine, and give rise to dulness on percussion. The floating kidney is usually recognized without difficulty by palpation, and the patient not infrequently has learned the most efficient means of causing it to come within reach. It may be that the sitting or lateral position is best adapted for this purpose, or the patient may better succeed by bending the body forward. The physician usually most conveniently recognizes the floating kidney when the patient lies on the back near the edge of the bed, the muscles being relaxed as thoroughly as possible, the knees perhaps being raised for this purpose. The finger-tips of the one hand (the left if the right kidney is being examined, and the reverse in the case of the examination of the left kidney) should be pressed firmly against the right lumbar region while counter-pressure is applied from the front, the finger-tips being moved about. If the circumscribed, smooth, rounded, and dense kidney is not to be felt, the patient should be asked to draw a long breath, when the lower portion of the descending kidney is often appreciated. The floating kidney when found out of place may lie as low as the brim of the pelvis, or on the opposite side of the median line, or directly beneath the anterior abdominal wall, and, as a rule, is readily returned to its normal position.

Most observers find that the urinary secretion is in no way modified. Landau, however, states that he often has observed alterations in the quality and quantity of the urine, which may be increased or diminished even to complete suppression, as in the class of cases referred to by Dietl, and may contain blood, especially when the region of the kidney is painful. The presence of pus is to be expected when pyonephrosis is a complication.

DIAGNOSIS.—The diagnosis of the floating kidney is made by palpation, which determines the seat, shape, size, and consistency of the abdominal tumor, which can usually be pushed into the site of the kidney. In cases of doubt the patient should be palpated in the knee and elbow position, in order to allow the kidney to fall forward. Since the symptoms attributed to a floating kidney may occur in its absence, and the floating kidney be often found without symptoms, the rational signs afford but little aid in diagnosis. The patient is often the first to find the tumor, which is regarded as a curiosity by some and the cause of serious disturbance by others. It may be judicious for a physician, if he is the first to discover the abnormality, to refrain from giving the information to his patient, since the latter might exaggerate its pathological significance.

Retained fæces, a dropsical gall-bladder, a tongue-shaped appendage to the right lobe of the liver from constriction or growth, and pedunculate tumors of the uterus or ovary, are the conditions most often to be differentiated. The free use of laxatives will cause the disappearance of the fæcal tumor. Tumors connected with the liver are more constantly superficial, and the degree of their mobility is more largely controlled by that of the diaphragm, while the inability to replace them in the region of the kidney will usually suffice to avoid confounding such tumors, as well as the pedunculate uterine or ovarian tumor, with the kidney. Cancer of the stomach or the intestine when physically simulating the movable kidney, if not at first to be differentiated by the symptoms, soon becomes characterized by severe digestive disturbances. The difficulties of differential diagnosis are such that an exploratory laparotomy has frequently been undertaken and the renal nature of the abdominal tumor first recognized by this means.

PROGNOSIS.—The floating kidney may eventually become fixed and incapable of recognition and the discomfort disappear. This result may be owing to the accumulation of fat-tissue, to pregnancy, or to mechanical or surgical treatment. The prognosis as regards relief from the permanent mobility of the kidney is, therefore, favorable. The symptoms attributed to the floating of the kidney may persist when the latter is no longer movable, and may disappear although the kidney is still palpable. The prognosis of the symptoms attributed to the floating kidney is rather that of the associated chlorosis, neurasthenia, or hypochondriasis, and the distress may often be relieved by an intelligent appreciation on the part of the patient of the significance of the floating kidney, as well as by the fixation of this organ. It may also be noted that the symptoms attributed to a floating kidney not infrequently disappear after the climacteric. If hydronephrosis or pyonephrosis is caused by the floating kidney, the prognosis may then be more favorable than if these conditions were due to other causes. The surgical treatment of floating kidney of late years has made its prognosis as to life more serious.

TREATMENT.—Restoration of floating kidney can usually be obtained without difficulty by placing the patient upon the back and gently pushing the kidney in place. If medical advice has been sought immediately after the forcing out of the kidney by some strain, it is worth while to attempt by enforced rest in the recumbent position to bring about permanent natural fixation of the organ. Very rarely, however, in practice do the circumstances favor such an attempt. The effort should always be made to keep the kidney in its place by mechanical means. We have seen complete cures thus obtained, although the result is often unsatisfactory. The best bandage is made of silk elastic closely fitted to the whole abdomen of the patient, and prevented from riding up by means of straps of soft rubber tubing or similar material, one on each side, passing from back to front between the legs. Over the position of the dislocated kidney is sewed on the inside of the bandage a round pocket of soft chamois-skin, left open above so that a pad can be pushed into it and changed on occasion. Success depends largely upon the skill of the maker in fitting and adjusting and the patience of the subject in enduring annoyance until habit has produced toleration.

The medicinal palliative treatment in floating kidney is often very effective in the relief of symptoms, but so closely depends upon the adaptation of the means to the individual case that we can do little more than point out the general principle that a dislocated organ is extremely irritable, and that the reflex and other symptomatic phenomena are chiefly due to this irritation. If a gouty diathesis exists, its effects will be greatly exaggerated, so that careful treatment for this condition often brings about the most happy results. Bromides and other sedatives allay nervous irritability for the time being, but are temporary in their action, and are to be avoided as much as possible.

The question of surgical operation is to be decided largely from the circumstances of the case. The daily occupation, the amount of influence upon the general health, the physical discomfort, and the courage of the patient, all are elements in making up an opinion. Two operations are performed,—nephrorrhaphy, or stitching the kidney to the posterior abdominal wall, and nephrectomy, or removal of the kidney. The former of these is a comparatively safe operation; according to Delvoie, in two hundred and fifteen cases there were five deaths and one hundred and thirty-five recoveries. Nephrectomy is much more serious; Sulzer gives the mortality at twenty-seven per cent., Newman at thirty per cent.

Nephrectomy for movable kidney is never justified unless the symptoms are very severe and disabling and nephrorrhaphy has been tried twice and failed.

ABNORMALITIES OF THE URINE.

The importance of an examination of the urine both in diagnosis and in prognosis is such that it is desirable to call attention to some of its most important abnormalities before considering the diseases of the kidney, the recognition of which is so frequently dependent upon the condition of the urine. The variations in the *quantity* and *quality* of the urine passed during the day and at night may make it important not only to ascertain the total amount passed within twenty-four hours, but also to make separate measurements and separate chemical and microscopical examinations of the urine voided during the day and of that secreted in the night. Errors due to the examination of a single specimen may be avoided if a sample, six or eight ounces, of the mixed and preserved total amount passed in the twenty-four hours is investigated.

Although the normal quantity passed in twenty-four hours is about three pints, variations in this amount, unless excessive (diminished to one pint or increased to three quarts), are within physiological limits. A diminution in quantity is of especial significance in varieties of nephritis, and if persistent in any disease is an important danger signal. An increased quantity of urine, three quarts and upward, polyuria, may be an important sign of renal disease, or a characteristic symptom of diabetes, although an excessive flow of urine may be indicative of no immediate danger, but rather of a favorable condition when it gives evidence of the absorption of pathological accumulations of fluid in various parts of the body.

The normal *color* of the urine varies largely in accordance with the quantity passed. Abnormal coloration is the result of the presence of pigments derived from the body or from without. Of the former, biliary pigment is the most frequent, and is considered in the section on jaundice. The urine may be abnormally colored from blood-pigment, or from a transformation of some of the constituents of the tissues, whether normal or pathological, or from the presence or action of agents introduced from without. Rhubarb and senna may produce shades of brown (reddish brown if the urine is alkaline), and santonin a yellow or greenish tint simulating the discoloration produced by biliary coloring matter. Carbolic acid, salol, and pyrogallic acid in sufficient quantity produce a dark-green or black color, the urine becoming darker in color the longer it is exposed to the air. When the urine contains chyle the appearance resembles that of milk.

Blood coloring matter may be present in the urine, either contained within the red blood-corpuscles, hæmaturia, or separated from them in solution or precipitated, hæmoglobinuria.

Hæmaturia.—Red blood-corpuscles, pigmented or decolorized, may be found in the urine with the microscope when not present in sufficient quantity to cause macroscopic alterations in color. Blood not proceeding from the urinary tract may be present in the urine, especially in that of the female, the source of the bleeding being uterine, vaginal, or anal. The physical examination of these possible sources, or catheterization of the bladder to eliminate extra-vesical hemorrhage, will suffice to make clear the region in which the bleeding takes place.

The color of the urine in hæmaturia varies in accordance with the quantity of blood present, the length of time it has been in the urine, and the secondary changes it may have undergone after the urine has been voided. The color, therefore, may be more red or brown. The presence of red blood-corpuscles is determined by the microscope, and blood-pigment is precipitated with the earthy phosphates by heating the urine in a test-tube after one-third its volume of liquor potassæ has been added. To determine the source of hemorrhage the urine is allowed to stand undisturbed for several hours. If the hemorrhage arises from below the kidney, and especially from the bladder, a sediment of red blood-corpuscles rapidly forms, and the overlying fluid, particularly in urine which has not been long retained, is relatively free from blood coloring matter, and contains but little albumin unless there is concurrent disease of the kidney. Blood-clots are likely to be present, perhaps worm-like and sometimes decolorized if from the renal pelvis or the ureter, while those from the bladder are larger, of irregular shape, and often shreddy. A vesical source of the hemorrhage is to be suspected if at the end of micturition the urine appears more bloody than that first passed, and if after emptying the bladder the catheter is inserted and repeated washings are returned stained with blood. If the blood is of renal origin, the color is of a less bright red: the sediment contains no clots unless large vessels are ruptured, and the uppermost portions of the urine contain more or less albumin. Both blood-casts and other varieties of casts may be found in the sediment, and, according to Gumprecht, fragments of red blood-corpuscles are present, which is not the case when the hemorrhage arises at a point below the kidney.

ETIOLOGY.—The causes of hæmaturia are local and general. Renal hæmaturia is the result of injury to the kidney, renal thrombosis and embolism, nephritis, both acute and chronic, malignant tumors, calculi, and parasites of the kidney. Calculi, local tuberculosis, acute cystitis, and villous tumors of the bladder are the most frequent local causes of hæmaturia from below the kidney. General causes of hæmaturia are scurvy, purpura, hæmophilia, and malaria.

DIAGNOSIS.—The recognition of a traumatic cause for renal hæmaturia is sufficiently obvious. The diagnosis of a thrombus of the renal vein is based upon an appreciation of the local and general causes of thrombosis. The diagnosis of embolic renal hemorrhage, which is but transitory, demands evidence of a probable source of embolism in the left ventricle of the heart or in the aorta. If the hemorrhage is due to nephritis, the small quantity of blood, the excessive amount of albumin, the nature of the sediment, and the accompanying dropsy suffice to explain the cause of the hæmaturia. Cancer of the kidney is always to be suspected as a source of prolonged renal hemorrhage of obscure origin, although the physical evidence of a tumor not infrequently may occur some time after the appearance of the hemorrhage; if the hæmaturia be accompanied by a general unaccountable failure of health and a heavy persistent pain, either in the kidney itself or, as often happens, above and anterior to the usual seat of renal pain, the diagnosis of cancer will be justified. The parasitic source of the hemorrhage is manifested by the discovery of filariæ in the urine or in the blood when sought for at night. Renal colic or vesical pain, obstruction to the flow of urine, discomfort on motion, and the frequent presence of pus are suggestive of calculi and cystitis as the cause of the hemorrhage, while the characteristic bacilli and villosities may make clear the diagnosis of tuberculosis or villous tumor. The symptoms characteristic of the general causes of hæmaturia are described at length in the consideration of the diseases mentioned. Malaria as the cause of hæmaturia is made evident by its association with the symptoms and signs of this disease, its frequent occurrence during a period of years, and the absence of other symptoms indicative of disease of the urinary tract.

TREATMENT.—The radical treatment of hæmaturia is that of the condition which produces it. For the relief of the hemorrhage, gallic acid—ten to fifteen grains every two to four hours—is usually the most effective remedy, and has the advantages of not being irritant to the kidney and of not influencing seriously the general system. Extract of ergot is sometimes useful. When there is no irritation of the kidney, various volatile oils may be serviceable; at the head of these we would place oil of erigeron, ten to twenty drops every two to four hours. Oil of turpentine is more irritating and usually less efficient. In continuing cases, ferric chloride, one to two grains, or tincture of ferric chloride, ten to twenty minims, may be used. Monsel's solution, two to five minims every two to four hours, sometimes does well. All these remedies must be given freely diluted.

Hæmoglobinuria.—This term is applied to the presence of the coloring matter of the blood in the urine, few or no red blood-corpuscles being present. According to Hoppe-Seyler, the coloration is due to methæmoglobin, whose presence is indicated by means of the spectroscope. That the abnormal color of the urine is due to blood-pigment is made evident by its precipitation with liquor potassæ, and, if necessary, by the production of hæmin crystals in the dried pigmented sediment when it is heated after the addition of a little glacial acetic acid and a few crystals of common salt. The diagnosis of hæmoglobinuria is thus based upon the presence of blood-pigment and the absence of red blood-corpuscles.

ETIOLOGY.—Hæmoglobin occurs in the urine when the blood coloring matter is freed from the red blood-corpuscles in the blood-vessels, hæmoglobinæmia, although it may take place when there is no appreciable free hæmoglobin in the blood, and may occur in the course of severe infectious diseases, especially in scarlet fever, erysipelas, typhoid fever, and malaria, in which micro-organisms or their products are probably the efficient cause in setting free the hæmoglobin. W. S. Bigelow has called attention to this condition in infectious diseases of new-born children, and Winckel has described its epidemic occurrence among them. A certain importance in etiology is to be attached to heredity. Hæmoglobinuria may be the result of extensive burns and of poisoning with various agents, the more important of which are mushrooms, carbolic acid, naphtol, pyrogallic acid, corrosive sublimate, and potassium chlorate, or of the introduction into the veins of foreign blood or serum; in an animal the intravenous injection of pure water may dissolve the red blood-corpuscles and produce hæmoglobinuria. Its periodical occurrence is usually regarded as a disease, paroxysmal hæmoglobinuria. The occurrence of hæmoglobinuria in connection with the above-mentioned causes adds greatly to the gravity of the prognosis.

The urine often is red when passed, becoming darker on standing, with subsequent fading of the color, and the odor may resemble that of fresh meat, from the presence of hæmatoporphyrin. This pigment is a derivative of hæmoglobin, essentially hæmatin without iron, and is to be recognized by means of the spectroscope. According to Garrod, it is almost always present in the urine, and a moderate variation in its quantity is of no especial significance. It has been found notably increased in poisoning by sulphonal and by trional, and has been observed in excess in the urine of the insane and the neurasthenic and of typhoid patients. Such excess is to be regarded as a bad prognostic sign.

TREATMENT.—The radical treatment of hæmoglobinuria is that of its cause. As the destruction of the red blood-corpuscles does not take place in the kidneys, it is better that they should remove liberated hæmoglobin from the blood: so that styptics are of little value.

Paroxysmal Hæmoglobinuria.—Syphilis and malaria are stated to offer a predisposition to this condition. It occurs most frequently in male adults, and the paroxysms may be excited by exposure to cold, even by dipping the hands or the feet in cold water, and, according to Chvostek, by ligature of the finger. Mental excitement or physical exertion may also bring on an attack.

SYMPTOMS.—The paroxysms consist frequently of a chill followed by fever, the temperature rising to 104° F. Pains are present in the back and hips and sometimes in the extremities, respiration is labored, and the chest feels constricted. The skin in general is pale, although the finger-tips and ears are cyanotic, and there is slight jaundice at times, also urticaria or circumscribed œdema. Defervescence, with sweating and relief to the symptoms, takes place during the course of a few hours. During the attack the urine has the characteristic dark red or brown appearance, and amorphous hæmoglobin is found with the microscope. Casts, renal epithelium, and crystals of calcic oxalate may be present. Albumin may be detected in the urine at the beginning of the attack before the appearance of the hæmoglobin, and it may persist for several days after the hæmoglobin has disappeared. Auscultation of the heart often discloses a faint systolic murmur, and the liver and spleen at times are found enlarged. Between the attacks the patient merely appears pale and weak.

PROGNOSIS.—Paroxysmal hæmoglobinuria is a chronic affection, lasting for years. It is not known as an immediate cause of death, and recovery may take place.

TREATMENT.—The treatment of paroxysmal hæmoglobinuria dependent upon malaria has already been described. The treatment of a nonmalarious attack should be purely symptomatic; between the attacks attention should be directed to the building up of the general strength of the patient, and especially to the removal of any diathetic or other irritation of the kidneys. If diuretics be used, they should always be of the non-irritating class, such as potassium bitartrate.

Urobilinuria.—Urobilin and indican, normally present in the urine, may be in such excess as to give rise to a dark red or brown color. Liebermann has shown that urine containing an excess of urobilin when shaken produces a yellow foam. Its presence may be determined by adding sufficient ammonia to the urine in a test-tube to make it strongly alkaline, then adding eight to ten drops of a ten per cent. solution of chlorate of zinc and immediately filtering the specimen. Transmitted light causes the filtrate to appear of a red color, but with a dark background and by reflected light the color is fluorescent green. The presence of urobilin may be inferred in high-colored urine if bilirubin and indican are shown to be absent. It is found in excess in fevers and in internal hemorrhage; it may also exist in diseases of the liver, especially in fibrous hepatitis, and serve as the cause of a mild jaundice. Its recognition may be of especial clinical importance in cases of concealed hemorrhage, especially in ectopic gestation, in which excessive urobilinuria may indicate that the absorption of extravasated blood is taking place.

Indicanuria.—An excess of indican, sulphate of indoxyl, also produces a dark brown color of the urine, and is to be suspected if the urine does not foam when shaken and contains no biliary coloring matter. Its existence may be determined by Jaffé's test, which consists in adding equal parts of urine and strong hydrochloric acid in a test-tube. A solution of chlorinated soda, 1 to 20, is then added drop by drop, the mixture being shaken. If indican is present a green color is formed, or if it is in excess the urine becomes blue from the formation of indigo, and the green or bluish color will disappear on the addition of an excess of chlorinated lime. If there is no increase of indican the urine becomes red.

This coloring matter is formed from indol, which is produced as the result of the action of intestinal bacteria on albumin. Indol is absorbed, transformed into indoxyl, and, in combination with sulphuric acid, eliminated in the urine as indican.

An excess of indican in the urine indicates increased putrefaction of albumin in the intestine: hence it is present when there is prolonged stagnation of the intestinal contents in the ileum. In stagnation of the contents of the large intestine there is no considerable increase of indican, since in this portion of the bowel the albuminous material is usually insufficient to produce an excess of indol. In the new-born child there is no indican in the urine, since the bowel contains no putrefactive bacteria. In starvation the indican may result from the decomposition of the albumin of the intestinal secretions. Von Jaksch has found excessive indicanuria in ichorous pleurisy. It may also be found in chronic wasting diseases, as ulcer and cancer of the stomach, in chronic tuberculosis, especially with diarrhœa, and in acute diarrhœa and cholera. The rapid putrefaction of an excess of albuminates in the urine, as in chronic cystitis, may occur, and cause the precipitation of indigo in the urinary tract with the formation of an indigo calculus, as reported by Ord, or the passage of a blue urine, or the urine may become blue on exposure to the air. The recognition of excessive indicanuria is of especial importance in the diagnosis of intestinal obstruction, which is often simulated by a general or circumscribed peritonitis, in which there is no excess of indican. In diffuse suppurative peritonitis, although indican is increased, it is not as abundant as in intestinal obstruction. The greatest excess of indican is to be found in the latter affection when the small intestine is obstructed.

Melanuria.—The urine at times is of a dark color from the presence of a black pigment, melanin, which may be in solution or in a granular form. Such discoloration is present when the urine is first passed, or becomes apparent after the urine has been exposed to the air for some time. The latter condition is explained by the presence of melanogen, which when oxidized becomes transformed into melanin. In either case the discoloration may be intensified by the addition of oxidizing agents, as sulphuric or hydrochloric acid.

Melanuria has been found in patients with melanotic tumors, although such tumors may be present and melanuria be absent. It has also been found in emaciated persons. Its occurrence is suggestive of a melanotic cancer or sarcoma, especially in those cases in which other symptoms or signs favor such a diagnosis.

Alkaptonuria. Brenzkatechinuria. Hydrochinonuria.—Urine which when passed is of a normal color may become dark-colored, sometimes black, on standing, even when melanin or melanogen is absent, and may be alike discolored by the addition of caustic potash or soda. Boedeker gave the term alkapton to the substance producing this discoloration. Baumann ascribed a similar modification of color to brenzkatechin; while importance has been attached to still other chemical compounds which have been isolated from such pigmented urine. It is therefore probable that the so-called alkaptonuria may be due to a variety of substances.

Alkaptonuria has been more frequently found in children than in adults. It has been observed in two children of the same parents, and has continued throughout life in a person attaining sixty years of age. So far as is known, it produces no ill effects and has no diagnostic importance, except that it may cause the urine to react like that of diabetes when Heller's, Trommer's, and Fehling's tests are employed, although it does not respond to the fermentation test. The urine may be brownish when passed, becoming darker, or even black, on standing, from the presence of hydrochinone, due to poisoning by carbolic acid, salol, resorcin, and uva ursi. The appreciation of this fact is of importance, especially during the therapeutic administration of carbolic acid, as evidence of a beginning toxæmia.

Chyluria. Lipuria.—Fat may be present in the urine either in the molecular form or as fat-drops or solidified fat.

In chyluria, or galacturia, the urine has the opaque white homogeneous appearance of milk, and if blood also is present a pink color is produced. A cream-like layer may form on exposure to the air, and a fibrinous clot be present. The urine contains albumin, sometimes in considerable quantity, and on microscopical examination molecular fat, small fat-drops, leukocytes, and at times red blood-corpuscles, are to be found. If the milky urine is made alkaline and shaken with ether, the fat is dissolved and the urine appears clear.

Chyluria is considered to be due to the flow of lymph into the urinary tract, although the urine is free from sugar, which is always present in lymph, and the quantity of fat in a chylous urine is greater than that present in lymph. The occurrence of chyluria in regions near the tropics is occasioned by the presence in the lymph-vessels of a parasite (the filaria sanguinis hominis), which probably enters the body with drinking-water, and which is further considered in the chapter on animal parasites (page 368). It is likely that a non-parasitic variety of chyluria also exists, from the fact that this symptom occurs in persons who have never been near the tropics, and may last for years without other disturbance than painful micturition from the presence of clots in the bladder.

In *lipuria*, or *adiposuria*, the fat is present either as large or small oil-drops or as solidified fat resembling lard or tallow. The fat is usually liquefied while the urine is warm, but may become solidified as the urine cools. It may be sufficient to grease blotting-paper, or may be recognizable only by means of the microscope, the appearances being controlled in case of need by the solution of the fat-drops in ether or chloroform or by their black discoloration with osmic acid. The solidified fat when heated becomes liquefied.

The fat may directly enter the urinary tract or be eliminated from the blood: in the latter case a lipæmia (free fat in the blood) exists, which is produced in various ways. In animals where an excess of fat is introduced into the blood, it is eliminated as oil by the kidney, and it is possible, though not probable, that excessive quantities of oil taken into the human stomach may be in part excreted by the kidneys as oil. Minute quantities of fat may pass through the kidneys when the extensive crushing of fat-tissues, especially of bone-marrow, as in fracture, has taken place. Lipuria has been observed in saccharine diabetes and in cancer of the pancreas. Fat is also present in the urine as the result of its direct admission, either in consequence of a fatty degeneration of the renal epithelium, of pus in pyonephrosis, of tumors projecting into the renal pelvis, or from inflammatory destruction of the perinephric fat-tissue. In phosphorus poisoning, in chronic alcoholism, and in phthisis the presence of fat in the urine is attributable to the elimination of fat from the blood as well as to its formation in the kidney. Excessive quantities of fat in the urine offer suggestive evidence of pyonephrosis, perinephritis, diabetes, or cancer of the pancreas.

ALBUMINURIA, GLOBULINURIA, NUCLEOALBUMINURIA (MUCINURIA), ALBUMOSURIA, PEPTONURIA.

A variety of albuminous substances may occur in the urine. Those which receive especial consideration are serum-albumin, serum-globulin (paraglobulin), nucleoalbumin (mucin), albumose, and peptone. Most constant and most abundant is serum-albumin, with which serum-globulin is usually, but not, according to Von Jaksch, always, combined. The cases are rare in which the former is absent and the latter alone present, although Senator and Werner have observed instances of acute nephritis in which globulin was the sole albuminous constituent of the urine. Strictly speaking, the term albuminuria applies to the presence in the urine of serum-albumin alone.

Albuminuria.—In most instances the albuminous material of the blood transudes through the walls of the blood-vessels of the kidney either into Bowman's capsules or into the tubules. This renal albuminuria is to be distinguished from an admixture of albumin with the urine, which may take place anywhere in the urinary tract from the presence of blood, pus, semen, lymph, or fragments of tumors. In renal albuminuria the quantity of albumin is the same in all parts of the fluid. In albuminuria from other sources the percentage of albumin is in proportion to the quantity of blood, pus, lymph, or other albuminous fluid present, and is often higher at the bottom of the vessel in which the urine has been allowed to stand for some time than in the upper portion of the urine. The absence of casts and renal epithelium, and the presence of blood and pus-corpuscles, of spermatozoa and degenerated cells of uncertain origin, are opposed to the renal origin of the albuminuria. It is, however, to be remembered that renal albuminuria also may be present in cases in which albumin enters the urine from sources beyond the kidney. The diagnosis of renal albuminuria may require the exclusion of such sources of albumin as well as a characteristic sediment, or the presence of the symptoms and signs of disease of the kidney.

In testing for albumin it is desirable, especially when small quantities are concerned, that the urine should be dilute, to avoid an excessive precipitation of urates, and transparent. If simple filtration does not suffice to render the urine clear, a preliminary shaking with calcined magnesia is advantageous ; but if the specimen is opaque from the presence of fat, the latter may be removed by shaking with potash and ether. The tests in common use and sufficient to permit the recognition of one-fiftieth of one per cent. of albumin are nitric acid and heat, and potassium ferrocyanide.

Nitric Acid and Heat.—A glass capable of holding an ounce is to be filled one-fourth with urine. Nitric acid is to be slowly poured down the side of the tilted glass until it forms at the bottom a layer one-third of an inch thick. The presence of albumin is indicated by an opaque white line at the junction of the two fluids, and may be made more conspicuous by placing the glass against a dark background. A like result is obtained if the urine, by means of a pipette, is made to form a layer upon the upper surface of the acid. The denser and wider the albuminous ring, the larger the quantity of albumin. Globulin, albumose, urates, and resins, when present, are also precipitated. The urates form a zone farther removed from the acid, disappearing on the addition of heat, as does albumose ; resins are dissolved in ether. For the sake of control another specimen of urine is to be boiled in a test-tube. If a precipitate occurs, it may be due to albumin or phosphates. The addition of acetic or nitric acid causes the latter to be dissolved, while the former persists or becomes increased, although a minute trace of albumin may be dissolved in acetic or nitric acid. If a precipitate does not form until the specimen becomes cold, it is suggestive of albumose.

Potassium Ferrocyanide.—This test is extremely sensitive, and requires that the urine, when of high specific gravity, should be diluted. A test-tube is to be one-fourth filled with the transparent diluted urine, which is then to be acidified with acetic acid. If an opacity occurs, it may be due to nucleoalbumin (mucin), globulin, urates, or resins, and the specimen is then to be filtered and a few drops of a ten per cent. solution of potassium ferrocyanide are to be added. The slightest trace of albumin is shown either at once or in a few minutes by an opacity, while a larger quantity is indicated by a flocculent precipitate. Globulin and albumose, if present, are dissolved by heating the fluid.

An approximate test of the quantity of albumin present, sufficient for practical purposes, is furnished by boiling a specimen of urine and adding one-tenth of its volume of dilute nitric acid and allowing the sediment to settle. If, according to Klemperer, after several hours the fluid is simply opaque, a trace of albumin, one-hundredth per cent., is indicated. If the concave portion of the test-tube is filled with the sediment, five-hundredths per cent. are represented. If the sediment occupies one-tenth of the volume of urine, it approximates one-tenth per cent. ; if one-fourth of the volume of urine, twenty-five-hundredths per cent ; if one-third of the volume of urine, five-tenths per cent. ; and if one-half of the volume of urine, one per cent. If the coagulated albumin reaches the surface of the urine, it represents from two to three per cent. of albumin. In the nitric acid test the thickness of the layer of albumin in fractions of an inch is estimated by Hofmann and Ultzmann to correspond to the like fractions of one per cent. For example, a thickness of one-fourth of an inch indicates twenty-five-hundredths per cent. of albumin.

Esbach's albuminimeter offers a convenient means of approximately determining the quantity of albumin. In a graduated tube definite proportions of urine and the precipitating reagent are mixed and allowed to stand for twenty-four hours. According to the height of the sediment is the percentage of albumin determined. The chief value, however, of the Esbach tube is in the comparison of albuminous precipitates in successive examinations of the urine of the same patient.

A trace of albumin has so often been found, especially by means of delicate tests, in the urine of apparently healthy individuals, perhaps for a short time only and in consequence of especial exciting causes, that it is generally admitted that albuminuria is not necessarily an evidence of disease. A distinction is hence drawn between a *physiological* and a *pathological* albuminuria, although the border-line is not to be sharply defined. Among the causes of the former are pregnancy, in which albumin has been found in the urine in more than one-half of a considerable number of cases, the eating of large quantities of food, especially if abundantly albuminous, as eggs and cheese, extreme mental or muscular activity, and cold baths. Such an albuminuria, in virtue of its dependence upon physiological functions and its recurrence with the excessive use of these functions, and from the fact that it is of most frequent occurrence among the young, even the new-born, has also been called *functional*, *transitory*, *cyclic*, *intermittent*, or the *albuminuria of adolescence.* Da Costa calls attention to the frequent association of uric acid and oxaluria with this variety of albuminuria, and is inclined to attribute the latter to disturbances in the kidney caused by the excretion of uric acid or oxalates.

Pathological albuminuria may also occur as a transitory condition independent of any serious disease of the urinary apparatus. It is more or less constantly present in acute infectious diseases, especially typhoid fever, acute rheumatism, pneumonia, erysipelas, and tonsillitis, and disappears in convalescence. Such an albuminuria is called *febrile* or *infectious*, and is regarded by some, especially when casts are present, as evidence of the first stage of a nephritis. In chronic diseases, especially those associated with modifications in the composition of the blood, as anæmia, leukæmia, pseudo-leukæmia, purpura, and scurvy, albuminuria may be present, and is to be regarded as pathological. It may also occur in neurasthenia, migraine, chorea, epilepsy, delirium tremens, and cerebral hemorrhage, and is then classified as a *neurotic albuminuria.* A transitory pathological albuminuria may be present in acute diarrhœa, in incarcerated hernia, and in jaundice. The albuminuria associated with disease of the urinary apparatus is always to be regarded as pathological, and may be either transitory or permanent. The former occurs in localized lesions of the kidney, as hemorrhagic infarction, abscesses, or tumors of this organ; also in prolonged retention of urine, especially when caused by obstruction of the ureter from compression or a twist. It may be transitory or permanent according to the cause in active and passive congestions of the kidney and in the inflammations and degenerations of this organ, including the nephritis of pregnancy.

The diagnosis of a physiological albuminuria should be made with caution, since evidence of a nephritis may eventually appear, and the causes productive of the physiological variety may aggravate a pathological albuminuria. An albuminuria may be regarded as physiological when it is slight, and when its existence and duration are dependent upon the presence and persistence of the causes above mentioned. Frequent and periodical examinations of the urine are therefore necessary in doubtful cases before freedom from renal disease can be positively asserted. On the contrary, the albuminuria, whether slight or severe, transitory or persistent, is to be regarded as pathological when not attributable to physiological conditions or when associated with evidence of disease of the urinary apparatus. Its persistence offers important, although not essential, evidence of its pathological nature.

The prognosis of albuminuria, whether physiological or pathological, depends upon the cause and the possibility of the removal of the latter. If the albuminuria is persistent, its prognosis is more serious than if it is transitory or intermittent, since persistent albuminuria in the course of time not infrequently becomes associated with other evidence of disease of the kidney. The mere loss of albumin has no especial significance, since it may be excessive in acute nephritis and recovery take place, and is least in chronic fibrous nephritis, the prognosis of which is hopeless.

Globulinuria.—Globulin may be tested for by diluting a considerable quantity of urine with distilled water to a specific gravity of 1001 or 1002.

If globulin is present, a white precipitate forms on the addition of a few drops of dilute acetic acid.

Nucleoalbuminuria.—Of the other albuminous substances which may be found in the urine nucleoalbumin and albumose (hemialbumose or propeptone) may be mentioned. Nucleoalbumin is precipitated by acetic acid, and is insoluble in an excess of this reagent. Such a precipitate was formerly called mucin, and was considered to be a normal constituent of the urine, and to be increased in inflammation of the mucous membrane of the urinary tract. Recent investigations, however, show that two substances have been called mucin, the one true mucin (a glycoproteid which reduces copper) and the other nucleoalbumin. The former has never been found in the urine; but nucleoalbumin is constantly present, and is found in excess in catarrhal affections of the urinary tract. Nucleoalbumin is tested for by adding three times as much water to the quantity of urine to be examined, filtering, if necessary, and adding an excess of acetic acid to the fluid, when nucleoalbumin is precipitated.

Albumosuria. Peptonuria.—The recognition of albumose in the urine has become of importance of late years, since it requires to be differentiated from a group of albumoses whose presence in the urine has been designated peptonuria. Albumose or propeptone is considered to represent that stage of the products of the digestion of albuminous substances immediately preceding the ultimate result,—peptone. Kühne found that what had been called peptone represented a series of albumoses, and limited the term peptone to substances not precipitated by ammonium sulphate. The term albumosuria or propeptonuria as now used designates the presence of albuminous substances soluble in heat, precipitated by cold and a concentrated solution of ammonium sulphate, and presenting the biuret reaction. A pure albumosuria may be suspected if nitric acid causes a precipitate in the urine which is dissolved by heat. Usually, however, albumosuria and albuminuria are combined, and the precipitate caused by nitric acid is only partially dissolved by heat and returns when the urine is cool. To determine the presence of albumose in such cases the urine must first be freed from albumin. The specimen to be examined, if concentrated, must be diluted with water, since albumose is insoluble in a concentrated saline solution. It is acidified with acetic acid. One-sixth of its volume of a concentrated solution of common salt is added, and the mixture boiled and filtered while hot. The presence of albumose is indicated by an opacity or precipitate as the filtrate becomes cool. This result also takes place when a concentrated solution of ammonium sulphate is added. Such albumosuria or propeptonuria has been found in osteomalacia, bone tumors, scarlet fever, measles, pemphigus, and urticaria, and we have observed it in a case of myxœdema. Senator has repeatedly found albumosuria to precede and follow albuminuria, also to alternate with it.

According to Stadelmann's researches, peptone never occurs in fresh urine, although it is possible for it to be present in putrid albuminous urine, since peptone may arise from the action of putrefactive organisms on albumin. What is called peptonuria is apparently an albumosuria, due to the presence in the urine of the albumoses to be detected by Salkowski's test. Such albumoses arise from the destruction of cells, especially pus-corpuscles, and, according to Senator, their appearance in the urine is indicative of the near approach of the crisis of pneumonia, and of the suppurative nature of an empyema, peritonitis, or meningitis. Salkowski's test is to be applied as follows. Filter the boiled urine in which albumin is precipitated and albumose is dissolved. Fifty cubic centimetres of this urine are acidified with five cubic centimetres of hydrochloric acid, and a precipitate obtained with phosphomolybdic acid. The precipitate, heated for a few minutes, adheres to the bottom of the dish. The free fluid is poured off, and the precipitate, after having been washed with distilled water, is dissolved in five-tenths of a cubic centimetre of a dilute solution of caustic soda (specific gravity 1.16). A deep blue color is formed which becomes yellow when heat is applied, the more rapidly if a few more drops of the solution of caustic soda are added. After the fluid, poured into a test-tube, is cold, add, drop by drop, with shaking, a one per cent. or two per cent. solution of sulphate of copper. If "peptone" be present the fluid will become red.

FIBRINURIA.

Fibrin is rarely found in the urine except as a clot in hæmaturia and chyluria. The clots are usually present when the urine is passed, but may first appear after the urine has been voided. In severe inflammation of the urinary tract the passage of the clots may be associated with renal colic and obstructed urination. Von Jaksch records the occurrence of spiral membranes composed of fibrin and nucleoalbumin (mucin) as evidence of a membranous ureteritis analogous to membranous colitis.

LITHURIA. URATURIA. URICACIDURIA.

English writers, in particular, have attached a considerable degree of importance to the presence in the urine of the brick-dust—lateritious—sediment, consisting chiefly of sodium, potassium, and ammonium urates, and of uric acid. The precipitation of these constituents of the urine occurs under normal conditions in an acid concentrated urine, especially in cold weather. The pigmented amorphous urates are easily dissolved by warming the urine, while the reddish crystals of uric acid suggest grains of pepper or sand, and sufficiently characteristic shapes are seen with the microscope.

The frequent appearance of the brick-dust sediment in acute inflammatory affections with profuse elimination of liquid from the skin or intestine, as in rheumatic fever, pneumonia, and gastro-enteritis, is well known. Although an increased production of uric acid and urates is recognized as taking place in fever, it is not essential in the production of lithuria, since the latter is often found in healthy and vigorous individuals. It is recognized that in gout an increased elimination of uric acid at times takes place, and that a deposition of urates takes place in various parts of the body, especially in the joints, although at the time of the deposition the excretion of uric acid may be diminished. The discovery of uric acid in the blood and lymph in such cases has led to the consideration of gout as the extreme illustration of a uric acid diathesis. In leukæmia there is also an increased elimination of uric acid, but with the exception of these two diseases it is doubtful whether there are any in which a persistent increase in the elimination of uric acid occurs.

The association of various disturbances of digestion with symptoms of functional nervous disturbance, as easily induced exhaustion, irritability, or depression, and headaches, with a brick-dust sediment, sand, or gravel, was attributed by Murchison to functional disorders of the liver resulting in the production of a disease, lithæmia, the conspicuous symptom of which was the lithuria or uraturia. Inasmuch as similar symptoms occur in the absence of the brick-dust sediment, even in alkaline urine, either with the deposition of phosphates, phosphaturia, or with the abundant presence of calcium oxalate in the urine, oxaluria, and as the brick-dust sediment takes place under various physiological conditions, it is obvious that this sediment is not proof of an increased formation of uric acid or of a uric acid diathesis. There is no proof that lithuria is connected with disturbances in the function of the liver, and the use of the term lithæmia should not imply that the existing disturbances are in any way attributable to an excess of uric acid.

OXALURIA.

Crystals of calcium oxalate are to be found in the urine of healthy persons as well as in that of the sick. They may form in the urinary tract, or first appear after the urine has been voided, from the progressive decomposition of acid sodium phosphate, which is the most important agent in holding them in solution, and are easily to be recognized as the familiar octahedral or dumb-bell crystals. They are present only in acid urine, and Fürbringer has shown that the number of crystals seen with the microscope does not indicate the quantity of oxalic acid present. Oxaluria becomes increased when certain articles of food, especially tomatoes and rhubarb, are taken. Its presence may alternate in the same case with that of uric acid and the urates in the urine. Oxaluria is of frequent occurrence in saccharine diabetes, in which it may be followed by glycosuria. Its association with jaundice, spermatorrhœa, digestive disturbances, and neurasthenia is to be recognized.

As in lithæmia, the combination of digestive disturbances, especially flatulence, with mental and bodily fatigue on slight exertion, listlessness, headache, wakefulness, mental depression, backache, and crystals of calcic oxalate in the urine, is regarded as characteristic of an oxalic acid diathesis and classified as oxaluria. As has been stated concerning lithæmia, the origin of oxalic acid in the body is obscure. The methods of determining its absolute increase belong rather to the laboratory than to the bedside, and the symptoms regarded as due to its presence in excess occur independently of an oxaluria and of any special chemical modification in the composition of the urine.

The escape of excessive quantities of crystals of calcic oxalate may mechanically irritate the urinary passages and produce pain in the ureter or the urethra. An occasional red blood-corpuscle may be found in the urine under such circumstances.

The relation of oxalates to conditions of the nervous system is, from a physiological and a pathological stand-point, extremely obscure. In ordinary melancholia there is commonly a severe oxaluria, which is with difficulty remedied, and in which the mental symptoms do not improve with lessening or disappearance of the oxalic acid. On the other hand, there are cases especially occurring in young men in which malaise and great repugnance to mental and bodily exercise are associated with marked depression of spirits, without other pronounced symptoms except the presence of oxalates in the urine, and in which nitrohydrochloric acid acts as a specific, causing the various symptoms to disappear *pari passu* with the oxalates. Exercise, regulation of the diet, and the avoidance of sweets and indigestible materials may be of service in these cases; but nitrohydrochloric acid is the main element in effecting a cure. From five to seven drops of the freshly prepared acid should be given three times a day.

PHOSPHATURIA.

Sodium and potassium phosphates and calcium and magnesium phosphates are precipitated in a neutral or alkaline urine, and the ammonio-magnesic or triple phosphates are added if an ammoniacal fermentation takes place. This precipitation may occur within the body if alkaline salts are abundant in the food or drink or are used as medicines. The appearance of the crystals is sufficiently characteristic for diagnostic purposes, and the addition of an acid causes them to disappear. The occurrence of a phosphatic sediment is no evidence of an absolute increase of the phosphoric acid in the urine, to which alone the term phosphaturia should be applied, and which, like the absolute increase of oxalic acid, must be determined in the laboratory. An increased elimination of phosphates takes place in meningitis, in epileptic convulsions, in saccharine diabetes, and in leukæmia. In diabetes, phosphaturia, like oxaluria, may alternate with glycosuria. Tessier has applied the term *phosphatic diabetes* to a class of cases in which phosphaturia and polyuria are combined with loss of flesh and strength.

Persistent alkalinity of the urine with a phosphatic sediment is frequently observed in persons suffering from indigestion, especially the variety due to a deficiency of hydrochloric acid. Such patients complain of mental and physical weakness, backache, and sexual irregularities, and are conspicuously neurasthenic or hypochondriacal. They frequently regard the sediment as evidence of inflammation of the bladder or of spermatorrhœa, and, through ignorance of its nature, often become the victims of charlatans.

In certain cases of neurasthenia there is an extraordinary increase in the elimination of the phosphates, but we know of no specific medication especially adapted to such cases. Sexual intercourse should be altogether or in great part interdicted. When there is danger of the formation of phosphatic calculi, boric or benzoic acid should be administered, as urine deposits phosphates much less readily when acid than when alkaline.

CYSTINURIA.

Cystin rarely occurs in the urine, and is seen as hexagonal plates, to be recognized by the microscope. Cystinuria is found more often in young persons, at times in several members of the same family, and has been observed by Ebstein combined with albuminuria, both of which simultaneously disappeared in acute rheumatism. Its presence is inconstant, and the quantity eliminated is variable. The clinical importance of this condition is due to the fact that a characteristic calculus may be formed, to be recognized by its smooth, yellow, translucent appearance and crystalline fracture. The presence of sulphur in cystin and in the taurin of the bile, and the occurrence of a case of cystinuria with chronic biliary obstruction, have suggested that cystinuria may be due to disturbance of the function of the liver, a view disproved by the observation that in cystinuria there is an increased elimination of sulphur in the bile. Baumann and Udranszky have found diamines (cadaverine, putrescine), which, according to Brieger, are due to specific bacteria, in the urine and in the fæces in cystinuria, but not in normal fæces: hence they suggest that diaminuria and cystinuria may be due to the same cause.

GLYCOSURIA. MELITURIA.

These terms are applied to the presence of grape sugar, glucose, in the urine. Other varieties of sugar, as milk sugar, muscle sugar, fruit sugar, and pentose rarely, may be found. Baumann states that traces of sugar are present in normal urine, and a distinction is hence to be drawn, as in albuminuria, between a *physiological* and a *pathological glycosuria.* Usually the quantity of sugar found under physiological conditions is so small that its presence is to be recognized only by the most delicate tests. A physiological glycosuria becomes important only when considerable quantities of sugar recognizable by ordinary tests are found in the urine. It may result from the excessive use of sugar, three or four ounces daily, and ceases with the removal of the cause, *alimentary glycosuria.* *Lacto-*

suria, milk sugar in the urine, has been repeatedly found in pregnancy, especially after childbirth, when abundant milk is retained in the breast. Such urine does not react to the fermentation-test, although polarizing light to the right.

Pathological glycosuria may be divided into the transitory and permanent varieties. The former occurs as a rare complication of the disease in which it is present, and has been observed in cerebral hemorrhage, in cerebro-spinal meningitis, and in disease of the brain in the vicinity of the fourth ventricle, in injuries to the head, according to Higgins and Ogden, in infectious diseases, as typhoid and malarial fevers, cholera, diphtheria, influenza, and scarlet fever, in gout, and in diseases of the heart, lungs, and liver, especially in fibrous hepatitis. It has also been observed in poisoning from opium, chloral, and carbonic oxide gas, and may be experimentally produced in a variety of ways,—*e.g.*, by puncture of the fourth ventricle, by intravenous injection of salt solutions, or by poisoning with amyl nitrite or phloridzin. Permanent glycosuria is the essential characteristic of diabetes mellitus, in which disease it may be found in the absence of other symptoms, the quantity of sugar becoming gradually increased on a saccharine and starchy diet.

The tests in common use for the detection of glucose in the urine are those of Moore, Trommer, Fehling, and the fermentation-test. Albumin is first to be sought for, and, if present, eliminated by boiling and filtration.

Moore's Test.—Mix with the suspected urine in a test-tube one-third of its volume of liquor potassæ. A precipitate of earthy phosphates takes place. Heat the upper portion of the fluid, which becomes of a dark-brown color if sugar is present; in normal urine a light-brown color, attributed to the presence of "mucin," frequently develops. On the addition of a drop or two of strong sulphuric acid to the brown solution when cold, the odor of burned sugar is produced if the brown color is due to the presence of glucose. This test is only of relative value, since a brown color is often produced in the absence of glucose, and small quantities of the latter may escape recognition.

Trommer's Test.—Mix with the suspected urine in a test-tube one-third of its volume of liquor potassæ. Add a few drops of a solution of cupric sulphate (1 to 10). A bluish-green, flocculent precipitate of hydrated copper oxide forms. If sugar is present the precipitate is dissolved on shaking, and the fluid becomes dark blue. Drops of the copper solution are to be added as long as the precipitate is dissolved. The mixture is then heated, and the rapid formation of a reddish-yellow precipitate of cuprous oxide before the boiling point is reached indicates the presence of glucose. This test is of no value unless more than one-half of one per cent. of glucose is present in the urine. The solution of cupric sulphate should be added drop by drop, since an excess of this reagent may prevent its reduction. The cupric oxide may also be reduced in concentrated urine containing a large quantity of uric acid and urates, in the presence of creatinine, alkapton, brenzkatechin, hydrochinone, mucin, or bilirubin, and after the use of benzoic acid, salicylic acid, glycerin, chloral, phenacetin, morphine, or chloroform. Such sources of error may be overcome by diluting the concentrated urine with three or four times its volume of water; by shaking the test-tube containing the pigmented urine in which a pinch of animal charcoal has been added, filtering the specimen, and testing the filtrate; and by avoiding the use of the above-mentioned drugs.

Fehling's Test.—This test also depends upon the reduction of copper which is dissolved in Rochelle salt. Fehling's solution is best made by the immediate mixture of solutions of its ingredients separately preserved. For this purpose 34.64 grammes of cupric sulphate are to be dissolved in 500 cubic centimetres of water and kept in a bottle. In another bottle is to be preserved a solution of 175 grammes of Rochelle salt in 100 cubic centimetres of caustic soda, specific gravity 1.34, and dissolved in 500 cubic centimetres of water. A mixture of equal quantities of these fluids is Fehling's solution. A drachm of this solution is to be poured into a test-tube, and two or three times as much water should be added if the presence of a high specific gravity of the urine does not suggest a considerable quantity of glucose. The solution is boiled to test its efficiency, since in old solutions of Rochelle salt products of decomposition may arise which reduce the copper. If the absence of such products is thus shown, an equal quantity of urine is added, the mixture heated, and a reddish-yellow precipitate of cuprous oxide takes place when glucose is present. The percentage of glucose may also be determined by the use of Fehling's solution, 10 cubic centimetres of which correspond to 0.05 gramme of glucose, and the necessary apparatus, solutions, and directions combined for this purpose may be bought.

Fermentation-Test.—Sources of error arising from the reduction of copper by other agents than glucose may be avoided by the fermentation of the urine, which has a further advantage in permitting a ready approximate determination of the percentage of glucose when it is above one-tenth per cent. This constituent alone in the urine causes alcoholic fermentation. About four ounces of urine are poured into a bottle, half a cake of finely subdivided yeast is added, and the mixture is placed in a room of ordinary temperature until the fermentation caused by the glucose is ended, usually in the course of twenty-four hours. The specific gravity of the fluid is then compared with that of another sample of the urine which has been set aside and exposed to the same circumstances as the fermented urine, excepting the addition of yeast. According to Roberts, each degree of density lost corresponds to one grain of glucose in the ounce of urine,—*i.e.*, 0.219 per cent. Hence the percentage of sugar present is ascertained by multiplying the difference between the specific gravities by 0.219. The simplicity, convenience, and practical accuracy of this test make it generally applicable.

ACETONURIA. DIACETICACIDURIA.

Although traces of acetone may occur in normal urine, its presence in considerable quantities is the result of pathological conditions. The accurate determination of the quantity requires distillation of the urine. For practical purposes when the question concerns an increased quantity Legal's test is sufficient. A few drops of a moderately concentrated, freshly prepared solution of sodium nitroprusside and a small quantity of the official solution of potash or soda are added to ten cubic centimetres of urine. If acetone is present a red color is produced, which quickly fades and becomes violet or purple if acetic acid is added. Acetone results from the destruction of the albumin of the food or of that of the tissues. Its excessive formation takes place in fevers, in diabetes mellitus, in wasting diseases, in peritonitis, in periodical vomiting, in certain nervous affections, as hysteria, convulsions, coma, cerebral plumbism, gastric crises of tabes, in certain auto-intoxications and chronic opium poisoning, and in connection with certain cases of cancer. It is not known that acetone produces injurious effects, although Cantani considers that mild or severe disturbances of the nervous system may result from excessive acetone in the blood. Although its occurrence in diabetes takes place in the advanced stage of this affection, the prognosis is unaffected by its presence. Its transformation into diacetic acid is associated with the symptoms of toxæmia.

Diacetic acid, aceto-acetic acid, does not occur in normal urine, but, when found, is associated with the presence of acetone. It is to be recognized by the cautious addition of a moderately concentrated solution of ferric chloride. If phosphates are precipitated, the urine should be filtered and the iron added anew. If the urine becomes of a claret color which fades or disappears when the urine is boiled, the presence of diacetic acid is indicated. Diaceturia occurs in fevers, in diabetes, and in certain cases of infantile convulsions and wasting affections. In adults it is of prognostic importance, since the patients concerned, whether febrile or diabetic, not infrequently die comatose, the symptoms resembling those seen in diabetic coma: in children it has no especial significance.

LIPACIDURIA.

Traces of volatile fatty acids, acetic, butyric, formic, and propionic acids, are to be found in normal urine. It is probable that they, like acetone, are due to albuminous oxidation. Their increased presence in fever, fibrous hepatitis, cancer of the liver, gall-stones, and diabetes mellitus is explained by the occurrence of an extensive albuminous destruction in these affections. It is possible that an excess of these acids may be formed in the intestine in abnormal digestion and be eliminated with the urine. Like acetonuria, lipaciduria has no clinical significance.

HYDROTHIONURIA.

Brief mention may be made of the rare occurrence in the urine of sulphuretted hydrogen. Its presence is usually indicative of an abnormal communication between the intestine and the urinary tract, but, the possibility of auto-intoxication by the absorption of sulphuretted hydrogen from the intestine being recognized, it is conceivable that this gas may be excreted with the constituents of the urine, or that it may enter the bladder from the intestine by diffusion through the intervening tissues. The significance of hydrothionuria is at present merely suggestive.

CASTS.

Among the most important of the morphological constituents of the urine are casts of the urinary tubules. They differ in composition and appearance, and are always evidence of a pathological condition, although their diagnostic and their prognostic significance vary within wide limits. They are present in the urine in various quantities, are usually, although not necessarily, associated with albuminuria, and are most easily found, when few, by permitting the urine to stand for several hours in a conical glass receptacle, that the casts may settle in a limited place. The use of the centrifuge permits the speedy isolation of casts even when few in number. The rapid growth of bacteria in the urine makes it desirable, if the specimen is allowed to stand for many hours, to add an antiseptic, as thymol, which neither coagulates albumin nor precipitates urinary salts. Hyaline casts are made more conspicuous when stained, and for this purpose aniline colors, carmine, hæmatoxylin, or an aqueous solution of iodine may be employed.

The basis of all casts, with the exception of certain epithelial and blood casts, is a hyaline material, which is probably of albuminous origin, although, according to Knoll, it is not identical with any known form of albumin. It is probably derived from the albumin of the blood, and is either passed through the Malpighian capillaries or is secreted by the renal epithelium, or, being transuded into the latter, is then transformed into the cast by the degeneration, death, and fusion of the epithelial cells. The combination of various materials with this hyaline substance gives rise to modifications in the appearance of the cast, to which special terms are given.

Hyaline casts are long or short, broad or narrow, homogeneous, pale bodies, in the form of cylinders, usually rounded at one end, and often to be recognized only by careful adjustment of the reflecting mirror or by staining. Thomas applies the term *cylindroids* to hyaline casts, which are unusually long, flattened, often streaked, bent, and serpentine, one end terminating in a point, the other rounded or translucent, broken, sometimes bifurcated or fused with the typical hyaline cast, with which, according to Rovida, it is identical in chemical composition. A distinction between the cylindroid and the "*mucous*" *cast* is not apparent.

Waxy casts, which also present a homogeneous appearance, are usually short and broad, yellow and glistening. They sometimes give the amyloid reaction, although their presence is in no way suggestive of amyloid disease of the kidney. It is probable that they are hyaline casts which have been long retained in the renal tubules. *Epithelial casts* occur in two varieties: the one, rare, represents the desquamation of a portion of the coherent epithelial lining of the straight tubules; the other, the ordinary epithelial cast, is formed by the agglutination of desquamated renal epithelium or of leukocytes within or upon the surface of the hyaline cast. *Granular casts* occur when granules of various origin are united by the hyaline material, and a subdivision is made between fine granular and coarse granular casts. Such granules may be albuminoid, resulting from a transformation of portions of the hyaline cast or from the disintegration of renal epithelium, leukocytes, or red blood-corpuscles, from a fatty degeneration of renal epithelium and leukocytes, from the deposition of urinary salts, sometimes as crystals (calcic oxalate), or from bacteria. *Fatty casts* are so designated when the granules are sufficiently large to present the optical appearances of fat. The fat-drops result from the degeneration of cells, and may be present within the adherent cell, or may appear as irregularly grouped, isolated drops of various size. Acicular crystals may be formed from the fat and project as spines from the surface of the cast. *Blood-casts* occur in two varieties: the one is due to the clotting of blood in the renal tubules; the other to the combination of red blood-corpuscles and the hyaline basis of the various casts. The red blood-corpuscles may be colored or decolorized, and homogeneous brown casts are at times to be found, the color of which is probably blood-pigment. In hæmoglobinuria colored casts are present, apparently due in considerable part to agglutinated fragments of hæmoglobin.

In general, hyaline casts are associated with albuminuria, although they may be found when albumin is absent, as in cholera, jaundice, and poisoning with sulphuric acid or alcohol. They may be temporarily absent when albuminuria is present, as in fibrous nephritis and amyloid degeneration, or permanently so in chyluria. Although they are indicative of a pathological condition of the kidney, they do not necessarily indicate a diseased state of the individual, since they disappear with a transitory albuminuria, and, as F. C. Shattuck has shown, they may be present for years, even combined with albuminuria, in persons in whom there is no other evidence of renal disease or of irritation of the kidneys. They may be overlooked, or found only on prolonged search, or, according to Sehrwald, be digested by the presence of pepsin in acid urine. The cylindroids, like the hyaline casts, are not characteristic of disease of the kidneys, since they may be seen in normal urine as well as in that of passive congestion of the kidney, nephritis, and cystitis. The presence of fat-drops and granular corpuscles is indicative of a fatty degeneration of cells, and is especially significant of a chronic parenchymatous or diffuse nephritis. Red blood-corpuscles may be present in both acute and chronic nephritis as well as in renal hemorrhage. Bacterial casts in urine relatively free from bacteria are suggestive of an infectious pyelonephritis or septic embolism of the kidney, but bacteria growing in stale urine readily adhere to the surface of casts, and are then merely evidence of decomposition of the urine.

PYURIA.

The presence of pus-corpuscles in the urine produces an albuminuria, but the latter may be due to the pus-corpuscles alone or to a combined nephritis. A few pus-corpuscles in the urine may result from a nephritis, and many pus-corpuscles from sources outside the kidney may be associated with a nephritis. It is, therefore, important to ascertain to what extent the accompanying albuminuria may depend upon the presence of the pus-corpuscles. According to Salkowski and Leube, two per cent. of pus in the urine corresponds to one-tenth per cent. of albumin. The percentage of albumin alone does not determine the dependence of its origin upon pus, since moderate pyuria, two per cent. of pus, may be associated with considerable albumin in the urine. If on boiling the urine a moderate precipitate of albumin, one-twentieth to one-twenty-fifth of the volume of the urine, is formed, its exclusive origin from pus is to be inferred if several pus-corpuscles are found in each drop of the shaken urine. In two per cent. of pus with one-tenth per cent. of albumin there are ten to fifteen pus-corpuscles in each microscopical field. Fewer pus-corpuscles than these with this amount of albumin suggest a simultaneous nephritis. With one-fifth per cent. of pus there is merely a trace of albumin, and if pus-corpuscles are not present in every field chronic nephritis is to be suspected, and casts should be sought for by repeated examinations if necessary. Pyuria may be caused by urethritis, cystitis, pyelitis, tuberculosis, or abscess of the kidney, and by the evacuation into the urinary tract of a perinephric or peritonitic abscess or an abscess of the abdominal wall. The presence of epithelial casts and pus-corpuscles is suggestive of a pyelonephritis, while characteristic bacilli are indicative of renal tuberculosis.

URÆMIA.

General Symptomatology.—When the elimination of the urinary constituents is prevented for some time, their accumulation in the blood results in a toxæmia to which the term uræmia is applied. Chemical and experimental investigations have been unsuccessful in demonstrating that this toxæmia is due to any especial component of the urine. It results from prolonged interference with the normal secretion of urine, whether due to disease of the kidney or to obstruction to the outflow of urine. Although the symptoms of uræmia are usually preceded by a diminution in the quantity of urine, *oliguria*, perhaps terminating in complete suppression of this secretion, *anuria*, uræmic symptoms may be present and the quantity of urine be normal although its specific gravity is much diminished.

The disturbances from uræmia affect particularly the nervous system and the digestive apparatus, and according to the rapidity or slowness of their development a distinction is drawn between acute and chronic uræmia, although there is no sharply defined line of demarcation, and the violent symptoms of acute uræmia may develop in a patient who has hitherto shown only the milder symptoms of chronic uræmia. The nervous symptoms of acute uræmia are usually severe, may be sudden and unexpected, often intermittent, or may be preceded by those of chronic uræmia. In the latter case a mild headache becomes intense; wakefulness, restlessness, or depression becomes delirium, perhaps mania or melancholia. Irregular twitchings and feelings of exhaustion may be followed by convulsions of an epileptiform character, or coma (uræmic apoplexy) may occur, in which the patient dies or from which he may rally and remain for a while comparatively comfortable. Localized nervous disturbances, also, suddenly occur, especially affecting the motor nerves, either as muscular tremors or cramps or as pareses or paralyses. Hemiplegia at times appears, with or without aphasia, disturbances of hearing, either deafness or the appreciation of abnormal sounds, take place, and amaurosis is frequent. The frequently brief duration of these general and localized symptoms suggests as their probable explanation temporary disturbances of circulation in the areas concerned. The digestive disturbances characteristic of acute uræmia are manifested by persistent vomiting or diarrhœa. The irritation of the bowels is sometimes associated with tenesmus and bloody stools suggestive of dysentery, and in such case a diphtheritic inflammation of the colon or the ileum has been found after death.

The symptoms of chronic uræmia may be so mild and their development so gradual that they are often overlooked until the sudden outbreak of the acute manifestations. It is then learned that the patient has been disturbed by headaches or peripheral neuralgia without especial cause, or has been either restless and wakeful or fatigued and listless. There may have been slight muscular tremor or spasm, and obstinate itching of the skin is not infrequent. It is, as a rule, the digestive disturbances which cause the patient to seek for medical advice. These, in particular, are loss of appetite, nausea, and vomiting. The patient may complain of dryness of the mouth and difficulty of swallowing, while the breath may have an odor suggesting that of urine. The nausea and vomiting are frequently independent of the quality or the quantity of food taken, and often occur when the stomach is empty. The occurrence of diarrhœa without assignable cause is more often significant of acute than of chronic uræmia.

Disturbances of respiration are frequent in chronic nephritis, and are usually spoken of as *uræmic asthma*. The difficulty of respiration varies in character, at times being slight, although more or less constant and aggravated by exertion; again, there are paroxysms of dyspnœa without obvious exciting cause, resembling attacks of asthma. Finally, the respiration may present the Cheyne-Stokes character. Although the dyspnœa is spoken of as uræmic, it is questionable whether uræmia is the cause, since the other symptoms of uræmia are not necessarily associated. Its characteristics are those of a cardiac dyspnœa, and corresponding lesions of the heart and the blood-vessels are frequently found. In many cases œdema of the lungs acts as a physical cause of the disturbed respiration.

DIAGNOSIS.—An examination of the urine, when possible, may suffice to establish the diagnosis of uræmia. This condition may be suspected as the cause of the severe symptoms, if there is a history of oliguria and especially of anuria. It is to be remembered, however, that cerebral hemorrrhage is of frequent occurrence in chronic nephritis, and that a complicating organic lesion of the brain may serve as the cause of the paralysis in any disease of the kidney. The examination of the urine is especially important in those cases of possible acute uræmia in which a similar grouping of symptoms may occur in the absence of organic disease of the brain, as in epilepsy, alcoholic intoxication, and opium poisoning. Time also may be necessary for the formation of the diagnosis, since temporary albuminuria may follow an epileptic fit or a cerebral hemorrhage, and an alcoholic odor of the breath or a contracted pupil does not exclude the possibility that an excess of alcohol or of opium has been taken by a sufferer from nephritis. In the absence of an obvious cause for an unexpected attack of persistent vomiting and diarrhœa, the examination of the urine and the history of oliguria may show its uræmic character. The routine examination of the urine becomes important in all cases of headache and digestive disturbance of obscure origin, and may make the diagnosis clear in those cases in which mild delirium, cerebral torpor, or stupor with elevation of temperature might be regarded as indicative of a meningitis. Indeed, the possible manifestations of uræmia are so manifold, and often so obscure, that the urine of every patient suffering from chronic disease of not entirely patent nature should be carefully studied.

PROGNOSIS.—The significance of uræmic symptoms is dependent in part upon their cause and in part upon their nature. If the former is removable, as a calculus, the symptoms may permanently disappear. The occurrence of symptoms of acute uræmia is usually indicative of early death, although in rare instances a severe attack may be followed by several years of freedom. The milder symptoms of chronic uræmia may exist for years and the individual consider himself in fair health. In such persons, in particular, the occurrence of convulsions or coma usually points towards an early fatal termination. In the estimation of the significance of the symptoms of chronic uræmia valuable information is to be derived from the occasional measurement of the total quantity of urine passed in the twenty-four hours and the determination of the amount of urea eliminated. Although, as has been stated, the symptoms of uræmia are not dependent upon any especial urinary constituent, the determination of the total quantity of urea eliminated in the twenty-four hours affords a valuable test of the principal function of the kidney. Normally the daily elimination of urine is in the vicinity of three pints, or fifteen hundred cubic centimetres, and the amount of urea in the vicinity of six hundred grains, or forty grammes. For clinical purposes the estimation of the total amount of urea passed in the twenty-four hours is most easily accomplished by the hypobromite method, with the apparatus of either Doremus or Squibb.

RENAL DROPSY.

One of the most important symptoms of renal disease is dropsy, although it may be absent in severe disease of the kidneys, and when present may vary in degree within wide limits. It represents the result of the transudation of the liquid portion of the blood through the walls of the blood-vessels. Since such a transudation through the walls of normal vessels takes place to only a limited extent, Cohnheim considered that an increased porosity was caused by the disease of the kidney. According to him, a toxæmia results from the insufficient elimination of the products of tissue-metamorphosis, in consequence of which the nutrition of the vascular wall becomes disturbed. Senator maintains that the glomerular capillaries are diseased when dropsy exists, although they may be affected and dropsy be absent. Renal dropsy is most frequently found in certain varieties of acute nephritis, in chronic diffuse nephritis, and in amyloid degeneration. Its occurrence in other varieties of renal disease is mechanical, due to a weakening of the heart's action and a consequent passive congestion. It is usually first observed in the face, especially as a puffiness of the eyelids, and at the end of the day is more likely to be found about the ankles. Eventually the subcutaneous tissue throughout the body may become involved, and finally the serous cavities and the lungs. The degree of dropsy is often intimately connected with the secretion of urine, being greater when the latter is less, and *vice versa,* a change in relation frequently taking place at short intervals. As a rule, the dropsy is more considerable when the quantity of albumin is large. Extreme degrees of dropsy produce cracks in the skin, hydrothorax, hydropericardium, ascites, œdema of the lungs and larynx, and intra-cranial œdema. The skin is then in danger of becoming infected, disturbances of respiration and circulation result from the fluid in the lungs, pleuræ, pericardium, and peritoneum, while suffocation is threatened from œdema of the larynx. The intra-cranial œdema may be a mechanical cause of many of the symptoms of uræmia already mentioned.

ALTERATIONS OF THE HEART AND BLOOD-VESSELS.

Alterations of the circulatory apparatus in nephritis are of such frequency as to require especial consideration. The most important of these lesions is *hypertrophy of the heart*, in particular of the left ventricle, without valvular disease, and is so constantly present in fibrous nephritis as to be of diagnostic importance. Although the extreme degrees of hypertrophy of the left ventricle are found in the latter affection, lesser degrees may be present both in acute and in chronic diffuse nephritis. Senator has called attention to the fact that hypertrophy without dilatation is the rule in the atrophic form of fibrous nephritis, while hypertrophy with dilatation occurs in other varieties of nephritis.

The arterial change which is especially noteworthy is the thickening of the wall, especially of the smaller arteries, the arterio-sclerosis, to which Gull and Sutton first called attention under the term *arterio-capillary fibrosis*. All the coats are thickened, and the arteries in various parts of the body are affected. It has been suggested that the vascular changes were the cause of the cardiac hypertrophy, and that the nephritis occasioned both. On the contrary, Gull and Sutton regarded the disease of the kidney as dependent upon the vascular changes. The former view prevails at the present time, although not free from objection. A pulse of high tension is often met with in the early life of individuals who present later the evidence of nephritis, and Johnson advanced the view that in chronic nephritis the blood becomes charged with products of tissue-metamorphosis which should have been eliminated by the kidney. He assumed that such altered blood caused contraction of the smaller arteries and thus produced the hypertrophy. The objection to this theory is the fact that hypertrophy of the heart is most constant and extreme in fibrous atrophy of the kidney, in the genuine form of which the elimination of the products of tissue-metamorphosis in the urine is not diminished except in the later stages. Senator admits Johnson's explanation for the occurrence of cardiac hypertrophy in all forms of nephritis except for the atrophied kidney, the genuine form of which is probably caused by a toxic condition of the blood acting simultaneously upon the kidneys and the blood-vessels, resulting in hypertrophy. In the arterio-sclerotic atrophied kidney both the cardiac hypertrophy and the renal atrophy may be attributable to the sclerosis of the blood-vessels.

CONGESTION OF THE KIDNEY.

Active and passive congestions of the kidney are to be discriminated, the former representing the presence of an increased quantity of arterial blood in the kidney, the latter due to the presence of an excessive quantity of blood in the renal veins. Arterial congestion of the kidneys occurs as the result of the presence in the kidney of irritants, and is of especial significance in connection with the production of inflammation. Extirpation of one kidney usually causes arterial congestion of the other. An arterial congestion of the kidneys is assumed in diabetes in both the insipid and the glycosuric variety, and in affections of the nervous system in which polyuria occurs. Its temporary occurrence is probable after exposure to cold, or when excessive quantities of fluid are taken into the stomach and largely eliminated by the kidneys. The effects of an irritative or inflammatory arterial congestion of the kidney are described in the article on nephritis. The immediate effect of the removal of one kidney upon the function of that remaining is a diminution in the flow of urine, which may contain albumin and red blood-corpuscles. Other causes of arterial congestion produce an increased flow of urine in which neither blood nor albumin is necessarily present.

Passive Congestion.

ETIOLOGY.—The causes of passive congestion of the kidney are either general or local. The former are to be found in diseases of the heart, lungs, and pleura, obstructing the flow of blood through these organs. The latter are conditions obstructing the flow of blood through the inferior vena cava or through one or both renal veins, whether from tumors, thrombi, or cicatricial stenoses in the vicinity, or from tumors or fluid in the abdominal cavity. Thrombosis of the inferior vena cava may also produce passive congestion of the kidneys. The effect of these causes is limited by the establishment of an efficient collateral venous circulation and by the heart's action. If the former occurs there may be little or no disturbance, but if, as usual, there is an insufficient compensatory circulation and the heart's action is weakened, the effect is essentially the same as when disease of the heart or of the lungs acts as the cause of the venous congestion.

MOBBID ANATOMY.—Long-continued obstruction to the outflow of venous blood from the kidneys produces a moderate diminution in the size of the organ following a previous state of enlargement, increased density, and a bluish-gray color, to which the term cyanotic induration is applied. The longer the obstruction is continued the more likely is the atrophied kidney to become pale, and to have the capsule adherent in places, with some of the Malpighian bodies obliterated, a condition representing a slight degree of granular atrophy. On microscopical examination casts and blood-pigment are found, also fat-drops in small quantity in the epithelium of the convoluted tubes. If thrombosis of the renal vein is a cause of the congestion, the kidney becomes greatly enlarged and is engorged with blood, and the microscope shows a necrosis of the epithelium.

SYMPTOMS.—The disturbances arising from the obstructed circulation in the kidney are so overshadowed by those produced elsewhere that the renal condition can be diagnosticated only by the examination of the urine. This secretion is acid, diminished in quantity, high-colored, its specific gravity in the vicinity of 1025, and there is a small quantity, usually less than one-eighth per cent., of albumin. A few hyaline casts and occasional red blood-corpuscles and leukocytes are to be found. A brick-dust sediment readily forms when the urine is allowed to stand.

disappearing when liquor potassæ is added or the urine is heated. The formation of the sediment is due to the concentration of the urine, and not to an increase of urea and urates. In the stage of extreme atrophy the specific gravity may be as low as 1010.

DIAGNOSIS.—Passive congestion of the kidney is to be inferred from the evidence of the presence of the above-mentioned causes, and is to be diagnosticated by the recognition of the described characteristics of the urine.

PROGNOSIS.—The prognosis depends upon the cause, which usually permits merely temporary improvement, and the treatment is that of the basal condition.

THROMBOSIS AND EMBOLISM.

The occurrence of thrombosis of the renal vein has already been mentioned in connection with passive congestion of the kidney. Thrombosis of the renal artery is rare, while embolism is of sufficient frequency to be of practical importance.

ETIOLOGY.—The emboli, as a rule, arise in the left ventricle or in the aorta from parietal thrombi, and produce mechanical disturbances, to which infectious results are added if the emboli contain bacteria.

MORBID ANATOMY.—The extent of the mechanical disturbances resulting from embolism of the renal artery is dependent upon the size of the vessel obstructed. An ischæmic necrosis occurs in the region supplied by the obstructed vessel, appearing as an irregular mass of an opaque grayish-white or reddish-gray color surrounded by a dark-red zone. The tubular epithelium is in a condition of necrosis. Fat-drops eventually make their appearance, and the dead portion of the kidney becomes absorbed, being replaced by a scar adherent to the capsule of the kidney and often containing blood-pigment. If pyogenic bacteria are present in the embolus, an abscess results.

SYMPTOMS.—Embolism of the renal artery has no characteristic symptoms, although in some cases sudden and unexpected pain in the region of the kidney has occurred, followed by the presence of blood in the urine.

DIAGNOSIS.—The diagnosis is based upon the concurrence of the above symptoms, or upon the occurrence of sudden hæmaturia alone in the urine of a person suffering from disease of the heart or of the aorta in which embolism is likely to take place.

TREATMENT.—The treatment must be largely symptomatic, having for its special object lessening of the irritation of the kidney. Absolute rest in bed, uniform temperature, warm clothing, and careful nursing should be insisted upon; the food should be restricted to milk, with, in feeble cases, eggs and small amounts of farinaceous food. Any diuretics used should be of the mildest character, and hæmaturia, if it occurs, should not be treated unless severe.

INFLAMMATION OF THE KIDNEY. BRIGHT'S DISEASE OF THE KIDNEY.

The study of inflammation of the kidney based upon the advances in histology and experimental pathology has resulted in such an addition to our knowledge that "Bright's disease" and "Bright's kidney" no longer represent a definite picture, but varieties of acute and chronic inflammation of the kidney are recognized distinct in etiology, anatomical changes, and symptoms. The combination of albuminuria and dropsy is not essential to all forms of nephritis, and the atrophied kidney of chronic Bright's disease has no necessary relation to the common varieties of acute nephritis. The lack of agreement as to what among the affections of the kidney shall be called Bright's disease makes it desirable to adhere as far as possible to an anatomical classification of inflammations of the kidney. Such a classification alone leads to distinctions of little clinical value, since the alterations of the kidney occurring in nephritis affect in various degrees the parenchyma, interstitial tissue, and blood-vessels of the kidney. Practically important is the arbitrary distinction to be drawn between acute and chronic nephritis, according as the symptoms have existed during a few or during several months. If of less than six months' duration, the disease is regarded as acute nephritis; if of more than six months' duration, the nephritis is called chronic.

ACUTE NEPHRITIS. ACUTE BRIGHT'S DISEASE.

SYNONYMES.—Acute catarrhal nephritis; Acute desquamative nephritis; Acute parenchymatous nephritis; Acute croupous nephritis; Acute diffuse nephritis; Glomerular nephritis; Glomerulo-capsular nephritis.

ETIOLOGY.—Infection and toxæmia, the former being the more common, are the principal causes of acute nephritis. The infectious varieties result from both micro-organisms and their products, and are those in which the symptoms characteristic of the infectious disease precede or accompany those of the nephritis. This group includes all the infectious diseases, although the severity, symptoms, and lesions of the nephritis vary within wide limits. The chief importance in etiology is to be attached to scarlet fever, diphtheria, infectious sore throat, cholera, typhoid fever, small-pox, erysipelas, cerebro-spinal meningitis, typhus fever, pernicious malarial fevers, dysentery, tuberculosis, pyæmia, and septicæmia. The nephritis in acute articular rheumatism and pneumonia is attributable to the infectious element prevailing in these diseases. In this series are also to be included many of the cases attributed to exposure to cold, also the instances following severe burns and those occurring in acute pemphigus. In the second or toxic group of causes are to be found cantharides, turpentine, copaiba, cubebs, the mineral acids, oxalic acid, carbolic acid, potassium nitrate, potassium chlorate, potassium chromate, potassium iodide, phosphorus, arsenic, and corrosive sublimate.

Pregnancy at times acts as a cause of nephritis, as well as of passive congestion of the kidney.

MORBID ANATOMY.—The appearances of the kidney vary according to the severity of the affection, its duration, and the localization of the changes. In mild cases the alterations may be so slight as to be easily overlooked. In the more severe cases the kidneys are increased in size and weight, flaccid (in septic cases) or brittle, the capsule easily separated, the surface of the kidney injected, perhaps dotted with punctate hemorrhages. On section the cortex is increased in volume, mottled from an increased opacity or from a yellow color of the convoluted tubes, and in the hemorrhagic cases speckled with blood. The glomeruli and blood-vessels are usually injected, but the former may project as translucent, pale-gray points (glomerular nephritis). The pyramids are of a reddish color, and may be streaked with opaque gray lines, due to a plugging of the tubular canals with cells escaping from the apices on pressure (catarrhal or desquamative nephritis), or to the presence of accumulations of bacteria (bacterial nephritis).

On microscopical examination the tubules are dilated in consequence of the swollen, granular, perhaps fatty or necrotic epithelium, and contain desquamated and disintegrated epithelium, red and white blood-corpuscles, and hyaline, granular, and epithelial casts, perhaps blood-casts. The glomeruli are enlarged, their nuclei increased; the capillary loops may be plugged with bacteria or hyaline clumps (glomerular nephritis). In other cases Bowman's capsules are thickened, and the lining epithelium is swollen and desquamated (glomerulo-capsular nephritis).

In addition to various degrees of the above-mentioned alterations, a coagulable exudation is often present in the interstitial tissue (croupous nephritis), or a cellular infiltration perhaps with red blood-corpuscles (diffuse nephritis).

SYMPTOMS.—In the milder varieties of acute nephritis there may be no symptoms calling attention to disease of the kidney, the examination of the urine in many of the infectious diseases alone giving evidence of the renal affection. The onset of the symptoms varies in accordance with the etiology. A chill, followed by moderate elevation of temperature, backache, nausea, and vomiting, and, in children, convulsions, may precede the dropsy, the most characteristic symptom, or the effusion may occur, as in scarlet fever, some time after the symptoms of the infectious disease which it complicates have disappeared.

The dropsy may follow the acute initial symptoms in the course of a day or two and rapidly progress, or may gradually appear in the course of convalescence from an acute infectious disease. It progresses the more rapidly the less the secretion of urine. It is usually first recognized in the face, especially as a puffiness of the eyelids, but rapidly involves the subcutaneous tissue of the lower extremities and the genitals, perhaps extending to the pleural cavities and the lungs. The frequency of dropsy in certain varieties of nephritis, especially in those due to scarlet fever, pregnancy, malaria, and to the unknown agents included under exposure to cold, and the rarity of its occurrence in the other varieties of infectious nephritis, are explained by Senator by the fact that in the former series the glomeruli are conspicuously altered, while in the latter the parenchyma is particularly changed. The conspicuous vascular disturbance in the kidney suggests that the cutaneous vessels also may be diseased, hence the dropsy.

The urine is usually lessened in quantity, although micturition may be frequent: such diminution may lie within normal limits, as in the nephritis of diphtheria and of many other infectious diseases, or there may be actual suppression. Frequently less than a pint is secreted during the twenty-four hours. It is high-colored and opaque when scanty, but when normal in quantity may show no alteration of color. If blood be present, the color will vary from a red or dirty red (smoky) color to a dark reddish-brown. The reaction is acid, and the specific gravity varies in accordance with the quantity eliminated, ranging from 1020 to 1035. The percentage of urea in the specimen examined may be high, but the total quantity eliminated in the twenty-four hours may be diminished to one-sixth the normal amount. Salkowski and Leube consider it probable that in glomerular nephritis there may be no considerable diminution in the elimination of urea. The quantity of albumin present usually varies from one-half to two per cent., although a lesser quantity may occur and extensive degeneration be present: further, the urine may contain no albumin in the early stage of acute nephritis, and the albumin may temporarily disappear, or the albuminuria may persist after the disappearance of the other symptoms. The sediment is, as a rule, abundant, and consists of uric acid crystals and urates, leukocytes, renal epithelium, fresh and decolorized red blood-corpuscles, and hyaline, granular, epithelial, and blood casts. Fatty degeneration of the epithelium is generally slight.

In the further unfavorable progress of acute nephritis, digestive disturbances, especially nausea and vomiting, persist, and are to be considered as the manifestations of a mild uræmia. The tongue is coated, and the bowels are usually constipated, although diarrhœa from uræmia may occur in the course of the disease. The patient loses flesh and strength, and pallor of the skin becomes conspicuous. The red blood-corpuscles are diminished, and hemorrhages from the nose and in the skin may take place. The uræmic disturbances of the nervous system are characterized by headache, restlessness, muscular twitchings or convulsions, wakefulness, delirium, stupor, or coma. The respiration is but little affected, unless uræmic dyspnœa occurs, or hydrothorax, pleurisy, or pneumonia takes place as a complication. The heart's action becomes intensified, and slight degrees of hypertrophy of the left ventricle may rapidly appear, indicated by an outward and downward displacement of the apex and an accentuation of the aortic second sound, but any considerable degree of cardiac hypertrophy is rare, especially in scarlatinal and diphtherial cases. Pericarditis sometimes develops, and the tension of the pulse is increased. At the outset micturition is often frequent and sometimes painful, though the total quantity is small, and anuria may occur, resulting fatally in the course of a few days. An increased flow of urine takes place as convalescence approaches, although the improvement thus indicated may be of temporary character, exacerbations and remissions with corresponding modifications in the flow of urine being frequent. The skin is not only pale, but often translucent, in the early stages, and dry in the later stages, of acute nephritis. Among the complications not already mentioned, retinitis, perhaps hemorrhagic, sometimes occurs, although less frequently than in chronic nephritis, and the inflammation of serous membranes, especially peritonitis, may take place without obvious local cause.

DIAGNOSIS.—The diagnosis of acute nephritis is often easily made by the examination of the urine alone, which should be repeatedly undertaken in all acute infectious diseases, especially in scarlatina and diphtheria, in pregnancy, and after exposure to toxic causes. Acute nephritis of less obvious origin is of more likely occurrence during epidemics of scarlet fever and diphtheria, and may be first suggested by the presence of dropsy, the renal nature of which is quickly disclosed by the urinary examination. The absence of any considerable degree of dropsy serves to differentiate a parenchymatous nephritis from the glomerular variety following scarlet fever, pregnancy, and malaria, and from the "idiopathic" variety of unknown origin. A glomerular nephritis is further suggested by the association of a high percentage of albumin with chiefly hyaline casts. The occurrence of hæmaturia and blood-casts is sufficient evidence of a hemorrhagic nephritis, while abundant epithelial cells and leukocytes in the sediment indicate a catarrhal or a desquamative nature of the affection. The association of bacterial casts and typhoidal symptoms is evidence of the septic or pyæmic nature of the process.

PROGNOSIS.—In accordance with the differences in the etiology and lesions of acute nephritis, and the variations in its course, the prognosis varies within wide limits. Its symptoms may disappear during the convalescence from the acute infectious disease which it complicates, or may persist for weeks or months, perhaps for years, in which case the acute nephritis is merely the early stage of a chronic nephritis. The prognosis, therefore, depends primarily upon the cause. It is usually favorable when the nephritis is due to infection, although in scarlatina and diphtheria the nephritis may be mild or severe according to the nature of the epidemic. Toxic varieties of nephritis also offer a favorable prognosis, provided the quantity of the poison taken has been small. The prognosis is uncertain in nephritis of doubtful or unknown origin, and in pregnancy. It becomes the more grave the longer the symptoms persist, since unfavorable symptoms may suddenly develop, due either to uræmia or to acute dilatation of the heart. The mortality is high among young children, and the prognosis is more serious in glomerular than in parenchymatous nephritis. An increased flow of urine and disappearance of the dropsy are favorable signs, while persistent oliguria, a low percentage of urea, and a high percentage of albumin are unfavorable signs. Anuria may prove fatal in the course of forty-eight hours, but often continues for a week without severe uræmic symptoms, and has lasted for eleven days in a patient with gout and renal colic and recovery taken place. Periodical examinations of the urine should be made for some time after apparent recovery, on account of the frequency with which chronic nephritis follows an acute attack.

TREATMENT.—The treatment of acute nephritis must vary with the cause and severity of the attack, but in all cases there are certain essential hygienic methods. The patient should always be put to bed and kept in a well-ventilated room at a uniform temperature day and night, and should wear an undershirt continuously. The slightest chilling of the surface of the body is very dangerous. The diet should be of the simplest form, and of such character as to throw the least possible strain upon the kidneys. If any solid food be allowed, it should be cracked wheat, oatmeal, or other farinaceous article. In most cases an absolute milk diet should be insisted upon. Large draughts of water should be taken at regular intervals; hot water is better usually than cold water, and ice-water should never be allowed. The total daily amount of liquid taken should be five or six quarts, if the stomach will bear it.

Almost invariably the patient should in the beginning of the attack be cupped over the kidney, and in many instances the cupping may be repeated with advantage. In sthenic cases following exposure or irritating poison, wet cups should be used: there should be no fear of abstracting blood very freely. In some cases venesection is justifiable. When the disease develops during an infectious fever dry cupping is often preferable to local blood-letting. During the course of the disease continuous counter-irritation over the kidney should be used, care being exercised not to employ cantharides, turpentine, or other substances which might be absorbed and increase the renal irritation.

The indications for medicinal treatment are—first, to soothe the kidneys and restore functional activity; second, to excite excretion through other organs and thus lessen the strain upon the kidneys.

Most of the vegetable diuretics are more or less irritant to the kidneys; the least so are probably digitalis and the double salicylate of theobromine and sodium, and even these should not be used in the early stages of acute Bright's disease. Potassium citrate (half an ounce to an ounce a day) may be given immediately, and may be in part substituted after two or three days by potassium bitartrate. When there is alarming lessening in the amount of renal secretion, the bitartrate, as the more active of the two, may be administered in small doses every two hours up to an ounce a day.

To lessen the strain upon the kidney and eliminate excrementitious material, purging with salines or with the elaterin pill (see formula 25) should be employed with a freedom proportionate to the strength of the patient. Once or twice in the twenty-four hours, according to the severity of the case, a very profuse perspiration should be produced, either by the vapor bath or by pilocarpine administered hypodermically or in combination with other diaphoretics. (See formula 7.) Very often the vapor or hot-water bath aided by a half dose of pilocarpine acts most happily. When there is fever, with arterial excitement, tincture of aconite is a very valuable remedy, which may be given continuously in such dose as materially to reduce the force and rate of the pulse and to keep the skin continually moist. In many cases a single full dose of aconite given once or twice with the pilocarpine acts very favorably.

During the later stages of an acute nephritis the milder vegetable diuretics are sometimes of service; and iron and other remedies useful in the chronic disorder may come into play before recovery is insured. During convalescence it is essential to keep the patient continuously from any chilling, and to avoid kidney strain by restriction to a diet largely farinaceous. Very frequently removal to a hot climate is most beneficial.

In acute suppression of urine the treatment is that of an acute congestion,—cupping, very active sweating, purging, saline diuretics; if the disease fail to yield, three five-grain doses of calomel may be given, an hour apart; the injection of a quart of aseptic, warm, normal saline solution (six-tenths per cent. of salt) into the buttock sometimes acts happily; in desperate cases flannels saturated with the tincture or poultices of the leaves of digitalis applied over the loins have brought relief, but are not free from danger, as E. F. Fannell has reported serious collapse from a single ounce of the tincture used externally.

CHRONIC NEPHRITIS. CHRONIC BRIGHT'S DISEASE.

Lack of agreement exists among medical writers as to the relation between the several chronic alterations of the kidney and the processes which give rise to them. It is recognized that an atrophied kidney may represent a terminal stage, "the third stage," of chronic diffuse nephritis, and that it may occur independently of the symptoms of this affection and probably with a different etiology. It is also recognized that an atrophied kidney may result from obstruction to its blood-supply dependent on chronic inflammatory changes in the wall of the renal arteries, producing stenosis. The anatomist recognizes the large white kidney of chronic diffuse nephritis and its contracted state, also the genuine atrophied kidney of chronic interstitial nephritis and an atrophied kidney the result of chronic endarteritis. The two latter forms of atrophied kidney have clinically little in common with the secondary atrophied kidney of chronic diffuse nephritis. The term "chronic Bright's disease" or "chronic nephritis," therefore, includes a series of widely differing anatomical changes which may represent successive stages of the same process, or may agree only in a final result, atrophied kidney. Clinical convenience is best served by distinguishing between a chronic diffuse nephritis and a chronic interstitial nephritis, since the symptoms permitting the diagnosis of the former widely differ from those attributable to the presence of the latter.

CHRONIC DIFFUSE NEPHRITIS. CHRONIC PARENCHYMATOUS NEPHRITIS.

SYNONYMES.—Bright's disease, second stage; Large white kidney; Fatty kidney.

ETIOLOGY.—Chronic diffuse nephritis occurs more often in men than in women, especially before middle life. It not infrequently represents the continuous progress of an acute nephritis, especially the scarlatinal and idiopathic (from unknown infection) varieties; also the nephritis of pregnancy. It may follow an acute attack although separated by a longer or shorter interval, and the etiology is then to be regarded as the same as that of the acute attack, either infectious or toxæmic. In other instances the onset is obscure, or the disease may arise as a fatty degeneration of the kidney in the course of chronic anæmia, especially in phthisis, cancer, gastric ulcer, and pernicious anæmia. The characteristic appearances of chronic diffuse nephritis at times are found associated with amyloid disease in the kidneys and elsewhere. It is, therefore, customary to assign an etiological importance to the general causes of amyloid degeneration, as syphilis, chronic suppurative arthritis and osteitis, and chronic tuberculosis of the lungs, intestine, and bones. The probable significance of these causes is strengthened by the occurrence of amyloid degeneration after the focal lesions of nephritis have become manifest.

MORBID ANATOMY.—The appearances vary according to the degree of the affection of the parenchyma or the interstitial tissue, the duration of the process, and, perhaps, the etiology. Especially characteristic is the large white kidney, which is increased in size and density, the capsule adherent in spots, the surface of a pale-gray color, mottled with white or yellow specks seen through a transparent film, and the stellulæ Verheynii often conspicuous. A mottled appearance is also seen on section, and is due to swelling, opacity, and fatty degeneration of the epithelium of the convoluted tubes. The Malpighian corpuscles are indistinct. Punctate hemorrhages are at times to be seen both on the surface and on the section of the kidney. On microscopical examination the tubules are dilated, the epithelium fatty, desquamated, and disintegrated. Leukocytes and hyaline and fatty casts are present in the canals, and the interstitial tissue is irregularly infiltrated with leukocytes. The glomeruli may show changes similar to those seen in acute nephritis. When anæmia is important in the etiology of chronic nephritis, the fatty changes predominate over the interstitial alterations.

The small white kidney represents the atrophic stage of the large white kidney, and is due to the shrinkage of the fibrous tissue and the absorption or excretion of the fat. The kidney is more nearly of the normal size, its density increased, the surface smooth or rough, more reddish-gray than white or yellow, although still spotted with the latter colors, the cortex diminished in volume. The microscopical examination shows a closer approximation of sound and atrophied Malpighian corpuscles, also irregular patches of fibrous tissue, in which the tubules have disappeared. According to the appearance of the surface a smooth atrophied kidney is contrasted with a granular atrophied kidney. Amyloid degeneration may be combined with chronic diffuse nephritis, in which case the pallor of the kidney is still more conspicuous. Hypertrophy of the left ventricle of the heart may be present, especially in the later stages. The appearances of a kidney in chronic diffuse nephritis may be modified by a focal limitation of the alterations involving larger or smaller portions of the kidney. Few or many scars throughout the kidney, or extensive atrophy of a limited portion, perhaps with compensatory hypertrophy of the remaining part, are to be seen.

SYMPTOMS.—As in acute nephritis, the characteristic symptoms are dropsy and the abnormal condition of the urine; the former may first suggest chronic disease of the kidney, or the latter may be found, as in examination for life insurance, before dropsy has made its appearance. In those cases in which the onset is gradual and the disease unsuspected, symptoms of mild uræmia are likely to be present. Digestive disturbances are frequent, as loss of appetite, nausea, and vomiting at irregular times, especially before breakfast, and diarrhœa, headache, and wakefulness may be associated. The patient loses strength and becomes pale.

The dropsy is usually first observed in the eyelids, ankles, and feet, the puffiness of the eyelids being seen in the morning, the swelling of the ankles and feet in the evening, and extends to the subcutaneous tissue throughout the body, and eventually into the serous cavities, lungs, and brain. In the atrophic stage with increased flow of urine the dropsy may diminish in severity.

The characteristics of the urine vary somewhat according to the stage of the disease. It is usually diminished to two pints or less, but the quantity may vary considerably from time to time. The color is high or dark red, perhaps smoky in appearance, according to the concentration or the presence of blood, and is opaque. The reaction is acid, and the specific gravity from 1020 upward. Urea is diminished. Albumin is abundant, from one to three per cent., the precipitate occupying one-half or the whole of the volume of the boiled urine. The percentage is least while the patient is at rest. The sediment is abundant, and consists of numerous hyaline, granular, epithelial, and fatty casts of various length and breadth, renal epithelium, and leukocytes, either granular or fatty, free fat-drops, fat-crystals, and fresh or decolorized red blood-corpuscles, sometimes so abundant as to indicate a hemorrhagic nephritis.

In the atrophic stage the quantity of urine is likely to be either normal or increased, the specific gravity 1010 and upward, the percentage of albumin from one-half to one per cent., the sediment containing abundant casts, but with less evidence of fatty degeneration and the addition of waxy or colloid casts. With the persistence of the disease the uræmic disturbances become more severe, and may prove the immediate cause of death, although dysentery, delirium, coma, and convulsions are less frequent than in acute diffuse and chronic interstitial nephritis. Uræmic paralyses and retinitis sometimes occur, especially late in the disease, but less frequently than in chronic interstitial nephritis. The heart, particularly the left ventricle, becomes somewhat hypertrophied and dilated, especially when the disease runs a protracted course. The aortic second sound is accentuated, and the tension of the pulse increased. Dyspnœa is frequent, may be asthmatic in character, and is due in part to uræmia and in part to œdema of the lungs or to hydrothorax.

The disease usually extends over a period of a few years, during which exacerbations and remissions may often occur, sometimes suggesting successive attacks of acute nephritis. Recovery is possible when the disease is limited to a portion of the kidney, but death usually results from progressive emaciation and exhaustion, with œdema of the lungs or larynx or concurrent acute inflammation, especially of the lungs or serous cavities, or from uræmia.

Diagnosis.—The diagnosis ultimately depends upon the examination of the urine, although persistent anæmia, frequent nausea and occasional vomiting without obvious cause, and œdema are sufficiently suggestive of a renal affection. The large white kidney of diffuse nephritis may be differentiated from that associated with amyloid degeneration by the failing etiology of the latter. Evidence of an associated amyloid spleen or liver is lacking, while the urine from an amyloid white kidney is paler, clearer, and its sediment less abundant, with predominating hyaline casts and but few red blood-corpuscles. It is important to discriminate between the acute exacerbation of a chronic nephritis and an attack of acute nephritis. In the former dyspepsia, anæmia, and œdema precede the immediate exacerbation, in which the dropsy rapidly increases; the flow of urine is more profuse than in acute nephritis, fatty casts are more abundant, and cardiac hypertrophy and albuminuric retinitis, if present, confirm the diagnosis of a pre-existing nephritis. The atrophic stage of chronic diffuse nephritis is to be diagnosticated when, after a prolonged period of dropsy with symptoms of mild uræmia, the former lessens, and the quantity of urine is increased, and perhaps excessive. The color is paler, the specific gravity lower, and the percentage of albumin higher than in the genuine form of atrophied kidney, while the sediment is more abundant, containing the variety of casts to be found in the urine from large white kidney, but with less fat.

PROGNOSIS.—The prognosis depends primarily upon the meaning to be attached to the term chronic diffuse nephritis. The milder varieties, essentially parenchymatous degeneration of the renal epithelium, usually terminate favorably, although the subsequent development of a more severe nephritis may be attributed to a prolonged period of latency following the original infection or toxæmia.

The severe variety of chronic diffuse nephritis demands an unfavorable, though guarded, prognosis. Recovery undoubtedly occurs when the pathological process is limited to a portion of a kidney. Hypertrophy of the rest of the kidney or of the heart then takes place, sufficient to compensate for the loss of the diseased portion of the kidney. The prognosis in recurrent attacks even is not necessarily fatal, since sufficient unaffected kidney may remain for physiological purposes. The prognosis of the case in hand depends upon the duration of the disease, the daily quantity of urine, the percentage of urea, and the severity of the symptoms, especially upon a sudden increase of the œdema or the onset of grave uræmia, and upon the occurrence of complicating diseases, as erysipelas, pericarditis, pleurisy, or pneumonia.

TREATMENT.—The indications for treatment in chronic Bright's disease are—first, to lessen the strain upon the kidney; second, if possible, to check diseased action in the kidneys themselves; third, to relieve symptoms as they arise.

The work of the kidney may be lessened by diminishing the production of excrementitious material in the body, and by increasing the activity of the other emunctories. Moreover, the irritation of the kidney under work may be diminished by dilution of the substances which act as irritants to the kidney. In order to lessen the amount of excrementitious material, the nitrogenous food taken should be reduced to the minimum, the chief reliance being upon farinaceous foods. In many, if not in all, cases a protracted trial should be made of "skimmed milk" diet, in which the patient should take at short intervals during the day such an amount of milk as will aggregate from two to four quarts in the twenty-four hours. There is no reason for believing that the effects of such diet are rendered nugatory by the moderate use of oatmeal, cracked wheat, or other thoroughly cooked farinaceous foods, which, moreover, tend to overcome the excessive constipation produced by the milk diet. In many cases buttermilk, koumiss, or matzoon may be advantageously substituted in part or altogether for sweet milk. During the twenty-four hours at least three quarts of liquid should be taken; and if the urine be acid, alkalies should be used *pro re rata.*

To maintain the action of the skin, the patient should, if possible, live in a warm, equable climate. If this be not attainable, the greatest care should be exercised, by the wearing of heavy woollen underclothing day and night, to prevent any chilling of the surface.

Sweating should be encouraged by the habitual free internal use of water; and at regular intervals, varying from one to four days, a sweat should be given by means of pilocarpine, or, as is usually better, by the vapor bath. In robust persons the sweat may be daily; when uræmia threatens, twice a day. It is usually better so to arrange that the patient shall not go out of his apartments after receiving the vapor bath until the next day.

Although it is doubtful whether any substance can favorably affect the nutrition of the chronically inflamed kidney, yet the power which has been ascribed by authorities to the strontium salts, especially the lactate, more than warrants its trial; thirty to fifty grains a day may be given in divided doses. Ferric chloride (ferri chloridum, from one to three grains, in its solid form, or in the form of the tincture or of Basham's mixture) is a standard remedy in the second stage of Bright's disease. It is a good ferruginous tonic, is somewhat diuretic, and also acts as an astringent. The pure diuretics are chiefly valuable in maintaining the action of the kidney or in soothing the irritated kidney. Irritant diuretics are usually harmful.

Although in renal dropsy it is, for obvious reasons, rarely possible to get rid of the excess of water by means of diuretics, it is often essential to stimulate the kidneys to their utmost. The diuretics which may be used for this purpose, enumerated according to their efficacy, are potassium bitartrate, sodium and theobromine salicylate, caffeine citrate, and pilocarpine hydrochlorate (one-twentieth of a grain every two hours). Scoparius is irritating to the kidneys, but is less so than squill; nevertheless a pill of calomel, squill, and digitalis, one grain each, is sometimes effective in desperate cases after the failure of other remedies.

When dropsy in renal disease is excessive there is usually a secondary cardiac failure, so that caffeine, strophanthus, and digitalis may by increasing the activity of the circulation be very serviceable. The chief reliance in renal dropsy must, however, be put upon sudorifics and purgatives. If in any case the water in spite of treatment so accumulates as to become a source of danger, relief may usually be obtained by puncturing the legs above the ankle, or by making several moon-shaped incisions just below the internal malleolus. Such a procedure should, however, always be delayed as long as possible, as very often the incisions refuse to heal.

The treatment of partial or complete urinary suppression in chronic Bright's disease is essentially the same as that of the same symptoms due to acute Bright's disease. (See page 1027.) Care must, however, be exercised in the abstraction of blood. For the treatment of uræmic symptoms see page 1038.

CHRONIC FIBROUS NEPHRITIS. CHRONIC INTERSTITIAL NEPHRITIS.

SYNONYMES.—Bright's disease, third stage; Gouty kidney; Contracted kidney; Cirrhosis of the kidney; Granular atrophy of the kidney.

Chronic interstitial inflammation of the kidney results in the production of an atrophy of the kidney. A similar result may occur in the late stages of chronic diffuse nephritis, in chronic endarteritis of the renal arteries, or of the aorta at the origin of these arteries, and in old age, the last two conditions being often associated. A distinction is thus drawn between the primary or genuine atrophy of the kidney due to a progressive inflammation of the interstitial tissue and a secondary atrophy of the kidney occurring in the course of chronic diffuse nephritis, localized chronic endarteritis, or advancing years. The first alone requires especial consideration as a well-defined pathological process, although the clinical distinction is not always to be sharply drawn between the several varieties of atrophied kidney.

ETIOLOGY.—The probable immediate cause of chronic fibrous nephritis, which is more common after middle life, is long-continued irritation of the kidney, the result of gout, syphilis, or malaria, or of alcohol, lead, or possibly other poisons. Prolonged obstruction to the outflow of urine, chronic pyelitis, and the development of multiple cysts, as in the multilocular cystic kidney, are associated with chronic interstitial nephritis, although in these affections the kidney is usually increased in size. It is not unlikely that malassimilation of food resulting from irregularity or excess in modes of living, especially in eating, drinking, working, and resting, may result in the prolonged elimination through the kidney of irritating products of defective digestion or disturbed tissue metamorphosis and thus prove of etiological importance. In this relation the frequent association of chronic fibrous nephritis and general arterio-sclerosis is significant. Attention has already been called to the possibility that the alteration of the blood-vessels may be due to the nephritis, and to the view of Senator, that in the genuine form of chronic fibrous nephritis a persistent toxæmia probably acts simultaneously as an irritant both to the blood-vessels and to the kidney. In those instances in which a general arterio-sclerosis is combined with chronic fibrous nephritis, it seems probable that the etiology is the same for each affection.

MORBID ANATOMY.—The kidneys are diminished in size, in extreme instances each weighing perhaps an ounce, and are of increased density. The capsule is thickened, and so adherent that, when torn off, portions of the kidney are separated with it. The surface is smooth or granular, and the granules, both large and small, are composed of relatively unaltered tissue, which is of a red or reddish-gray color according to the quantity of blood present. A distinction of no essential importance is sometimes thus drawn between a red and a pale granular kidney. Numerous minute cysts, and white or yellow specks from lime salts or urates, are frequently seen upon the surface and the section of the kidney, and similarly colored streaks from a like cause may be found on section of the pyramids. The cortex is thin and the pyramids short; the glomeruli and tubular regions are indistinct, although the larger blood-vessels are unusually conspicuous. On microscopical examination, more or less extensive, disseminated patches of fibrous and granulation tissue are seen, in which some of the tubules are obliterated while others are dilated and varicose and frequently contain globular and cylindrical hyaline concretions. The arteries show a tendency to become obliterated, and the walls both of tubes and of arteries are thickened; the glomeruli, with or without thickened capsules, are transformed into homogeneous, glistening, sclerosed bodies, which perhaps are infiltrated with lime salts. The combination of chronic fibrous nephritis and the deposition of urates is spoken of as a gouty kidney, and is often found among persons leading a life of luxury; while the fibrous kidney associated with lime salts has been regarded as evidence of a poor man's gout.

The general nutrition of persons with chronic fibrous nephritis may be but little affected: indeed, the atrophied kidney is not infrequently embedded in abundant fat-tissue. A most important associated lesion is concentric idiopathic hypertrophy, sometimes extreme, of the heart, either of the left ventricle alone or of both ventricles. The degree of the hypertrophy depends upon the duration of the disease and the general nutrition of the patient; the cause has been mentioned in the consideration of the cardio-vascular changes in nephritis. The hypertrophied heart may subsequently undergo fatty degeneration and the ventricular cavity become dilated. Retinal hemorrhages and albuminuric retinitis are more frequently found than in chronic diffuse nephritis. Chronic endaortitis (atheromatous degeneration of the aorta) and sclerosis of the smaller arteries in various organs of the body may be associated with a chronic fibrous nephritis, also pulmonary emphysema, cerebral hemorrhage or softening of the brain, and fibrinous inflammation of the serous membranes of the body.

Symptoms.—The symptoms of chronic fibrous nephritis may be so slight, ill defined, or remotely related to the kidney that the condition of this organ is unsuspected until an examination for life insurance discloses an abnormal urine, or a troublesome heart-beat or a feebleness of eyesight leads to a physical examination of the heart or retina, revealing lesions at once suggesting a chronic nephritis. The patient may consider his kidneys exceptionally sound, since urine is excreted with unusual freedom. As a rule, the earliest symptoms are those due to a mild uræmia. The patient complains of dyspepsia. He suffers from slight nausea, perhaps from vomiting at irregular times and independently of the ingestion of food. For a long time the digestive power of the stomach is unaffected and the general nutrition of the patient is excellent. During this period there is occasional vertigo, perhaps headache, usually mild, although sometimes persistent, also neuralgia, either occipital or following the distribution of the facial nerve. Itching of the skin may be complained of, and persistent eczema is not infrequent. The patient becomes short-breathed on exertion, or has asthmatic attacks, and recognizes a powerful beating of the heart. Thirst occurs combined with frequent micturition, especially noticed at night, and polyuria, symptoms which lead the patient to suspect diabetes. In virtue of one or more of these disturbances, an examination of the urine is made, and reveals the following characteristics. The quantity is excessive, three to four quarts (3000 to 4000 cubic centimetres). The color is pale and the urine transparent, the reaction acid, the specific gravity from 1005 to 1010, being higher in the urine passed during the day than in that passed after rest. The urea is somewhat diminished, while albumin is present from a trace to one-fourth per cent. At times it may be temporarily absent, or not recognized for weeks or months, or it may be present only during the day. Indeed, we have seen fatal uræmia from contracted kidney with urine that was of normal specific gravity and free from albumin. The percentage of albumin is usually less in the urine passed during the night than in that voided by day. The sediment is slight and contains but few, usually narrow, hyaline casts, and repeated examinations may be necessary before casts are seen. Granular casts are exceptionally found, and fat-drops are infrequent. Renal epithelium, leukocytes, and red blood-corpuscles are rare.

In the further progress of the disease the dyspeptic symptoms, particularly the nausea and vomiting, persist, and may be associated with obstinate diarrhœa, and the patient often, though not necessarily, loses flesh and strength. Anxiety and wakefulness are frequent. Muscular twitchings, at times convulsions, may take place, also disturbance of vision, and, especially towards the end of life, stupor or coma. The more severe uræmic disturbances may last but a short time or may recur with intervals of comparative health.

The recognition of the cardiac hypertrophy without valvular disease is of the greatest importance in calling attention to the condition of the kidney. It is manifested not only by palpitation and displacement of the apex downward and outward, but also by the increased area of cardiac dulness, especially to the left, and by the accentuation of the aortic second sound. Increased arterial tension is indicated by the resistant, radial pulse, the cord-like character of the artery suggesting a sclerosis of its wall. Throbbing of the temporal arteries, headache, and perhaps hemorrhages, nasal, cutaneous, or cerebral, are additional evidence, in part at least, of increased arterial tension. In the later stages of the disease, when the nutrition of the heart becomes impaired and dilatation is added to hypertrophy, as indicated by an extension of dulness to the right and perhaps a systolic souffle at the apex, the pulse becomes weaker, more frequent, and irregular, the dyspnœa more persistent, and dropsy may occur, the absence of which throughout the greater part of the disease strongly contrasts this variety of chronic nephritis with that due to diffuse inflammation of the kidney.

The early disturbances of respiration are probably the result of a mild uræmia, but in the further advance of the disease the dyspnœa, though often paroxysmal, is aggravated by mental or physical strain, and may assume a Cheyne-Stokes character, or be combined with œdema of the lungs or with bronchial catarrh. Such modifications in the character of the respiration suggest that the late dyspnœa is due rather to the failing competency of the heart than to uræmia alone.

The disturbances of vision range between an enfeebled eyesight and complete blindness. The latter may occur as a manifestation of uræmia in the absence of observable retinal lesions. As a cause of the obscured vision characteristic retinal changes are often found, which are included under the term albuminuric retinitis. Such are multiple hemorrhages and opaque glistening patches due to a fatty degeneration and sclerosis of the retina. More rare are other causes of loss of vision, as separation of the retina, embolism of the central artery, hemorrhage into the vitreous, or inflammation of the choroid.

Of other affections which may arise, acute inflammation of serous membranes is frequent, as are also acute pneumonia and dysentery, which not infrequently represent the immediate cause of death in a chronic disease whose date of origin is uncertain, and whose duration from the onset of symptoms attributable to renal disease may extend over a period of ten or more years.

Diagnosis.—Repeated examinations of the urine are usually necessary for making the diagnosis, although a suspicion of contracted kidney should always arise when palpitation, dyspepsia without obvious cause, wakefulness, frequent micturition, or unusual disturbance of vision is complained of. Stewart has emphasized the diagnostic importance of an appreciation of the symptoms of chronic fibrous nephritis, since there may be repeated failures to detect albumin, and casts may be only occasionally found. A pulse of high tension even with a cord-like radial artery is no necessary evidence of present or impending fibrous nephritis, since both may occur without any involvement of the kidney, and a failing cardiac compensation in the last stage of fibrous nephritis simulates that present in chronic valvular endocarditis. Repeated examinations of the urine are especially necessary in the former case, and, although as the heart fails the percentage of albumin may increase and the specific gravity rise, the latter is still low in proportion to the quantity of urine eliminated, nearer 1010 than 1020, whereas in chronic passive congestion of the kidney the specific gravity is always high. The characteristics of the urine in the atrophic stage of a chronic diffuse nephritis are sufficient to exclude this variety of fibrous kidney, although in senile atrophy and in atrophy due to chronic endaortitis the composition of the urine may resemble that of genuine chronic fibrous nephritis; but failing health in such cases is not associated with the progressive development of the symptoms recorded during a period of years. It is probable that the senile and arterio-sclerotic varieties of atrophied kidney are those in which albuminuria and casts may occur at times during a period of years in individuals in seeming health.

PROGNOSIS.—Although genuine chronic fibrous nephritis is a progressive affection, eventually proving fatal, a long period of years may elapse during which the individual leads an active, useful, and enjoyable life. Death may then occur unexpectedly from sudden cerebral hemorrhage or grave uræmia, or more gradually from an acute inflammation, as of the serous membranes, lungs, or intestines. In other cases death is of more gradual onset in consequence of the failure of cardiac compensation, which is often indicated by a diminution in the quantity of urine before œdema of the lungs or of the larynx or severe uræmic symptoms, such as Cheyne-Stokes breathing, convulsions, sleeplessness, sopor, or coma, become manifest.

TREATMENT.—It is manifestly impossible to restore the tissue of the kidney which has undergone contraction. In many cases the structural alteration is due to recurrent subacute attacks, caused by irritant poisons which are usually the products of malassimilation within the body. Recurrent attacks of oxaluria, or more frequently of lithuria, may each be accompanied by urine of low specific gravity containing a little albumin and hyaline tube-casts, although between the paroxysms the urine is entirely normal. These temporary attacks, which are almost invariably overlooked until the kidneys are ruined, may be looked upon as representing the early stages of gouty or other form of contracted kidney. It is therefore of the greatest importance that the urine of middle-aged persons be repeatedly examined during periods of uric acid elimination, after heavy eating or drinking, or whenever sediment is noticed. The detection of such renal attacks should lead to the institution of strenuous measures to prevent the formation in the body of irritant substances, and to aid in the elimination of these substances when formed.

At the basis of many cases of contracted kidney is the habit of overeating of nitrogenous foods and of under-exercising. In any case the diet should be made as far as may be farinaceous; systematic exercise should be insisted upon, and every care be taken to maintain the activity of the skin by warm clothing and frequent bathing. Alike to the overworked men and to the indolent, over-luxurious women who make up a large proportion of the richer population in our large cities, the formation of the Turkish bath habit in early middle life is important as a prophylaxis against renal contraction.

The management of a case of contracted kidney is in its general principles similar to that of other forms of chronic Bright's disease. (See page 1031.) Usually, however, there is no hope of affecting the kidney for good, and the utmost that can be done is to regulate the diet so as to lessen kidney strain, to stimulate the skin and intestinal tract to eliminate as much as they can, to give iron to aid in blood reconstruction, and to meet uræmic symptoms as they arise.

In the treatment of uræmia, the chief effort must be to bring about elimination of the poisonous materials from the blood. The diuretics used should be as far as possible unirritating. By means of pilocarpine and the vapor baths very free sweating may be induced. As elaterium is accredited with the especial power of causing excretion of urea from the intestines, it is generally to be preferred among the purgatives. (See formula 25.) In uræmic congestion or œdema of the lungs, free dry or sometimes wet cupping, with administration of oil of eucalyptus and other stimulating expectorants, is in order. In uræmic convulsions, venesection, anæsthetics, chloral, and bromides may be used, with due adaptation to the individual case. In uræmic vomiting, cocaine, carbolic acid, and other anti-emetics may be given, but usually fail to accomplish much: whilst the food should be of the lightest kind, and in extreme cases should be given for a time solely by the rectum.

AMYLOID DEGENERATION.

Amyloid degeneration or infiltration of the kidney may occur as a complication of chronic diffuse and chronic fibrous nephritis, and at times in an otherwise normal kidney. In the latter case it is of no practical importance, but in the former it produces modifications worthy of note in the course of chronic Bright's disease.

ETIOLOGY.—Amyloid degeneration in the kidney, as elsewhere in the body, is the result of chronic tuberculosis of the lungs, intestines, and bones in particular, and then especially when associated with ulceration or cavity formation. It also occurs in the sequence of chronic suppuration, particularly of the bones, and in empyema and syphilis; lead poisoning, gout, and malaria have also been considered as of etiological importance. It is usually associated with amyloid disease of the spleen, liver, and intestine, but it may be limited to the kidneys, in which case the etiology is obscure. The suppurative conditions are generally considered as sometimes concerned in the production of chronic diffuse nephritis, while lead poisoning, gout, and malaria are mentioned in the etiology of chronic fibrous nephritis. It is therefore possible that chronic nephritis and amyloid degeneration of the kidney may be independent results of the same cause, as it is to be recognized that in consequence of these causes nephritis without amyloid degeneration and amyloid degeneration without nephritis may occur. It is also possible, although less probable, that existing amyloid degeneration of the kidney may occasion the nephritis.

Morbid Anatomy.—The amyloid degeneration of an otherwise normal kidney may produce no characteristic gross changes, with the exception of pallor and translucency of the glomeruli, but the presence of amyloid disease in chronic diffuse nephritis is always to be suspected when a large white kidney is found, and the paler the kidney the more probable is the presence of the amyloid material. The amyloid degeneration may occur in atrophied kidneys of whatever origin, though it is perhaps most frequently found in the secondary atrophy of chronic diffuse nephritis. Pallor of the kidney and translucency of the glomeruli, again, may be the only appearances suggestive of the presence of the amyloid substance, which usually manifests itself first as a homogeneous translucent thickening of the walls of the glomerular capillaries and of the afferent arteries. It may also be present in the blood-vessels and in the basement membrane of the tubules in the pyramids. Its presence is to be recognized by the application of the compound solution of iodine, which produces a mahogany-brown color. The effect of amyloid degeneration of the blood-vessels is to produce a diminution of their calibre, and this may act as one factor in the causation of the fatty degeneration of the epithelium which is present in the large white amyloid kidney.

Symptoms.—The symptoms connected with amyloid degeneration of the kidney vary in character according to the associated lesion of the kidney. They may, therefore, simulate either those of a chronic diffuse or of a chronic fibrous nephritis, or there may be no suggestive symptoms that the amyloid degeneration is present if the kidney is otherwise normal. Albuminuria is constant and dropsy frequent. Polyuria, uræmic symptoms, and symptoms referable to hypertrophied heart or to retinitis are rare. The patient is pale and weak, and is likely to suffer from diarrhœa in consequence of associated amyloid enteritis, and to present evidence of enlargement of the liver and spleen from amyloid disease of these organs. The quantity of urine is not especially increased, and, when the kidney alone is affected, may be diminished to about one thousand cubic centimetres in the twenty-four hours. It is acid, clear, of a pale-yellow color, its specific gravity 1010 to 1015. Albumin is usually copious, in the vicinity of two per cent. Senator states that globulin is abundant. There is but little sediment, and this contains comparatively few hyaline, granular, fatty, and waxy casts, leukocytes, and red blood-corpuscles. The combination of amyloid degeneration and diffuse nephritis may be associated with a diminished quantity of urine and an abundant sediment of casts and leukocytes, while the quantity of urine may be profuse, with a high percentage of albumin if the amyloid degeneration has affected a preceding granular atrophy of the kidney with hypertrophied heart. If amyloid degeneration is present in an atrophied kidney without hypertrophied heart, the quantity of urine and the sediment may be slight and the quantity of albumin considerable. Cases occur in which in the absence of hypertrophy of the heart and a diminished quantity of urine albumin is absent, or is present merely in traces, and only a rare narrow hyaline cast is found.

Diagnosis.—The presence of amyloid degeneration of the kidney is to be inferred when an efficient cause for general amyloid disease exists and there is evidence of amyloid disease elsewhere, especially enlargement of the liver and spleen and chronic diarrhœa. It is rendered probable when the urine contains abundant albumin and but little sediment and there is no evidence of hypertrophy of the heart.

Prognosis.—Since amyloid degeneration usually represents a late stage of the diseases in which it occurs, and of which the prognosis is grave, the outlook for the patient is serious. It is possible that the progress of amyloid degeneration may cease with the arrest of the disease, and it is known that amyloid material may remain in the body indefinitely without being productive of disturbance. Its presence in the kidneys, however, is usually connected with incurable extensive disease in these organs and elsewhere.

Treatment.—Amyloid degeneration of the kidney cannot be met by any specific treatment, and often does not modify the therapeutics of the primary disease which causes it. Gallic acid is sometimes useful in controlling an excessive loss of albumin.

SUPPURATIVE NEPHRITIS. ABSCESS OF THE KIDNEY.

Suppurative inflammation of the kidney results in the presence of abscesses, which may be few or many, sharply localized or disseminated throughout the kidneys. Such abscesses are to be regarded as dependent upon the entrance into the kidney of pyogenic bacteria, which are admitted directly, as in traumatism, or indirectly through the circulation or by means of the urinary tract. When the bacteria are brought through the circulation, the abscess is sometimes called metastatic; while the term *surgical kidney* is often given to that variety of suppurative nephritis which results from the extension of an inflammation upward from a lower point in the urinary tract, since such a progress has been the frequent immediate result of a surgical operation on the urethra or the bladder.

The metastatic abscesses of the kidney which result from the transfer of bacteria by means of the blood-current are usually of embolic origin, infectious or septic emboli containing bacteria being transferred from the heart, as in infectious endocarditis, or from an infectious thrombus in the lungs, or in a body vein through an open foramen ovale. It is also possible that a suppurative nephritis may arise from the combination of bacteria in the renal circulation and a local disturbance in the kidney favoring their growth. Abscesses of the kidney are therefore grouped as the traumatic, resulting from injuries whether by accident or by design; the embolic, occurring in those diseases in which a malignant endocarditis exists as the local expression of a general infection, whether regarded as pyæmia, septicæmia, puerperal fever, rheumatism, osteomyelitis, diphtheria, typhoid fever, dysentery, tuberculosis, or actinomycosis; finally, the pyelonephritic, in which the inflammatory process is extended from the urethra, bladder, ureter, and renal pelvis, whether induced by surgical operations, including catheterization, by gonorrhœa, by renal calculus, or by unknown causes.

MORBID ANATOMY.—One or many abscesses are present in the kidney, varying in size from that of a pin-head to one involving the space between the capsule and the pelvis of the kidney. The abscesses are either discrete or confluent, and the wall smooth or shreddy. The suppuration may extend to the paranephric fat-tissue, and result in a paranephric abscess, which, as well as the renal abscess, may communicate with the pelvis of the kidney or with the digestive tract, especially the colon, duodenum, or stomach. A depressed scar or a calcified nodule is sometimes found in evidence of the previous presence of an abscess of the kidney.

SYMPTOMS.—The presence of the abscesses is usually indicated by irregularly recurring chills, with corresponding elevations of temperature, and a continued intervening fever, with less extreme elevation of temperature, and with the disturbances of digestion, circulation, and respiration and the nervous symptoms characteristic of fever. There is but little pain, except when the kidney is injured or the abscess extends beyond the kidney, especially when the peritoneum is approached. Although albuminuria is present and is sometimes abundant, the percentage may be no higher than exists in febrile conditions in which there is no local disease of the kidney. Hyaline and epithelial casts, renal epithelium, and leukocytes may be present, but red blood-corpuscles are almost always absent. If the abscess breaks into the renal pelvis, pus in considerable quantity suddenly appears in the urine.

DIAGNOSIS.—A traumatic suppurative nephritis is apparent when the symptoms mentioned are the immediate result of an injury. The embolic variety of suppurative nephritis is to be inferred from the presence of signs of a malignant endocarditis in a patient in whom increased albuminuria, and especially the sudden appearance of pyuria and perhaps pain in the region of the kidney, indicate a localized inflammation of this organ. Especial consideration will be given to that variety of suppurative nephritis which extends from below upward, pyelonephritis, and to the extension of the abscess from the kidney to the paranephric fat-tissue, paranephric abscess or suppurative paranephritis.

PROGNOSIS.—The pus from an abscess of the kidney may become absorbed or discharged, the prognosis in the former case being mild, in the latter serious, perhaps grave, according as the pus escapes into the urinary or into the digestive tract. The pus may be discharged into a neighboring hollow organ, with the production of a fistula, which, if in communication with the digestive tract, is likely to end fatally.

CHAPTER II.

CYSTS AND TUMORS OF THE KIDNEY AND DISEASES OF THE RENAL PELVIS AND THE BLADDER.

RENAL CYSTS.

CYSTS are of frequent occurrence in the kidneys, and may be found in an otherwise normal kidney or in one which is diseased. In the former there may be one or several, usually not larger than an apricot, although sometimes of sufficient size to produce an abdominal tumor which may cause mechanical disturbances.

The cysts to be found in diseased kidneys are multiple, sometimes innumerable, and in greater or less number are a constant accompaniment of chronic fibrous nephritis, in which affection they are of secondary importance. They are sometimes in such numbers and of such size as to produce extreme enlargement of the kidney, forming abdominal tumors, the multilocular cystic kidney, and being eventually accompanied by symptoms of grave renal disease. The cysts in an otherwise normal kidney or in one of chronic fibrous nephritis are considered to arise from a dilatation of the tubules or of Bowman's capsules by obstruction to the escape of urine. The larger result from the fusion of the smaller cysts, and contain an albuminous fluid, either serous or gelatinous, in which crystals of uric acid, calcic oxalate, and cholesterin, also fat or blood, may be present.

MULTILOCULAR CYSTIC KIDNEY. CYSTIC DROPSY OF THE KIDNEY.

DEFINITION.—Enlargement of the kidney due to the formation of innumerable cysts in one or usually in both kidneys, and producing symptoms resembling those of atrophy of the kidney.

ETIOLOGY.—The multilocular cystic kidney is a congenital affection, and its method of origin is variously explained. A fœtal nephritis, congenital closure of the papillæ, or an irregular development of the Wolffian bodies has been offered in explanation. This abnormity has repeatedly occurred in children of the same mother, and has been associated with other deformities, as hydrocephalus, cleft palate, club-foot, and cysts of the liver.

MORBID ANATOMY.—Both kidneys are usually symmetrically enlarged, forming tumors in the adult as large as an infant's head and in the new-born child of the size of the fist. The enlarged kidney is lobulated from the presence of innumerable cysts, from those of microscopic size up to others as large as plums, separated from each other by an intervening fibrous structure in which the renal tubules and glomeruli are here and there to be recognized. The contents are a thin watery fluid in which urinary salts and albumin are often found, and in which hemorrhages are not infrequent. Blood-pigment, fat, and cholesterin may also be present.

SYMPTOMS.—The multilocular cystic kidney may exist for years without the production of symptoms, since they are not infrequently found in persons dying of acute disease. At the same time such a degree of distention of the abdomen may occur in the fœtus that childbirth is interfered with, or the descent of the diaphragm is so prevented as to cause the death of the infant from asphyxia. The longer the individual lives the larger the diseased kidney, since the size and perhaps the number of the cysts increase with the growth of the possessor, while the secreting structure of the kidney lessens. In the adult, therefore, symptoms are likely to become manifest, but the tumors are rarely of such size as to produce mechanical disturbances, although the diseased kidney has been removed by the surgeon ignorant of its nature. The disturbances which arise are rather those of digestion, circulation, and nutrition, with an excessive flow of pale urine of low specific gravity, and with a trace of albumin due to the associated chronic fibrous nephritis, which increases in degree with the age of the individual. As a rule, these symptoms, like those of the granular kidney, become conspicuous after middle life.

PROGNOSIS.—The prognosis is that of fibrous nephritis, necessarily grave, although years may elapse after the discovery of the tumors and the recognition of their cause before death occurs. Early death may be expected if appreciable enlargement of the kidneys from the presence of cysts is found in the new-born child.

TREATMENT.—The medical and hygienic treatment of cystic kidney is that of chronic fibrous nephritis. As both kidneys are invariably more or less diseased, surgical excision is rarely, if ever, justifiable.

TUMORS OF THE KIDNEY.

Of the benignant variety of tumors of the kidney, fibroma, lipoma, myxoma, angioma, and adenoma may be mentioned, although they are of insufficient size and number to be of any practical importance. Grawitz has shown that the adenoma of the kidney, the gross appearance of which often resembles the lipoma, may proceed from displaced portions of the suprarenal capsule. The malignant tumors of the kidney are sarcoma and cancer. The former sometimes, especially in children, contain muscular fibres, constituting myosarcoma.

Cancer and sarcoma of the kidney occur both as primary and as secondary tumors, present similar gross appearances, produce like symptoms, and are to be distinguished, sometimes with difficulty, only by a microscopical examination.

ETIOLOGY.—Primary malignant disease of the kidney occurs oftener in males than in females, rather in early or late adult life than during the intervening period, and occasionally is to be found at birth. Secondary cancer owes its origin to disease in the vicinity, and particularly to primary malignant disease of the testis.

Morbid Anatomy.—One or both kidneys may be affected, and the right kidney is more often diseased than the left in secondary cancer of the kidney. Both the primary and secondary varieties occur as a diffuse infiltration or as nodules, the latter being more frequently the case in secondary cancer. As a rule, the affected kidney is increased in size, sometimes enormously, forming a tumor as large as a man's head, weighing twenty pounds, and producing an appreciable distention of the abdomen and displacement of the abdominal viscera. The surface is usually lobulated. On section the fibrous capsule of the kidney may overlie the tumor, or may have been perforated by the latter, or the neoplasm may be everywhere separated from the kidney by the capsule, in which case the adrenal origin of the growth is to be suspected. The shape of the kidney is usually maintained. The consistency of the mass varies throughout or in different portions, from the scirrhous to the soft medullary or encephaloid mass. The infiltrating cancer of the kidney even when extensive often preserves the distinction between the cortex and the pyramids. The color of the tumor varies from gray or grayish white through yellow to red or black, as fatty degeneration, necrosis, hemorrhage, and pigment formation exist often in the same tumor. The neoplasm may grow into the renal vein, thence into the inferior vena cava, and frequently projects into the pelvis of the kidney, which may be dilated and completely filled.

Symptoms.—Malignant disease of the kidney may progress without localizing symptoms. Usually progressive loss of flesh and strength and anæmia are present. Among the earliest symptoms suggesting that the region of the kidneys is concerned are pains, either fixed or shooting outward and downward, and attributable to an extension of the growth to the lower dorsal and lumbar nerves. Most important as an early symptom is hæmaturia, which may be constant or intermittent. slight or so considerable as even to prove rapidly fatal. With extensive hemorrhage and perhaps excessive attacks of renal colic, worm-like clots of blood may be voided with the urine. Cells or fragments of the tumor may also escape with the urine. The cells, unless containing glycogen, are of little value in diagnosis, owing to the variety of shapes assumed by the epithelium of the urinary tract. Attention has already been called to the diagnostic importance of the glycogenic generation in such cells. If fragments of the tumor are voided, a microscopical examination may reveal their nature. Most important in diagnosis is the recognition of the tumor, which is usually easily accomplished by bimanual palpation. Its renal nature is to be suspected from the shape and the seat in the lumbar region, the position behind the colon determined by inflation of the latter, and the usual lack of mobility. It is overlaid and also separated from the liver and spleen by the resonant intestine when not pushed aside, while the spleen, if enlarged, may be felt as a moving body in front of the diseased kidney. If the surface is lobulated and the consistency soft, the sensation of fluid may be suggested, and the aspirator may be used to determine the liquid or solid nature of the mass. Urea or fragments of cancer may be aspirated.

DIAGNOSIS.—Persistent renal hæmaturia, suggested by the dark-red color of the urine, the absence of clots, and the presence of albumin throughout the fluid, and of fragmented red blood-corpuscles, with or without pain, and with cachexia, points to cancer of the kidney even in the absence of enlargement. Progressive cachexia with persistent violent pain in the region of the kidney which cannot otherwise be accounted for warrants the gravest suspicion of malignant disease, even if there be no discoverable tumor and no abnormality of the urine. Tumors of the kidney are usually readily distinguished from an enlarged spleen by the mobility and the characteristic edge of the latter. Tumors of the liver and a distended gall-bladder are also easily movable, and are not overlaid by the distended colon. Pelvic tumors proceed from below, and are not covered by intestine. The determination of the solid nature of the renal tumor may require the use of the aspirator and the chemical and microscopical examination of the aspirated contents.

PROGNOSIS.—When but one kidney is the seat of the tumor the prognosis is more favorable than if both kidneys are affected, since extirpation of the diseased kidney when sarcomatous has been followed by relief of the symptoms and prolongation of life. Malignant disease of the kidney under medical treatment generally proves fatal in the course of a year or two after its recognition. Death occurs from prolonged cachexia, or from the occurrence of gangrene of the tumor, which often results from the establishment of fistulæ between the growth and the intestine or the surface of the body, or the fatal issue may take place rapidly from hemorrhage, from rupture of vessels near the surface of the tumor, either into the urinary tract or into the peritoneal cavity.

TREATMENT.—The medical treatment of malignant tumor of the kidney practically amounts to the administration of narcotics for the relief of pain. Only in very rare cases can the diagnosis be reached sufficiently early in true cancer to justify excision. According to Abbe, even in sarcoma of the kidney it is very seldom that the case survives beyond three years. The immediate mortality from nephrectomy for malignant disease of the kidney has been about fifty per cent. (Barth.)

PYELITIS. PYELONEPHRITIS.

Inflammation of the pelvis of the kidney, and inflammation of the kidney as a result of the former, demand conjoint consideration from their unity of origin and their frequent concurrence. A pyelonephritis is due to a pyelitis, although the latter may occur without the former.

ETIOLOGY.—Inflammation of the mucous membrane of the pelvis of the kidney, pyelitis, is the result of the presence of an irritant, usually bacterial, conveyed from below upward, from the bladder along the ureter, or from above downward, as may occur in infectious diseases, *e.g.*, scarlet fever, diphtheria, small-pox, typhoid fever, pneumonia, and tuberculosis, or in the elimination of such poisons as cantharides, turpentine, copaiba, and cubebs. In this series belong the instances of pyelitis occurring in diabetes attributed to the elimination of glucose. The irritant may be transferred from adjacent parts, as in the case of the rupture of a neighboring abscess into the renal pelvis. Pyelitis may be induced or aggravated by the presence in the pelvis of the kidney of a foreign body, as a calculus, parasite, or blood-clot, or by the presence of a tumor growing and degenerating in the pelvis of the kidney. Finally are to be recognized cases of pyelitis called idiopathic or spontaneous, which are attributed to exposure to cold.

The bacterial varieties of pyelitis, especially when due to an extension of inflammation from the bladder, are those productive of pyelonephritis, and have been most frequently occasioned by the use of unclean catheters and other instruments, and by the failure to avoid sepsis in operations on the bladder and urethra. The occasional occurrence of pyelitis after parturition is also to be thus explained. Both pyelitis and pyelonephritis are more frequent in men than in women, and in adult life than in youth.

MORBID ANATOMY.—The inflamed mucous membrane presents the characteristics of a catarrhal, suppurative, or diphtheritic pyelitis, the first indicated by swelling and injection of the mucous membrane, perhaps with the addition of punctate hemorrhages and of opaque residual urine in the pelvis of the kidney. In the suppurative variety the mucous membrane is thickened, and the injected blood-vessels are less conspicuous than in the catarrhal state, but pus is present in greater or less quantity in the pelvis of the kidney, and the mucous membrane may be ulcerated. In diphtheritic pyelitis superficial necroses are present, in which urinary salts are frequently precipitated, and the apices of the pyramids may also be necrotic or destroyed. When hemorrhages occur in the inflamed mucous membranes, the term hemorrhagic pyelitis is applied. If the inflammation has extended from below upward, similar appearances may be found in the bladder and ureters, although the latter often show evidences of a catarrhal inflammation while an ulcerative or a diphtheritic inflammation is present in both the bladder and the pelvis of the kidney. The alterations may affect one or both kidneys.

Pyelonephritis is characterized by the presence of opaque gray or yellowish-gray, more or less beaded streaks continued along the pyramids from their apices into the cortex of the kidney. Such streaks and spots have an injected border, and, as they enlarge, tend to become confluent and softened in the centre, forming an abscess. In the earlier stages bacterial colonies, frequently of the colon bacillus, are found within the tubules, the epithelium of which is necrotic, while the adjacent intertubular tissue is infiltrated with leukocytes. A granular degeneration of the epithelium of the convoluted tubules is associated. The remains of a previous pyelonephritis are at times to be found in various parts of the kidney as foci of fibrous tissue, in which the tubules and glomeruli have been obliterated.

SYMPTOMS.—The symptoms of pyelitis are often insignificant or subordinate to those of the disease in which it occurs as a complication, and the existence of the affection is often determined by the examination of the urine alone. This is equally true in acute and in chronic pyelitis. There may be frequency of micturition, although this symptom is more suggestive of cystitis. There may be dull pain in the region of one or both kidneys, with sensitiveness on pressure, or the pain is more severe, following the course of one or both ureters. There may be but little constitutional disturbance if the inflammation is limited to the mucous membrane of the renal pelvis. The urine in simple pyelitis presents a normal color and specific gravity, and has an acid reaction. The percentage of albumin is small, and corresponds to the quantity of pus present. The sediment also varies in quantity according to the amount of pus present, which is more abundant in chronic than in acute pyelitis. The sediment contains pus-corpuscles, few or many red blood-corpuscles, and epithelial cells in considerable number. The epithelial cells from the pelvis of the kidney have no characteristics either of shape or of arrangement by which they are to be discriminated from those of the bladder. If the pyelitis is limited to one kidney, the flow of purulent urine may be followed by that of a normal urine from temporary obstruction of the ureter continuous with the diseased renal pelvis. Within the last few years the diagnosis of such a limited pyelitis in the female has been repeatedly accomplished by catheterization of the ureters, a method of diagnosis made familiar by Kelly, of Baltimore.

The onset of a pyelonephritis is indicated by the occurrence of chills, fever, and sweating, in addition to the possible pain in the region of the kidneys and ureters. The frequent recurrence of the chills often suggests a malarial fever, although they occur without periodicity. The disturbances of appetite and digestion and of the nervous system characteristic of fever are associated. In addition to the characteristics of the urine already mentioned, hyaline and epithelial casts and renal epithelium are to be found. The urine is more likely to be alkaline in pyelonephritis than in pyelitis, since the decomposition of retained urine is a most important factor in its production.

DIAGNOSIS.—The diagnosis of pyelitis is often to be made by the recognition of pyuria, which may be the sole symptom. In such cases the acid character of the urine and the absence of vesical tenesmus are sufficient to indicate the source of the pus. In chronic pyelitis, especially when there is polyuria, the symptoms are somewhat suggestive of fibrous nephritis, but there is often persistent pain in the region of the kidney. The diagnosis of pyelitis with dilatation of the pelvis of the kidney will be considered under pyonephrosis.

Prognosis.—The prognosis of pyelitis depends largely upon the ease with which the cause can be removed. It is, therefore, favorable in the infectious diseases and when the inflammation is the result of toxic irritants, but when the removal of the irritant demands a surgical operation the prognosis becomes more grave. The prognosis of a pyelonephritis is also grave, especially when it occurs in a person in whom, in virtue of prolonged obstruction to the escape of urine from a stricture or an enlarged prostate, chronic disease of the urinary tract has preceded the occurrence of pyelonephritis. Even in such cases the patient may recover, although a permanent atrophy of the kidney results.

Treatment.—The treatment of pyelitis depends mainly upon its cause. Under all circumstances the greatest care should be exercised to prevent chilling of the surface, and in acute or subacute cases confinement to bed is essential. The food should be always largely farinaceous, and in many cases trial of absolute milk diet should be made. In times of exacerbation the long-continued lukewarm bath often acts favorably. Astringent remedies are rarely of any value, but when the discharge is excessive, gallic acid will sometimes reduce the amount in cases of recent date and of moderate intensity, in which class of cases buchu, salol, and especially uva ursi, or, better, its active principle; arbutin (from twelve to twenty grains a day), may also be tried in large doses.

In older cases with much suppuration boric acid may be employed (ten grains from three to six times a day in diluted watery solution), or the more stimulating remedies, such as oil of copaiba, oil of sandal wood, and even oil of turpentine, may be given in ascending doses. If in any case the urine is strongly acid, very dilute solutions of alkalines may be used. Commonly the patient should be encouraged to drink very freely of water.

It is evident that in a large number of cases pyelitis depends upon a cause which is not to be relieved by medical treatment, and must come under the notice of the surgeon. When there is stricture, it should be attended to without delay; if in any case the tumor becomes perceptible and the symptoms are severe, there should be immediate surgical exploration, followed by such operation as may seem advisable. (See also Renal Calculi and Tubercular Kidney.)

HYDRONEPHROSIS AND PYONEPHROSIS.

Definition.—Dilatation of the pelvis of the kidney, the contents of the dilated pelvis being urine alone, hydronephrosis, or mixed with pus, pyonephrosis.

Etiology.—The chief cause of the dilatation is prolonged obstruction to the outflow of urine, which may be occasioned by contraction, compression, or obstruction of the urinary tract at any point below the pelvis of the kidney, and may affect one or both pelves. Contraction is the result of inflammation of one or both ureters, which may end in obliteration of the canal. Obstruction to the outflow of urine may be due to congenital causes, as atresia, valvular folds, twists, or oblique insertion of the ureter, but more important are the acquired causes of obstruction, as inflammatory processes around the ureter, a displaced uterus, and uterine, ovarian, or rectal tumors compressing or constricting the ureters. Obstruction may also be the result of causes acting from within the urinary tract, as inflammation, stricture, enlarged prostate, calculi, and tumors. The occasional occurrence of hydronephrosis without recognizable obstruction is to be borne in mind.

Pyonephrosis arises when the dilated renal pelvis becomes the seat of a suppurative inflammation. On the other hand, a chronic pyelitis may result in dilatation of the renal pelvis, with associated atrophy of the kidney, either from a weakening of the wall of the renal pelvis or from obstruction, perhaps temporary, to the outflow of urine.

Morbid Anatomy.—A cystic tumor results, occupying the site of the kidney, and assuming its shape, but perhaps large enough to appear as an abdominal tumor containing several gallons of fluid. The larger its size the more likely are the abdominal organs to be displaced, and the colon may lie upon the tumor or at one side. It is composed chiefly of the dilated renal pelvis and calices, the more or less atrophied kidney appearing as an appendage to the former, and the larger the tumor the greater is the degree of atrophy of the kidney. The interior of the sac shows communicating sacculi, the dilated calices, at the bottom of which the flattened pyramids are to be recognized. Chronic interstitial nephritis arises as the dilatation progresses, and the liquid contents of the cyst present the characteristics of the urine in this affection. The contents are essentially a urine of low specific gravity, although in hydronephrosis of long duration uric acid and urea may be absent. There is usually no sediment, but blood, pus, fat, cholesterin, or chalky material may be present in virtue of complicating hemorrhage or inflammation. If the obstruction is in the ureter, the latter may become so dilated as to suggest the small intestine. In pyonephrosis the sac contains pus in addition, and instead of the smooth and shining grayish-white wall of hydronephrosis there is a rough, opaque yellow, perhaps granular or ulcerated, wall.

Symptoms.—In general the symptoms of hydronephrosis are due to the resulting tumor, and eventually to the associated fibrous nephritis. The usually gradual formation of a tumor produces no disturbance until a considerable size has been attained. In unilateral hydronephrosis the sound kidney excretes a normal urine, while in bilateral hydronephrosis the urine may be normal until towards the close of life, when it presents the characteristics of interstitial nephritis. In such cases the frequently associated hypertrophied heart is compensatory and prevents oliguria. Cases of intermittent hydronephrosis are to be recognized, in which temporary obstruction to the outflow of urine takes place, the rapid formation of the tumor being associated with pain in the region of the affected kidney. There is temporary diminution in the quantity of urine passed, with perhaps vomiting and fever, lasting a few days, when relief follows the evacuation of a considerable quantity of urine in which blood may be present. The tumor is unilateral or bilateral, according as the obstruction affects one or both ureters, and, even if but one kidney is involved, may fill the greater part of the abdomen. The tumor is resistant, and when small may descend with the diaphragm; its surface is smooth, and a sense of fluctuation is more or less distinctly transmitted. When the colon crosses the tumor the former, if empty, may be felt as a movable cord, and can be distended by inflation of the intestine. The larger the tumor the more likely is the colon to be displaced laterally. The tumor may produce the sensation of fluctuation, occasion pain extending into the thigh, cause persistent constipation, or give rise to dyspnœa by interfering with the movements of the diaphragm. The tumor in pyonephrosis rarely attains so large a size as that of hydronephrosis. If the obstruction is intermittent in character, the diminution in the size of the tumor is associated with the presence of pus, blood, and albumin in the urine. In persistent pyonephrosis the symptoms are those of a suppurative pyelitis. The evacuation of the tumor, whether the latter is due to the presence of urine or of pus, may take place into the intestine, pleural cavity, or lung. As a rule, with the progressive enlargement in double hydronephrosis symptoms of chronic uræmia occur, the urine presenting the characteristics of that of chronic fibrous nephritis.

DIAGNOSIS.—Persistent hydronephrosis progresses as an abdominal tumor, the renal nature of which may be suspected from the seat, especially when the colon lies in front. The greater the size of the tumor, the more likely is it to be confounded with cystic or solid enlargements of the abdomen, whether of renal, hepatic, ovarian, or ascitic character. The tumor of pyonephrosis presents similar physical characteristics, and, except that it rarely attains so large a size, may also be confounded with other abdominal tumors, but is more likely to be mistaken for a circumscribed peritonitis, especially when in the right half of the abdomen or with a paranephric abscess. The presence of pus in the urine may serve to exclude a circumscribed peritonitis, while aspiration may be necessary to determine the cystic nature of the tumor, and thus to differentiate it from solid abdominal tumors, and to permit an examination of the fluid, the renal origin of which is usually indicated by the presence of urea and uric acid. Even aspiration may fail to establish a correct diagnosis in unilateral hydronephrosis of long standing in which the kidney has been practically destroyed.

PROGNOSIS.—Unilateral hydronephrosis may produce purely mechanical discomfort. Bilateral hydronephrosis, when progressive, has an unfavorable prognosis, essentially that of chronic fibrous nephritis. Rapidly fatal termination may follow the rupture of the sac in either variety of hydronephrosis.

TREATMENT.—Congenital hydronephrosis when bilateral cannot be reached by any treatment. When unilateral it may be palliated by tapping,—an operation, however, which involves serious risk of rupturing the sac or of producing peritonitis. In rare cases the fluid can be forced out by manipulation of the abdominal walls. Surgical operation with the purpose of relieving the kinks or twists of the ureter, or of removing other remediable obstructive causes of the disorder, or of removal of the kidney itself, is certainly justifiable, but there are at present no reliable surgical statistics.

Acquired hydronephrosis has been relieved by massage, with a sudden discharge of the urine and immediate subsidence of the tumor; but some danger attends the massage, and too much force may rupture the sac. Aspiration affords temporary relief, and has by repetition produced cure in recorded cases. The aspirating needle (not trocar) should be inserted on the right side, two and a half inches behind a line perpendicular to the anterior superior spine of the ileum, and midway between the crest of the ileum and the last rib. On the left side the needle should be inserted about an inch higher up. Nephrotomy, which has been performed in a number of cases, seems to be very rarely if ever fatal, but in more than fifty per cent. of the cases produces a permanent fistula. (Bruce Clarke.) Nephrectomy, according to the statistics of Newman, has had a mortality of forty-one and three-tenths per cent.

Intermittent hydronephrosis often does not cause serious symptoms, and in such cases should be left to nature. When it is due to a movable kidney, and consequent kinking of the ureter, it may be relieved by nephrorrhaphy.

In pyonephrosis, as soon as the existence of pus has been determined by aspiration, nephrotomy by a lumbar incision should be performed; almost invariably nephrectomy is ultimately necessary, but the result is undoubtedly better when the major operation is deferred until the patient has recovered from the pyæmic condition produced by the pyonephrosis.

SUPPURATIVE PARANEPHRITIS. PERINEPHRIC OR PARANEPHRIC ABSCESS.

DEFINITION.—A purulent inflammation of the fat-capsule of the kidney.

The importance of the recognition of an abscess in the fat-tissue surrounding the kidney makes it desirable to give separate consideration to this condition, which frequently is the result of disease of the kidney, although it may arise from other sources. A certain degree of confusion in the exact designation of the process has arisen from the failure to discriminate between the fibrous and the fat capsule of the kidney. The former may be inflamed, perinephritis, even acutely, but contains no extensive accumulation of pus, nor does any large abscess ever lie between the kidney and its fibrous capsule. The fat capsule of the kidney, on the contrary, is continuous with the subperitoneal fat-tissue, and frequently becomes the seat of extensive suppurative inflammation, which has been commonly called a perinephritic abscess, although there may be no inflammation either of the kidney or of its fibrous capsule. Paranephritis may be acute or chronic, the former being characterized by the presence of pus, the latter resulting in the formation of fibrous tissue.

Etiology.—This affection occurs twice as often in men as in women, and more frequently in adult life than in youth. Its origin is never spontaneous, though it may be so concealed as to receive this term, but represents the extension of an inflammatory process from elsewhere, usually from the immediate vicinity. It may follow a wound, as from a knife or a bullet. It often occurs from the extension of a suppurative nephritis or pyelitis, appendicitis, septic thrombosis or lymphangitis following parturition or operations upon the pelvic organs, testes, or spermatic cord, abscesses of the liver and suppurative perihepatitis, caries of the spine or pelvis, and suppurative inflammation of the lungs or pleura. Suppurative paranephritis may also occur in the sequence of infectious diseases, as typhus and typhoid fevers and small-pox. This affection most frequently occurs in suppurative nephritis and chronic renal tuberculosis.

Morbid Anatomy.—Inflammation of the fat-tissue rapidly results in the formation of pus, and the peritoneum is separated from the fibrous capsule of the kidney by a trabeculated cavity with ragged walls and communicating sinuses containing pus, blood, and sloughs. In the further progress of the inflammation the abscess extends both upward and downward, and perforation may occur in the loin, perineum, or groin, or into the renal pelvis, bladder, colon, duodenum, or the peritoneal or pleural cavity.

Symptoms.—The onset of a suppurative paranephritis is usually obscure, since this affection occurs as a complication of a variety of well-characterized diseases in which fever is a common feature. The latter is often intermittent and associated with chills, and accompanied with loss of appetite and disturbed digestion, which favor progressive emaciation and debility. The patient suffers from thirst, the bowels are constipated, the pulse is feeble, and respiration may be quickened. Eventually the patient assumes a typhoidal aspect, especially if evacuation of the pus has not occurred. Pain and swelling are the localizing symptoms. The pain is in the region of the kidney, becomes aggravated on pressure, and may extend into the legs. The swelling is best appreciated on bimanual examination while the patient is in the dorsal position. It occupies the lumbar region, and the space between the lower ribs and the crest of the ilium may distinctly bulge. The skin in this region may be congested and œdematous, and a localized sensation of fluctuation may indicate the approximation of the abscess to the surface of the body. The urine may show merely a trace of albumin or a few red blood-corpuscles, as in congestion of the kidney. The presence of pus in the urine indicates an associated pyelitis, or, when sudden and in large quantity, the rupture of the abscess into the urinary tract.

Diagnosis.—The nature of the painful tumor in the region of the kidney associated with fever is determined by an appreciation of the possible causes. Its position behind the peritoneum may be fixed by inflation of the colon. The appreciation of its exact nature may require aspiration, for the examination of the urine is of but little value. Suppurative paranephritis on the right side originating from appendicitis, as a rule, is of sudden onset and rapid progress.

Prognosis.—The outcome of a suppurative paranephritis is largely dependent upon the cause. If this is remediable, the prognosis is favorable, and recovery has followed both the absorption of the pus and its spontaneous evacuation. The latter result is favorable only when free drainage and freedom from sepsis concur. In the absence of these conditions death is likely to result from septicæmia, embolic abscesses, progressive emaciation and debility, or amyloid degeneration.

Treatment.—Absolute rest, milk diet, saline purgation, or local depletion may possibly arrest a forming perinephric abscess, but so soon as there is reasonable ground for believing that pus is present, a free lumbar incision should be made and thorough drainage established.

NEPHROLITHIASIS. RENAL CALCULUS.

Definition.—The conditions associated with the formation of precipitates from the urine in the kidney or the renal pelvis.

Several of the constituents of the urine may be precipitated in the urinary tract, especially in the pelvis of the kidney and in the bladder. The resulting sediment is accumulated in greater or less quantity, and is designated as sand, gravel, or stone, calculus, according to the size attained. The calculus is called renal when found within the kidney or its pelvis, and during its passage from the kidney to the bladder.

Etiology.—Calculi occur more often in men than in women, and most frequently in children and elderly persons. Heredity, locality, and sedentary and luxurious habits appear to be favorable to their formation. They are frequent in gout, and, though often a cause of chronic pyelitis, they may be a result of that disease. The immediate cause of the formation of calculi is uncertain, although clots, shreds of tissue, or the ova of parasites may serve as nuclei.

Composition and Appearances.—Uric acid and the urates, either separately or together, calcic oxalate, calcium phosphate, the triple phosphates, calcium carbonate, cystin, xanthin, and indigo, may be precipitated. Phosphatic calculi usually have a nucleus of uric acid or of calcic oxalate. The calculi of uric acid, calcic oxalate, and phosphates are relatively common; the other varieties are very rare. The phosphatic calculus is present in alkaline, the others in acid, urine. Calculi may occur in one or both kidneys, are single or many, as many as a thousand having been found in one renal pelvis, and vary in size from that of a grape-stone up to that of a goose-egg. They may be round or smooth, granulate or spinous, or be irregularly branching with protuberant knobs projecting into the dilated calices. Uric acid calculi are yellow, red, or brown in color, and lamellated when broken. The calcic oxalate or mulberry calculus is dark brown and dense. Phosphatic calculi are gray, friable, somewhat porous. Cystin forms a calculus resembling wax. Xanthin appears as a hard brown calculus, indigo as a dark-blue mass.

The renal calculus is usually associated with dilatation of the pelvis of the kidney, sometimes extreme, and pyelitis. Pyelonephritis, suppurative paranephritis, and atrophy of the kidney, may occur.

SYMPTOMS.—Renal calculi may be present and produce no symptoms. Gravel, sand, or small calculi may be passed repeatedly through a period of years without discomfort. As a rule, however, disturbances are connected both with the presence and with the passage of the calculus.

The presence of the calculus in the renal pelvis is usually indicated by lumbar pain and tenderness near the kidney concerned, hæmaturia, pyuria, and perhaps a tumor in the region of the affected kidney. The pain is rather a dull ache, more or less constant, though sometimes aggravated by motion. Although usually limited to the affected kidney, it may be referred to the sound kidney. Blood-corpuscles are frequently found in the urine, sometimes in sufficient number to produce discoloration. Pyuria is frequent, since pyelitis is the usual result of a calculus retained in the pelvis of the kidney. The quantity of pus is commonly small, and there may be intervals when none is passed. The existence of the pyelitis is also made evident by the occurrence of chills and fever. If the calculus obstructs the outflow of urine, dilatation of the pelvis of the kidney occurs, either a hydronephrosis or a pyonephrosis, according as pyelitis is absent or present. Abscesses of the kidney or paranephritis may follow the pyelitis, the latter being indicated by an increase in the size of the renal tumor and persistent elevation of temperature. If a chronic inflammation of the kidney results, symptoms of mild uræmia develop, or a prolonged cachexia from amyloid degeneration of the kidney may follow.

The passage of the stone produces more characteristic symptoms. These are renal colic and hæmaturia. The pain usually begins instantaneously, especially during exertion, though it may arouse the patient from a sound sleep. It is cutting or stabbing, and is sharply defined, extending along the course of the ureter, either towards the testicle or into the thigh. It may be most severe in the back, or may radiate upward, perhaps into the epigastrium. The pain may cease suddenly, either from the escape of the stone into the bladder or from its return to the pelvis of the kidney, and a recurrence of the paroxysm take place with the renewal of efforts at the expulsion of the stone. The attack of pain may be associated with a chill or chilliness and a sensation of faintness, and in children be accompanied with convulsions. The spasms of renal colic may last for an hour or more, or recurrent paroxysms be continued for many hours, with sudden relief if the stone enters the bladder. Hæmaturia associated with the attack of renal colic may be sufficient to produce a distinct redness of the urine and an appreciable bloody sediment, or merely a smoky color, and may persist for several days after the passage of the calculus. There is increased frequency of micturition, and the quantity of urine passed may be either less or greater than normal. Rarely there is suppression of urine lasting for days, although the stone is usually impacted in but one ureter. If the calculus is impacted in the ureter, ulceration, perforation, abscess, and peritonitis may occur. The patient prefers to lie on the affected side with updrawn knees, and the testicle on the affected side is frequently retracted and sometimes swollen when the pain extends into the scrotum.

Diagnosis.—Renal colic may be confounded with intestinal colic, and, when on the right side, with hepatic colic or the pain from perforating appendicitis. Intestinal colic is not so sharply defined and more rapidly improves, while the frequent limitation of the pain to the course of the ureter, the hæmaturia, and the absence of localized extreme tenderness exclude appendicitis ; the lack of jaundice and dilatation of the gall-bladder are important in eliminating hepatic colic. It is to be remembered that severe pain even in paroxysms, but without hæmaturia or the passage of calculi, may be referred to the region of the kidney and ureters. To such attacks, which are sometimes associated with a movable kidney, the term *nephralgia* is applied : patients suffering from them have been operated upon with a view to the removal of a probable calculus, although, according to Ransohoff, in forty-four recorded cases the surgeon has failed to find a calculus. In several instances, however, the nephralgia, like the similar hepatalgia, has been relieved by the operation. The calculous nature of a pyelitis or a nephritis is to be suspected, in the absence of attacks of renal colic, from the persistence of pain limited to the region of the kidney, aggravated on motion and often associated with blood in the urine. Especial importance is to be attached to the persistent presence of red blood-corpuscles recognizable by the microscope. The finding in the urine of crystals is of little aid in the diagnosis of a calculus, though of value in determining the nature of a calculus which has been diagnosticated by other evidence.

Prognosis.—An attack of renal colic rarely proves fatal, and successive attacks may be borne without risk to the life and general good health of the patient. The occurrence of anuria is a serious complication, although recovery has taken place after its existence for twenty days. A calculus too large to pass through the ureter may be retained in the pelvis of the kidney without producing serious disturbance. There are no known means by which it may be dissolved while in the pelvis of the kidney, and its presence there is always a source of danger to the life and health of the patient, through the production of the serious inflammatory and degenerative conditions mentioned.

TREATMENT.—In renal colic the patient should be put in a hot bath, and hypodermic injections of morphine should be given, with inhalations of ether or chloroform, as in biliary colic. Very hot fomentations over the seat of pain are usually grateful to the patient. Manipulations of the part do no good, but it is said that cases have occurred in which inversion of the body has been followed by slipping back of the stone and immediate relief. The drinking of large quantities of feebly alkaline water, or, if there be nausea, of carbonic acid water with potassium citrate and lemon juice, may be of service in lessening the renal irritation.

The treatment of a patient suffering from habitual renal gravel or calculus depends largely upon the nature of the concretions. Whenever there is a calculus in the pelvis of the kidney, violent, especially sudden, exertions, jolts, or falls must be sedulously avoided, for fear of dislodging the stone and throwing it into the ureter. If the calculus be uric acid, the diet should be largely farinaceous; if the calculus be oxalic acid, sweets and starchy food should be avoided. In all cases the habit should be formed of drinking large quantities of water between meals, two to three pints a day. In uric acid nephrolithiasis Saratoga, Vichy, or other alkaline mineral waters may be used; they are, however, probably inferior to artificial waters made by the addition of a known quantity of alkalies to carbonic acid water,—one or two drachms of sodium bicarbonate and twenty to thirty grains of potassium bicarbonate to the pint. Alkaline waters may often be advantageously substituted, especially in uric acid gravel, by the benzoated water given in formula 4; it is very effective in removing uric acid, probably by converting it into hippuric acid.

Although there is no sufficient reason for believing that calculus once formed in the kidney can be dissolved, nevertheless, in obedience to authority, trial may be made with alkaline solvents,—potassium citrate given well diluted to the extent of half an ounce a day, or piperazin in doses of fifteen grains three times a day, kept up for several months. This treatment is, however, not free from danger: not only may the general bodily condition be depressed by the long use of the alkali, but the continuous alkalinity of the urine may lead to deposition of the phosphate about the calculus.

The only radical cure when stone is permanently lodged in the kidney is in surgical operation,—either nephrotomy, followed by the extraction of the stone, or nephrectomy, the removal of the kidney. Nephrectomy should never be practised unless the kidney has undergone hopeless, secondary changes, a condition which should never be allowed to occur when the case comes under treatment sufficiently early. Persistency of attacks of renal colic may of themselves demand nephrolithotomy, but the appearance of pus in the urine or persistent albuminuria between the attacks should lead to its immediate performance.

The value of the early operation as contrasted with the late is well shown in the statistics collected by Thorndike as contrasted with those of Newman and Legue. In one hundred and twenty-eight cases, including those with and those without marked purulent discharges, there were eighteen deaths following nephrolithotomy, giving a mortality of fourteen per cent., the majority of the deaths being in suppurative cases (Thorndike); in eighty-two cases free from suppuration, reported by Newman and Legue, the mortality was only two and four-tenths per cent. White gives the average mortality of nephrolithotomy at five per cent.

DISEASES OF THE BLADDER.

ENURESIS. INCONTINENCE OF URINE.

Incontinence of urine is the result of paralysis of the sphincter or of contraction of the compressor muscle of the bladder, in either case the bladder being unable to retain the urine. *Paralytic incontinence* is characterized by frequent micturition, often excited by sneezing, coughing, or other sudden general muscular action, and by more or less constant dribbling. *Spasmodic incontinence* is made evident by frequent micturition occurring at irregular intervals. Paralysis of the compressor muscle may also produce frequent micturition, perhaps constant dribbling, but the bladder becomes hyperdistended from retention, and voluntary muscular effort does not increase the rapidity of the outflow.

The causes of incontinence of urine are general muscular or nervous weakness, disease of the spinal cord, faulty innervation of the bladder, excessive distention from obstruction or polyuria, cystitis, vesical calculi, and irritation of the genital tract or rectum.

Enuresis is normal in infants; it frequently occurs in young children, especially at night, *nocturnal enuresis*, and occasionally also during the day. Such children are sometimes pale, often excitable, but not infrequently free from other disturbances. The escape of urine oftenest takes place during the early part of the night, the child usually being unaware of its passage. The inability to retain the water may be due to the habit of freely drinking milk or water before going to bed, to local irritation, as from worms in the rectum, to a vesical calculus, to concentrated or saccharine urine, to hyperæsthesia of the neck of the bladder, to peripheral irritation from phimosis or a narrow meatus, or to reflex irritation, as teething. It is to be remembered that wetting the bed may be the sole evidence of an epileptic fit during the night, and that it may be an early symptom of organic disease of the brain or cord.

TREATMENT.—Functional nocturnal enuresis usually in the end yields to increase of age and of general bodily strength. The treatment should be primarily directed to the strengthening of the general system by tonics, by out-door exercise, and especially by strychnine; if the urine be acid and irritant, alkalies may be employed. Tincture of belladonna given in as large doses as can be borne in the latter part of the day is probably the most efficacious remedy. The child should avoid drinking in the evening, and should always be taken up at ten o'clock to empty the bladder: punishment is cruel.

NEURALGIA OF THE BLADDER. IRRITABLE BLADDER.

Frequent and painful micturition results from hyperæsthesia of the neck of the bladder, which may be a symptom of organic disease, as inflammation or calculus, or may exist without apparent cause, constituting irritable bladder. The hyperæsthesia of the neck of the bladder is then attributable to an enfeebled nervous system from faulty hygienic surroundings, digestive disturbances, sexual excess, and uterine or ovarian disease. The frequency of micturition is accompanied with tenesmus, an insuperable desire to pass water. There is usually pain at the end of micturition, and a sense of discomfort in the perineum or symphysis before and after micturition. When the pain is due to spasm of both compressor and sphincter muscles it may be so intense as to cause symptoms of collapse, the skin being covered with a cold sweat and the pulse quickened and enfeebled. The passage of a sound produces distress when the hyperæsthetic region is reached. Patients suffering from an irritable bladder are usually thin and pale, and complain of headache, backache, and physical weakness. They are often irritable or depressed, and suffer readily from imaginary evils.

TREATMENT.—In most cases of irritable bladder attention should be chiefly directed to the relief of the neurasthenia which is at the base of the complaint. Not rarely extremely acid urine or local disease exists, and should be appropriately remedied. Suppositories of extract of belladonna or opium are efficient in violent paroxysms, and may, if necessary, be aided by local hot baths. Belladonna given continuously may be of service, and sometimes treatment as for a mild cystitis is judicious.

CYSTITIS. INFLAMMATION OF THE BLADDER.

ETIOLOGY.—The causes of inflammation of the bladder are both general and local, although such a distinction is not always to be absolutely made. Most important among the former are the infectious diseases, especially typhoid fever, acute articular rheumatism, pyæmia and septicæmia, erysipelas, influenza, mumps, scarlet fever, and small-pox, in which slight degrees of cystitis are frequent. In these diseases also the milder varieties of acute nephritis are common, and the inflammation of the bladder, like the nephritis, is probably the result of the local action of the bacteria or toxins demonstrably or presumably concerned in the origin and progress of these diseases. The frequent association of cystitis and gout is most satisfactorily explained as the result of a direct irritation by the concentrated urine of the mucous membrane of the bladder. The more purely local causes are injuries to the bladder, which may result from the use of instruments or of irritating urethral injections, or from the pressure of fæces in the rectum, of pessaries in the vagina, or of the fœtal head at childbirth. Also important among the local causes are foreign bodies, including calculi and invading bacteria, especially the gonococcus. Certain medicinal agents, as cantharides, copaiba, cubebs, and mustard, when absorbed and eliminated by the kidneys, may produce a cystitis. Retention of urine, whether induced by stricture, prostatic enlargement, or vesical tumors, or by defective muscular contraction, as in paraplegia, is capable of exciting a cystitis. Inflammation of the bladder may also be occasioned by the extension of an inflammatory process from neighboring parts, as the urethra, rectum, uterus, vagina, or peritoneum, as is illustrated in the use of an unclean catheter.

MORBID ANATOMY.—The anatomical changes to be found are either characteristic of a catarrhal inflammation or are indicative of a pseudomembranous or a phlegmonous process. In acute catarrhal cystitis the mucous membrane is reddened and swollen and the contents of the bladder are either slimy or purulent, in accordance with which differences a cystitis is regarded as catarrhal or suppurative. In chronic cystitis the mucous membrane is of a bluish slate color in spots, and the contents of the bladder are more slimy than purulent. The pseudomembranous cystitis is characterized either by the presence of fibrinous clots, or more frequently by ecchymoses, ulcerations, and superficial necroses of the mucous membrane, diphtheritic cystitis. These necroses appear as opaque gray or yellow patches, especially at the neck of the bladder and upon projecting folds of mucous membrane, and may contain urinary salts. In phlegmonous cystitis the submucous tissue is destroyed, and the mucous membrane may be detached in shreds or flakes, or even be exfoliated as a cast of the interior of the bladder.

SYMPTOMS.—The earliest as well as the most distressing and persistent symptom of inflammation of the bladder is pain. This may be preceded by a chill and fever, and the latter may last for some time during the progress of the acute inflammation. The pain is usually referred to the region of the symphysis pubis, but may extend to the perineum and the rectum, and is somewhat relieved by micturition. More severe and distressing is the frequently associated vesical tenesmus, when intense called strangury, compelling frequent micturition, perhaps every few minutes, at the end of which a few drops of blood may escape. The urine is opaque, high-colored, and acid or alkaline. At the outset it may be free from albumin, although later albumin occurs in consequence of the presence of blood or pus. A grayish sediment, the so-called mucous cloud, is formed, in which are particles of slime, giving the reaction of nucleoalbumin (mucin), and numerous polynuclear leukocytes, cells of vesical epithelium, occasional red blood-corpuscles, and often abundant bacteria. If the urine is alkaline the precipitate usually contains amorphous phosphates, crystalline triple phosphates, and ammonium urate. In the severer forms of acute cystitis blood and albumin are abundant, and clots of fibrin or shreds of tissue may be present. The presence of casts and a higher percentage of albumin than is accounted for by the presence of pus and blood are indicative of a complicating inflammation of the kidney.

In the milder varieties of acute cystitis the fever subsides in the course of a few days. Vesical pain and tenesmus gradually disappear, and the urine becomes normal. In chronic catarrhal cystitis the vesical pain and tenesmus may be comparatively slight. The opacity of the urine becomes greater and the sediment more abundant, containing larger numbers of pus-corpuscles and a correspondingly increased amount of albumin. The urine is usually alkaline, and the pus is often transformed into a gelatinous mass, which adheres to the vessel in which it is contained. Digestive disturbances, with slight loss of flesh and strength, often result from chronic catarrhal cystitis.

The severer forms of acute cystitis may be such from the outset, or may be due to an acute exacerbation in chronic cystitis, and usually represent the result of a diphtheritic or gangrenous inflammation of the mucous membrane, or the extension of the inflammation to the subperitoneal and paracystic fibrous tissue. The severity of the symptoms may be also due to a complicating pyelonephritis. The febrile disturbance is greater, the course is irregular, and the range of temperature is higher, with frequent wide daily variations between the extremes. The patient may be delirious, somnolent, or in a condition of stupor. The formation of abscesses is indicated by localized induration, pain, and tenderness, often apparent on rectal examination. The abscess may be evacuated into the bladder, with relief to the pain and discomfort, or may extend towards the peritoneum, with the production of a peritonitis. Sloughs of mucous membrane may plug the urethra, so that in the female they may be withdrawn by forceps. With the continuance of the severe symptoms the patient may collapse, the temperature being subnormal and the pulse inappreciable. Englisch and, recently, Paul Thorndike have called especial attention to the occurrence of prevesical inflammation in the space defined by Retzius as in part the result of cystitis, the characteristic localizing symptom being a sharply defined, usually symmetrical tumor above the symphysis, terminating in suppuration, although sometimes undergoing resolution.

Diagnosis.—Vesical pain and tenesmus suggest inflammation of the bladder, and the diagnosis is confirmed by examination of the urine.

PROGNOSIS.—The longer the continuance of the cystitis the more doubtful is the prognosis. Recovery readily takes place in the milder varieties of acute catarrhal cystitis, whereas the prognosis becomes grave if the cystitis extends towards the kidney or to the neighboring fibrous tissue. The prognosis in chronic cystitis is always serious, from the frequent impossibility of removing the cause, and from the liability to acute exacerbations.

TREATMENT.—Whenever from the existence of disease of the spinal cord, or from other cause, there is reason to fear the development of cystitis, the greatest care should be exercised to remove, if possible, the existing cause. Before beginning an habitual catheterization, boric acid or salol may be exhibited, so as partially to sterilize the urine. The catheters should be preferably of rubber, and should be kept in a bichloride solution, and washed in hot water after use; as the catheter goes through the urethra a solution of bichloride, 1 to 4000, should be sent through it so as to disinfect the urethra. The bladder should then be washed out with a strong solution of common salt (a large tablespoonful to a quart), and afterwards followed by a solution one-fourth as strong. After a time, when catheterization is daily practised, the tissues become so hardened and difficult of infection that absolute asepsis as to the catheter is all that is required.

In acute cystitis the patient should be put to bed with the hips slightly elevated and the knees bent over a pillow, and given a mild, non-irritating diet from which all spices and alcoholic drinks are assiduously excluded. When the symptoms are severe, absolute milk diet should be insisted upon. In order to render the urine as little irritating as possible, water should be taken very freely between meals, in the form either of the natural mineral water or of medicated waters, the selection being in accordance with the character of the urine: if this be ammoniacal, boric or benzoic acid may be given; if it be irritatingly acid, alkalies should be used. When there is a large amount of uric acid, especially if there is gravel, formula 4 may be employed. In very severe asthenic cases, especially when there are excessive tenesmus and irritation of the neck of the bladder, the perineum should be freely leeched. The warm sitz-bath, or fomentations with hot water, often are very useful: poultices are uncleanly, and have no special advantage. Hot-water enemata sometimes do good, and should always be used where there is constipation. If such local applications fail to give relief, opium suppositories, or preferably suppositories containing extract of opium (one grain) and extract of belladonna (one-quarter grain), may be tried. Rarely hypodermic injections of morphine are necessary. Opiates are to be avoided if possible, and belladonna given by the mouth may be all that is required. Various drugs whose active principles are eliminated by the urine have been employed for the purpose of affecting the mucous membrane of the bladder. They should always be given in dilute solution. The older of these remedies are uva ursi (fluid extract one teaspoonful every three hours), or arbutin, its active principle (fifty to eighty grains a day), buchu (fluid extract one teaspoonful every three hours), triticum repens (fluid extract one teaspoonful every three hours; very little value). Salol, or preferably sodium salicylate, is often given, forty to fifty grains a day; potassium chlorate is especially commended by Strümpell, twenty grains of it dissolved in at least six ounces of water being administered three to four times in the twenty-four hours.

When cystitis becomes subacute or chronic the stimulating diuretics are often serviceable: they are oil of cubeb, oil of copaiba, oil of sandal wood, terebene, and even oil of turpentine. In acute inflammatory conditions of the bladder these remedies are harmful.

If in acute cystitis relief be not obtained in from twenty-four to forty-eight hours, the bladder should be washed out. Sometimes simple sterilized warm water suffices. In our experience a solution of boric acid (ten grains to the ounce to four per cent. (saturated) solution) has been especially efficient, but other substances (see Chronic Cystitis) may be used. In chronic cystitis, after a careful examination of the parts, and the removal of any strictures, calculi, or other removable cause of irritation of the bladder, the chief reliance must be placed upon local irrigation. Boric acid, or corrosive sublimate 1 to 15,000, or plumbic acetate 1 to 1000, or tannic acid 1 to 300, or carbolic acid 1 to 500, or alum 1 to 2000, may be employed. Silver nitrate, in one-half to two per cent. solution, is, according to the opinion of various surgeons, the most generally efficacious of all the local applications. In many cases, however, it produces great pain, and it should therefore be first used in small quantities and in the weakest solution; but it should be tried persistently. In washing out the bladder it is better never fully to distend the viscus; when the soft rubber catheter has reached the bulbo-membranous portion of the urethra, sterilized water should be sent through it by means of a fountain syringe and allowed to flow back, so as to wash out the urethra. The catheter should then be passed into the bladder, and from one to two ounces of fluid injected and afterwards withdrawn. About the same quantity should be injected various times until the viscus is thoroughly cleansed. To prevent absorption, the final washing should be with simple sterilized water. We have seen serious poisoning from the use of the saturated solution of boric acid. In most cases the character of the medicated solution should be varied from time to time.

When a cystitis depends upon spinal paralysis, very little can be achieved by any local treatment. As the urine is always ammoniacal, boric, benzoic, and other acids given by the mouth are often very useful. Sodium hyposulphite in half-drachm doses has been especially recommended in hopeless cases. Draining the bladder through a suprapubic or peroneal opening, as practised by some surgeons, is justified only when the cystitis is threatening life or is due to local obstruction.

FORMULARY.

THE following formulæ are given largely as examples to medical students and young graduates to aid them before they have thoroughly acquired the art of extemporaneous prescription writing. They are not to be slavishly followed, but to be altered, modified, or entirely substituted by other combinations to suit the individual case. With very few exceptions they have, however, been much used in practice, and we believe are trustworthy as practical working prescriptions.

No. 1.

℞ Tr. guaiaci ammoniatæ, fʒvi;
Tr. ferri chloridi, fʒiss;
Tr. cantharidis, ♏xlviii;
Vini aloes, fʒvi;
Alcoholis, q. s. ad f℥iii.
M. S.—Teaspoonful after meals in milk.

The proportions of this formula must be modified for the individual case. The aloes should be increased or lessened, as may be required, until the patient has one or two softish stools each day. There is also much difference in the irritability of the bladder in relation to cantharides.

No. 2.

℞ Sodii sulphatis,
Magnesii sulphatis, āā ʒiii;
Aquæ, f℥vi.
Misce.
S.—Half to one ounce three or four times a day in a glass of water.

No. 3.

℞ Sodii sulphatis,
Magnesii sulphatis, āā ʒiii;
Ferri sulphatis, gr. viii;
Aquæ, f℥vi.
Misce.
S.—Half to one ounce in water three or four times a day in a glass of water.

No. 4.

℞ Lithii benzoatis,
Lithii bicarbonatis, āā gr. xv;
Potassii bicarbonatis, gr. xx;
Aquæ acidi carbonici, Oi.
Misce et dispensa in siphone.
S.—Two pints daily.

No. 5.

℞ Bismuthi subcarbonatis, ʒiii;
Acidi carbolici, gr. xviii.
Misce et dispensa in capsulis xxiv.
S.—One or two every one to three hours, pro re nata.

In making these capsules it is essential for the ingredients to be thoroughly incorporated before being put in the capsules, which should also be kept well covered with some dry powder like lycopodium. In certain cases a mixture is preferred as follows:

℞ Acidi carbolici, gr. xviii;
Bismuthi subnitratis, ʒiii;
Glycerini, f℥ss;
Aquæ, f℥iiss.
Misce.
S.—Shake thoroughly to complete mixture. Dose, dessertspoonful in water.

No. 6.

℞ Acidi sulphurici aromatici, fʒii;
Ext. hæmatoxyli, ʒiii;
Syr. zingiberis, f℥iss.
Misce et adde
Tr. opii camphoratæ, f℥iss.
S.—Dessertspoonful in water.

In many cases it is well to diminish the amount of paregoric contained in this prescription, increasing proportionately the ginger syrup.

No. 7.

℞ Antipyrini, gr. xv;
Pilocarpinæ hydrochloratis, gr. ss;
Tr. aconiti, gtt. viii;
Aquæ, f℥iss.
Misce.
S.—Take a tablespoonful, immediately followed by a hot general or foot bath (ten minutes); then, the patient being covered in bed, one dessertspoonful in a tumbler of hot toddy, repeated, if no sweating occur, in twenty minutes. When there is pain, if morphine does not disagree with the patient, one-sixth of a grain may be added to the mixture.

No. 8.

℞ Misturæ cretæ, f℥iii;
Tr. kino,
Tr. cinnamomi,
Tr. opii camphoratæ, āā f℥i.
Misce.
S.—Tablespoonful in water, pro re nata.

No. 9.

℞ Spiritus camphoræ, f℥ss;
Olei caryophylli, ♏xxx;
Chloroformi, fʒiii;
Tr. opii deodoratæ, fʒii;
Tr. capsici, fʒii.
Misce.

S.—Shake well. Thirty to forty drops in water every half-hour to two hours, pro re nata.

No. 10.

℞ Tincturæ ferri chloridi, fʒii;
Hydrargyri chloridi corrosivi, gr. ii;
Glycerini, fʒix;
Aquæ, f℥ii.
Misce.

S.—Teaspoonful in water after meals.

The amount of corrosive sublimate in this prescription may be increased or diminished according to the needs of the individual case.

No. 11.

℞ Potassii iodidi, ℥ss;
Syr. sarsaparillæ comp.,
Ext. sarsaparillæ fld. comp., āā f℥iii.
Misce.

S.—Teaspoonful to tablespoonful in water as directed.

To the above formula corrosive sublimate may be added as desired; no precipitate will be formed.

No. 12.

℞ Bismuthi subnitratis, ʒi;
Antipyrini, gr. x;
Cocainæ hydrochloratis, gr. iii;
Mucil. acaciæ,
Aquæ, āā f℥ss.
Misce.

S.—Shake well. Throw, by means of an ordinary dropper, about five minims into each nostril every two or three hours.

No. 13.

Allen's Antiseptic Nasal Wash.

℞ Acidi benzoici,
Sodii boratis, āā gr. lxxxv;
Acidi salicylici, gr. xx;
Acidi borici, ʒiv;
Thymoli, gr. xvi;
Eucalyptoli, gtt. viii;
Ol. gaultheriæ, gtt. viii;
Mentholi, gr. v;
Ol. pini, gtt. viii;
Glycerini, ʒv, ♏xx;
Alcoholis, ℥iv;
Aquæ destill., q. s. ad ℥xvi.
Misce.

S.—Add a teaspoonful to four ounces of water, and use as a spray or wash.

No. 14.

Seiler's Antiseptic Nasal Wash.

℞ Sodii bicarbonatis, ℥i;
Sodii boratis, ℥i;
Sodii benzoatis,
Sodii salicylatis, āā gr. xx;
Thymoli,
Eucalyptoli, āā gr. x;
Mentholi, gr. v;
Ol. gaultheriæ, gtt. vi;
Glycerini, ℥viiiss;
Alcoholis, ℥ii;
Aquæ, q. s. ad Oxvi.
Misce.

This wash is very stimulating to the nasal mucous membrane, and often needs dilution.

For practical purposes it is often much more convenient to have this solution in the form of tablets. All that is necessary for this purpose is to omit the glycerin, alcohol, and water from the above prescription, and divide the other ingredients, after thorough incorporation, into one hundred and twenty-eight tablets, one of which dissolved in two ounces of water will give a solution practically of the strength of that of the formula.

Another alkaline wash much used for cleansing the nose is *Dobell's solution*, which is composed of—

℞ Sodii boratis,
Sodii bicarbonatis, āā ʒi;
Acidi carbolici, gr. xxx;
Glycerini, f℥i;
Aquæ, Oii.
Misce.

No. 15.

A powder for use by asthmatics may be made by taking out all the pieces of stems and leaf-stems from stramonium, so as to use only the leaves themselves, powdering these sufficiently fine to go through a No. 8 sieve, and then saturating thoroughly this powder with a saturated solution of potassium nitrate, and drying.

No. 16.

℞ Potassii citratis, ℥i;
Succi limonis, f℥iss;
Syr. ipecacuanhæ, f℥ss;
Syrupi, f℥i.
Misce.

S.—Dessertspoonful every two hours.

In this formula one grain of apomorphine hydrochlorate, or, if the patient be robust, half to one grain of tartar emetic, may be substituted for the ipecacuanha, the syrup being increased to an ounce and a half.

No. 17.

℞ Ammonii chloridi,
Ext. glycyrrhizæ, āā ʒii;
Mucil. acaciæ, f℥iss;
Aquæ, f℥iiss.
Misce.
S.—Dessertspoonful every two hours.

A simple solution of the ammonium salt in water is preferred by many patients, and is often more acceptable to the stomach.

No. 18.

℞ Belladonnæ fol., gr. xcvi;
Hyoscyami fol.,
Stramonii fol., āā gr. xlviii;
Ext. opii, gr. iv;
Tabaci, gr. lxxx;
Aquæ, Oi.
M., ft. sol. et adde
Potassii nitratis, gr. clx;
Potassii arsenitis, gr. cccxx.
Saturate bibulous paper and dry for use.
S.—Paper is rolled into cigarettes, one of which is smoked until relief is afforded or some giddiness produced.

No. 19.

℞ Sodii phosphatis, ℥iii;
Sodii sulphatis, ℥i;
Potassii iodidi, ʒi.
Misce et fiat pulvis subtilissimus.
M. S.—Teaspoonful to tablespoonful as required.

No. 20.

℞ Pulveris sennæ, ℥ii;
Pulveris zingiberis, ℥i;
Pulveris aloes, ʒii.
Misce.
S.—Put in a pint of whiskey; agitate frequently, and after three days take the clear liquid at bedtime, thirty to sixty drops, more or less, as required to produce a free fæcal discharge.

If more convenient, the druggist can be directed to exhaust the powder with a pint of dilute alcohol.

No. 21.

℞ Ext. rhois glabræ fld., f℥iii;
Pulv. potassii chloratis, ʒiss;
Glycerini, f℥ss.
S.—Shake well. One to three teaspoonfuls to a wineglassful of water as gargle.

No. 22.

℞ Picis liquidæ, f℥iii;
Triturentur cum liquore calcis, Oviii, ad saturationem, et percolentur per prunum virginianam, ℥viii.
S.—Wineglassful one to two hours after each meal.

No. 23.

℞ Acidi nitrohydrochlorici, fʒii;
Aquæ, f℥iss;
Strychninæ sulph., gr. i.
Misce et adde
Tr. gentianæ comp.,
Tr. cardamomi comp., āā q. s. ad f℥vi.
S.—Dessertspoonful after meals in water.

This solution is a very elegant stomachic tonic, which can be used in cases of general debility with failure of appetite and digestion occurring during convalescence, and at other times.

When there is a tendency to diarrhœa, and no hepatic stimulation is required, hydrochloric acid may be substituted for nitrohydrochloric; when there is a distinct tendency to diarrhœa, the following modification will often be found very useful:

℞ Acidi sulphurici diluti, fʒii;
Syr. zingiberis, fʒi;
Strychninæ sulph., gr. i.
Misce et adde
Tr. gentianæ comp., f℥ii;
Tr. cinnamomi, f℥iii.
S.—Dessertspoonful after meals in water.

No. 24.

℞ Glycerini,
Olei ricini, āā f℥i;
Olei caryophylli, gtt. iv.
Misce.
S.—Shake thoroughly. Tablespoonful contains dessertspoonful of castor oil.

If this mixture, after it is shaken to thorough homogeneity, be put into an ice-cold spoon or ice-cold water, it will congeal into a very thick mass which can be swallowed without disgust.

No. 25.

℞ Elaterini, gr. ss;
Ext. belladonnæ, gr. i;
Olei caryophylli, gtt. x.
Misce et fiant pilulæ vi.
S.—One every four to six hours until purgation occurs.

No. 26.

℞ Acidi hydrocyanici dil., gtt. xxiv;
Cocainæ hydrochloratis, gr. iv;
Elixir. aromatici, f℥iss.
S.—Teaspoonful, repeated in forty minutes if needed.

No. 27.

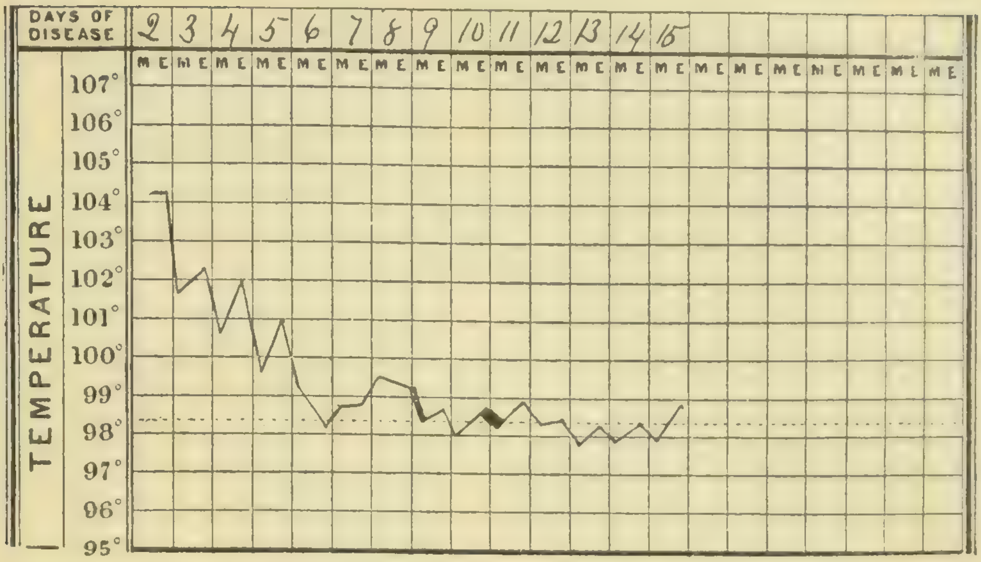
℞ Strychninæ sulph., gr. i;
Ext. quassiæ, gr. xxiv;
Oleoresinæ capsici, gr. iv;
Olei caryophylli, gtt. xxiv.
Misce et fiat massa in capsulas xxiv dividenda.
S.—One directly after meals.

No. 28.

℞ Strontii salicylatis, ʒii;
Strychninæ sulph., gr. i;
Naphtoli, gr. xxiv:
Acidi carbolici, gr. xviii.
Misce et fiat massa in capsulas xxiv dividenda.
S.—One directly after meals.

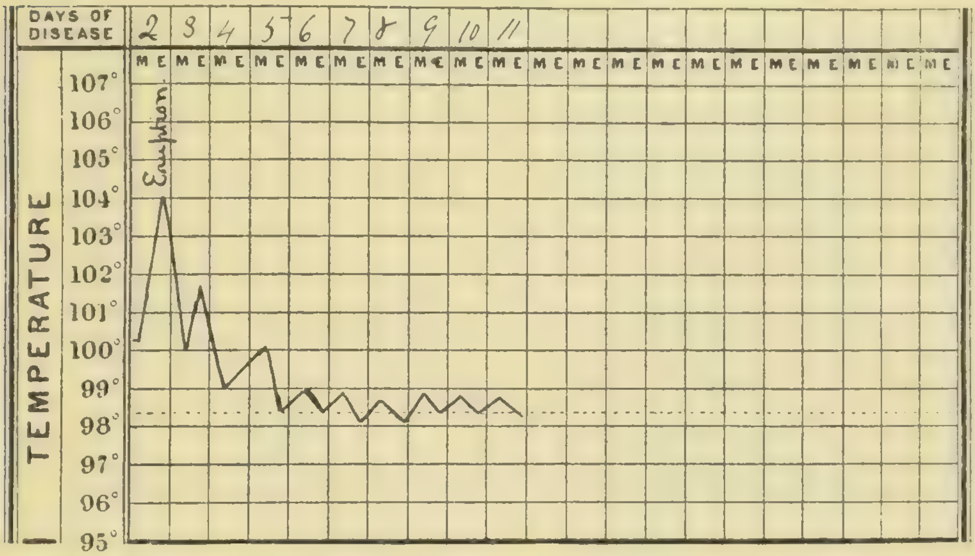
CHARTS OF TEMPERATURE.

Chart I.



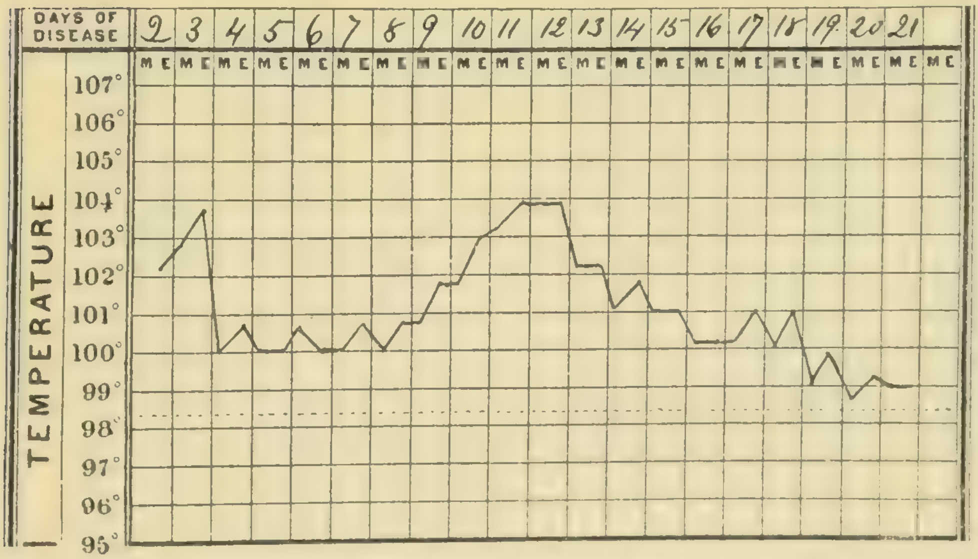
Scarlet Fever. (Boston City Hospital, Department for Contagious Diseases.)

Chart II.



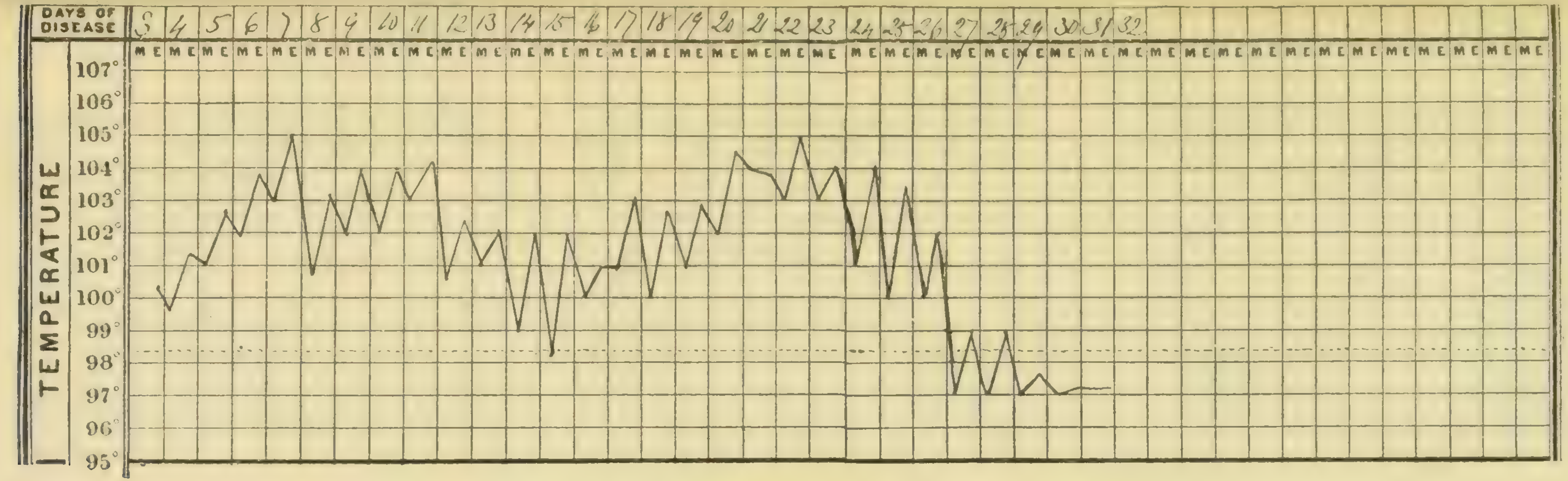
Measles. (Boston City Hospital, Department for Contagious Diseases.)

Chart III.



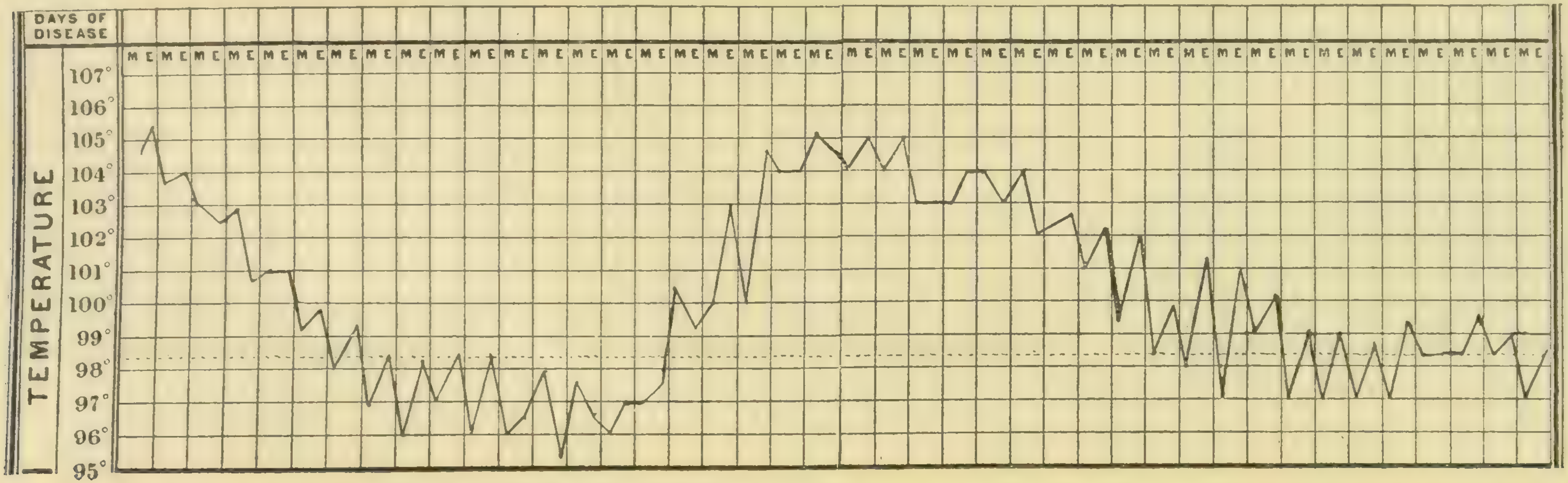
Small-Pox. (Boston City Hospital, Department for Contagious Diseases.)

CHART IV.



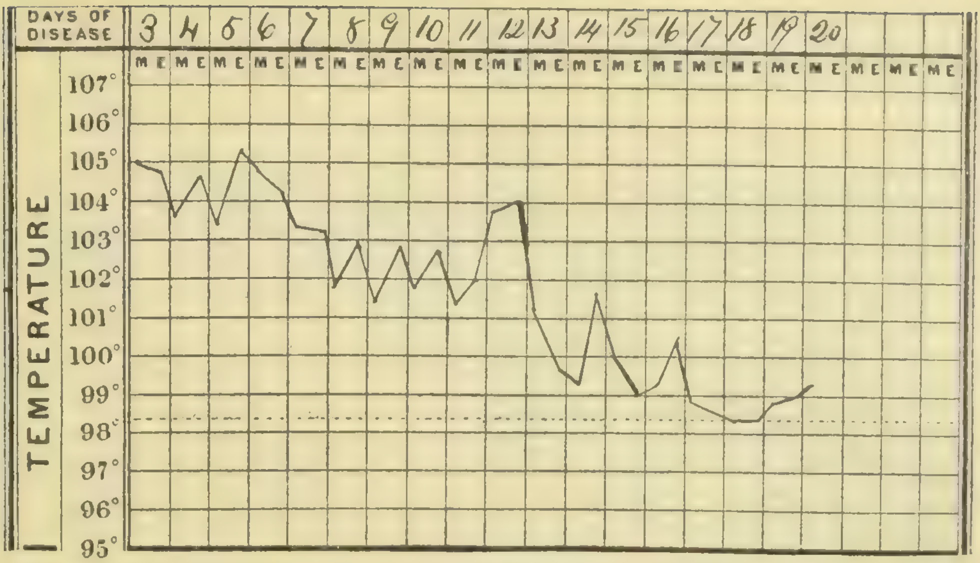
Typhoid Fever. (Massachusetts General Hospital.)

CHART V.



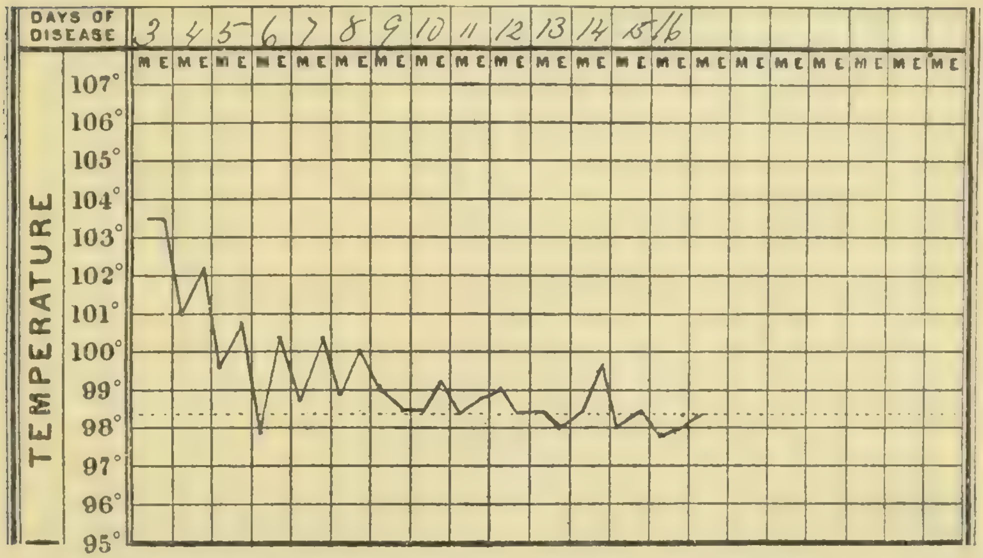
Typhoid Fever, defervescence and relapse. (Massachusetts General Hospital.)

CHART VI.



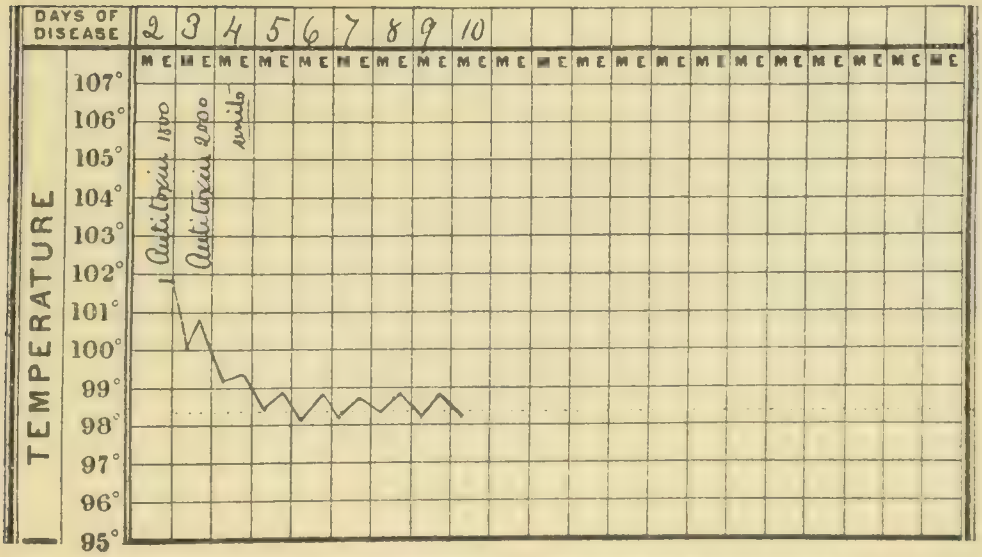
Typhus Fever. (Murchison.)

CHART VII.



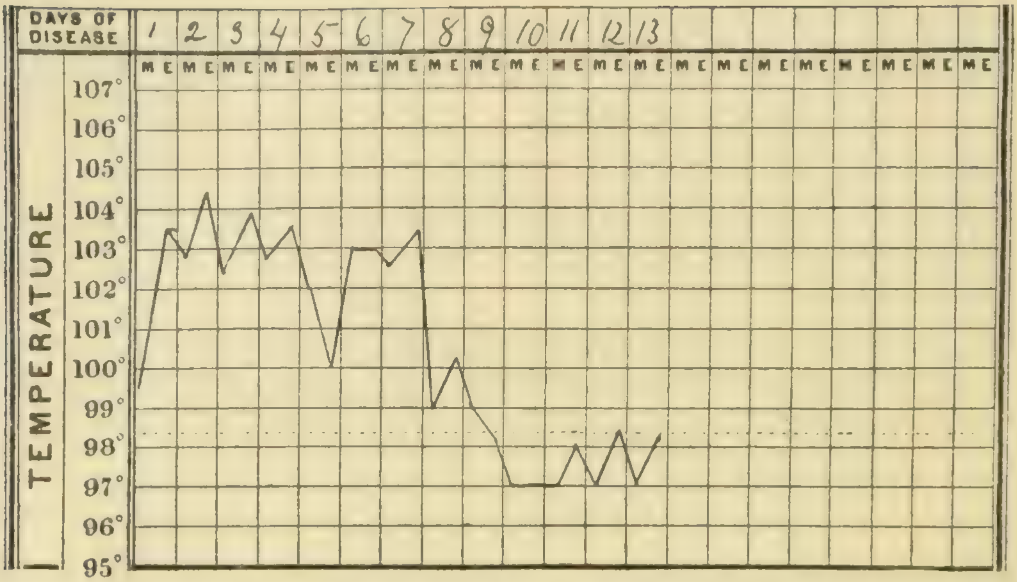
Diphtheria, without antitoxin. (Boston City Hospital.)

CHART VIII.



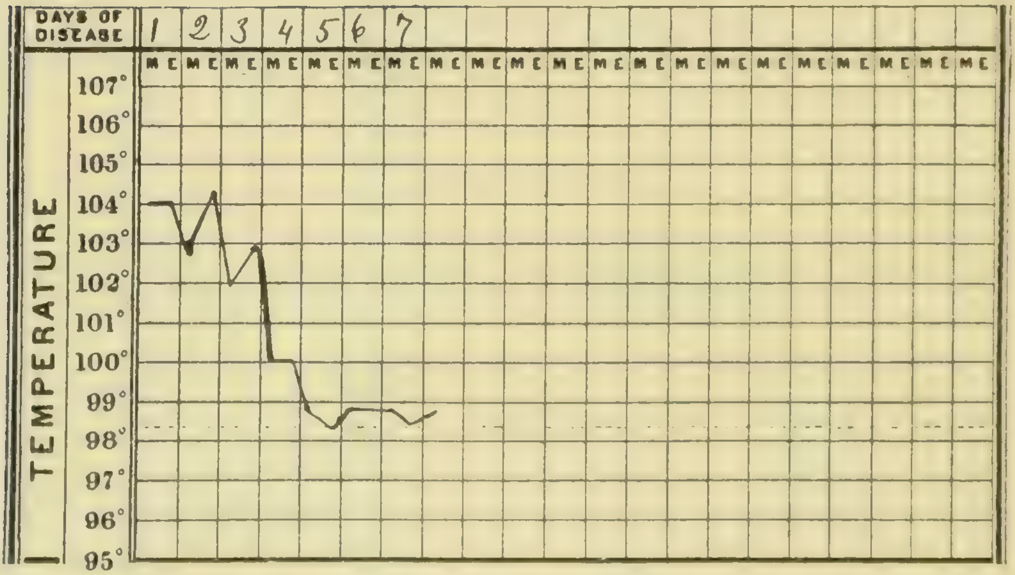
Diphtheria, with antitoxin. (Boston City Hospital.)

CHART IX.



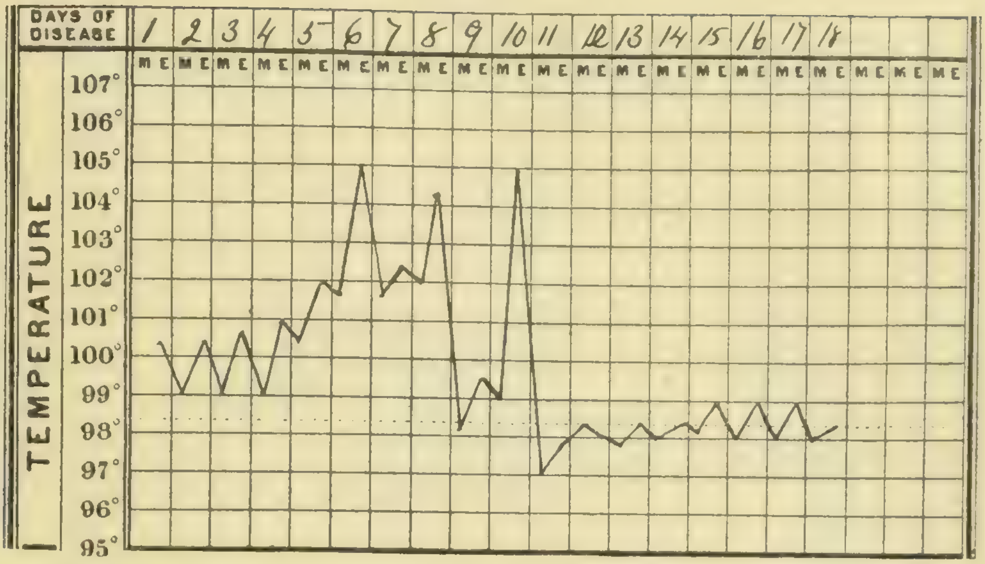
Pneumonia, pseudo-crisis on the fifth day. (Massachusetts General Hospital.)

CHART X.



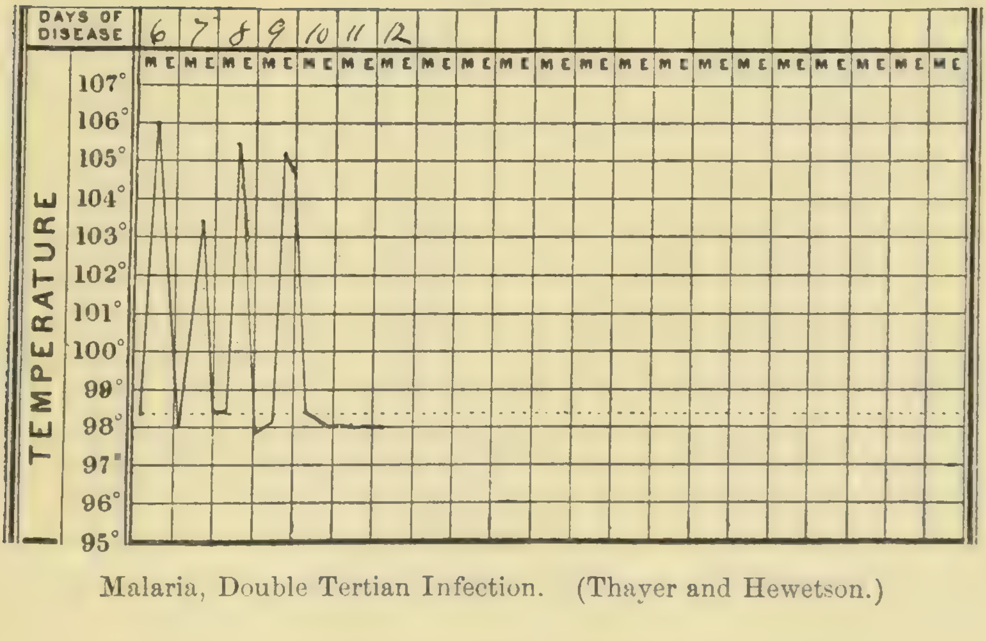
Yellow Fever. (Bemiss.)

Chart XI.



Malaria, Tertian Fever during convalescence from ovariotomy. (Massachusetts General Hospital.)

Chart XII.



Malaria, Double Tertian Infection. (Thayer and Hewetson.)

INDEX.

A.

- Abdominal aorta, aneurism of, 696.
  - dropsy, 967.
- Abscess, iliac, 876.
  - of brain, 510. (*Vide* Suppurative Encephalitis.)
  - of kidney, 1040.
  - of liver, 918.
  - of spleen, 30.
  - paranephritic, 1051.
  - perinephritic, 1051.
  - pulmonary, 766.
  - retropharyngeal, 799.
  - spinal, 547.
  - subphrenic, 914.
- Abulia, 383.
- Acarus scabiei, 346.
- Acetonuria, 1012.
- Achromatopsia in hysteria, 409.
- Aconite, poisoning by, 358.
- Acoria, 849.
- Acromegaly, 626.
- Acro-paræsthesia, 623.
- Actinomyces, 231.
- Actinomycosis, 231.
- Acute articular rheumatism, 61.
  - ataxia, 554.
  - bronchitis, 712.
  - infectious jaundice, 151.
  - polymyositis, 44.
  - tuberculosis, 244.
- Addison's disease, 41.
- Adénie, 16.
- Adenitis, tubercular, 281.
- Adiposuria, 1000.
- Adrenal glands, diseases of, 41.
- Aerophagy, 850.
- Æstivo-autumnal fever, 207.
- African hypnosis, 463.
  - sleeping disease, 463.
- Ageusia, 477.
- Aglyphia, 484.
- Agraphia, 484.
  - literal, 485.
  - musical, 484.
  - verbal, 485.
- Ague and fever, 201.
- Albuminimeter, 1003.
- Albuminuria, 1001.
  - cystic, 1003.
  - febrile, 1004.
  - functional, 1003.
  - infectious, 1004.
  - in lead poisoning, 365.
    - tests for, 1002.
- Albuminuria, intermittent, 1003.
  - neurotic, 1004.
  - of adolescence, 1003.
  - pathological, 1003.
  - physiological, 1003.
  - transitory, 1003.
- Albumosuria, 1001, 1005.
- Alcohol, acute poisoning by, 351.
  - chronic poisoning by, 368.
- Alcoholic insanity, 373.
- Alcoholism, 368.
- Alexia, 484.
- Algesia, 381.
- Alimentary canal, tuberculosis of, 265.
- Alkaptonuria, 999.
- Allocheiria, 579.
- Alopecia, syphilitic, 308.
- Amaurosis, 479.
- Amblyopia, 479.
- Amentia, 383.
- Amimia, ataxic, 484.
  - sensory, 485.
- Amnesia, periodic, 467.
- Amœba coli, 321.
  - dysenteriæ, 321.
- Amusia, ataxic, 484.
  - sensory, 484.
- Amyloid liver, 928.
- Amyotrophic lateral sclerosis, 588.
- Anæmia, 2.
  - cerebral, 492.
  - essential, 9.
  - idiopathic, 9.
  - lymphatic, 16.
  - of spinal cord, 545.
  - pernicious, 9.
    - progressive, 9.
  - secondary, 3.
  - simple, 2.
  - spinal, 409, 545.
  - splenic, 16.
  - tropical, 3.
- Anæsthesia, 382.
  - psychical, 391.
- Analgesia, 712.
- Anarthria, 484.
- Anatomist's tubercle, 250.
- Anchylostomum duodenale, 9.
- Aneurism, 691.
  - arterio-venous, 691.
  - by anastomosis, 691.
  - cirsoid, 692.
  - dissecting, 691.
- Aneurism, false, 691.
  - morbid anatomy, 692.
  - nodular periarteritis in, 692.
  - of abdominal aorta, 696.
  - of aorta, 693.
    - diagnosis, 693.
    - physical examination in, 694.
    - prognosis, 696.
    - treatment, 697.
  - of heart, 648.
  - serpentine, 692.
  - symptoms, 692.
  - tracheal tugging in, 695.
  - treatment, 697.
  - true, 691.
  - varicose, 691.
- Aneurisms, cerebral, 497.
- Angina, 797.
  - Ludovici, 187.
  - pectoris, 682.
    - diagnosis, 683.
    - prognosis, 683.
    - treatment, 684.
  - vaso-motor, 682.
- Angioneurotic œdema, 622.
- Animal parasites, 321.
- Ankylostoma duodenale, 339.
- Ankylostomiasis, 339.
- Anorexia, 849.
- Anterior lobe, tumors of, 525.
- Antero-lateral sclerosis, 585.
- Anthracosis of lung, 756.
- Anthrax, 235.
    - diagnosis, 236.
    - prognosis, 236.
    - treatment, 237.
  - bacillus of, 235.
  - internal, 236.
  - malignant, 236.
  - œdema, 236.
- Antimonial poisoning, 360, 368.
- Antiphthisin, 241.
- Antitoxin of tetanus, 200.
  - treatment of diphtheria, 177.
- Anuria, 1015.
  - in hysteria, 410.
- Aorta, aneurism of, 693.
- Aortic insufficiency, 665.
  - stenosis, 666.
- Aphasia, 484.
  - ataxic, 484.
  - cortical localization of, 486.
  - motor, 484.
  - sensory, 484.
  - subcortical, 487.
  - transcortical, 487.

Appendicitis, 876.
  abscess in, 878, 882.
    diagnosis, 883.
    etiology, 876.
    induration in, 880.
    morbid anatomy, 877.
    mortality of, 884.
    perforation in, 878.
    peritonitis in, 878, 882.
    prognosis, 884.
    surgical treatment, 889.
    symptoms, 879.
    termination of, 881.
    treatment, 887.
  chronic, 885.
  recurrent, 885.
  relapsing, 885.
Apoplexy, 498 (*note*).
  serous, 493.
Argyll-Robertson pupil, 478, 580.
Arithromania, 386.
Arsenical poisoning, 360, 367.
Apraxia, 487.
Arachnitis, 489.
Arhythmia, 681.
Arteries, alterations of, in nephritis, 1018.
  atheromatous degeneration of, 686.
  diseases of, 686.
  syphilis of, 311.
Arterio-capillary fibrosis, 1019.
Arterio-sclerosis, 686.
  diagnosis, 690.
  morbid anatomy, 687.
  prognosis, 690.
  pseudo-cartilaginous plates in, 687.
  treatment, 690.
Arthritis deformans, 71.
  rheumatic, chronic, 70.
  rheumatoid, 71.
  uratica, 76.
  urica, 76.
Arthropodes, diseases due to, 345.
Articular rheumatism, acute, 61.
    chronic, 70.
Artificial feeding of infants, 869.
  respiration, 354.
Ascariosis, 337.
Ascaris lumbricoides, 337.
Ascending paralysis, 548.
Ascites, 967. (*Vide* Hydroperitoneum.)
  adipose, 968, 987.
  chylous, 968.
Asiatic cholera, 219.
Asphyxia, local, 620.
Astasia abasia, 415.
Asthma, 725.
  bronchial, 725.
  cardiac, 660, 725.
  diagnosis, 727.
  dyspeptic, 726.
  nervous, 726.
  paroxysms, treatment of, 728.
  prognosis, 727.
  renal, 725.
Asthma, sputum in, 726.
  symptoms, 726.
  thymic, 40, 725.
  thyroid, 725.
  treatment, 727.
  uræmic, 1016.
  uterine, 726.
Ataxia, acute, 554.
  Friedreich's, 589.
  hysterical, 415.
  locomotor, 575.
Ataxic aphasia, 484.
  paraplegia, 588.
Atelectasis, 736.
  acquired, 736.
  congenital, 736.
  fœtal, 736.
Atheroma of arteries, 686.
Athetosis, 380, 483.
  double, 483.
Atrophic myopathy, 48.
Atrophy, muscular neuritic, 615.
Atropine, poisoning by, 355.
Aura epileptica, 420.
Automatic movements, 378.
Automatism, epileptic, 422.
Azoturia, 93.

B.

Bacillus anthracis, 235.
  comma, 220.
  Klebs-Loeffler, 164.
  of anthrax, 235.
  of cholera, 220.
  of diphtheria, 164, 170.
  of erysipelas, 185.
  of leprosy, 301.
  of pneumonia, 741.
  of syphilis, 304.
  of tetanus, 194.
  of typhoid fever, 119, 135.
  of tuberculosis, 239, 241.
    staining of, 256.
  of whooping-cough, 179.
  tussis convulsivæ, 179.
Bacteria in acute endocarditis, 651.
Balantidium coli, 322.
Bantingism, 59.
Barlow's disease, 55.
Basedow's disease, 33.
Bedbug, 348.
Beef tape-worm, 324.
Bell's disease, 513.
  palsy, 611.
Beri-beri, 609.
Biceps jerk, 379.
Big-jaw in cattle, 231.
Bile-ducts, diseases of, 931.
Biliary tract, tumors of, 947.
Bilious fever, 207.
  remittent fever, 207.
  typhoid fever, 151.
Bilirubin in jaundice, 933.
Black measles, 104.
  small-pox, 113.
Bladder, diseases of, 1057.
  inflammation of, 1058.
  irrigation of, 1062.
  irritable, 1058.
Bladder, neuralgia of, 1058.
  tuberculosis of, 283.
Blepharoclonus, 431.
Blepharospasm, 431.
Blood, examination of, in malaria, 209.
Blood-flukes, 335.
Blood-vessels, alterations of, in nephritis, 1018.
  tuberculosis of, 283.
Bone typhoid, 193.
Bones, tuberculosis of, 285.
Bothriocephalus cordatus, 324.
  cristatus, 324.
  latus, 324.
  liguloides, 324.
Brach's symptom, 579.
Bradycardia, 680.
Brain, diseases of, 509.
    of membranes of, 488.
  dropsy of, 246.
  motor centres of, 473.
  softening of, 518.
  tubercle of, 242.
  tuberculosis of, 284.
  tumors of, 524.
Brenzkatechinuria, 999.
Bright's disease, 1022.
    acute, 1023.
      diagnosis, 1025.
      morbid anatomy, 1023.
      prognosis, 1025.
      symptoms, 1023.
      treatment, 1025.
      urine in, 1024.
    second stage, 1028.
      diagnosis, 1030.
      prognosis, 1031.
      symptoms, 1029.
      treatment, 1031.
    third stage, 1033.
      diagnosis, 1036.
      morbid anatomy, 1033.
      prognosis, 1037.
      symptoms, 1034.
      treatment, 1037.
British plague, 118.
Bronchi, diseases of, 712.
Bronchial asthma, 725.
  catarrh, acute, 712.
    chronic, 717.
  obstruction, 723.
Bronchiectasis, 721.
  morbid anatomy, 722.
  symptoms, 722.
  treatment, 723.
Bronchitis, acute, 712.
    diagnosis, 714.
    symptoms, 713.
    treatment, 714.
      of first stage, 715.
      of second stage, 716.
  capillary, 713.
  cheesy, 252.
  chronic, 717.
    symptoms, 718.
    treatment, 719.
  dry, 718.
  epidemic capillary, 104.
  membranous, 720.
  putrid, 718.
Bronchocele, 31.

Broncho-pneumonia, 759.
    diagnosis, 761.
    prognosis, 762.
    symptoms, 760.
    treatment, 762.
  lobular, 760.
  nodular, 759.
Bronchorrhœa, 718.
  serous, 718.
Brow ague, 206, 456.
Bulbar palsy, 537.
Bulimia, 850.
Bursitis, omental, 951.

C.

Cachexia strumipriva, 38.
Caisson disease, 449.
Calabar bean, poisoning by, 357.
Calculi, biliary, 939.
  pancreatic, 955.
  renal, 1053.
Camp-fever, 145.
Cancer of gall-bladder, 948.
  of gall-duct, 948.
  of intestines, 900.
  of liver, 928.
  of lung, 767.
  of œsophagus, 812.
  of pancreas, 960.
  of peritoneum, 986.
  of stomach, 843.
Cancrum oris, 792.
Cantharides, poisoning by, 361.
Carbolic acid, poisoning by, 352.
    antidote, 353.
Cardiac asthma, 660.
  collapse, 330.
  dilatation, 670, 674.
  dropsy, 662.
  epilepsy, 423.
  hypertrophy, 669.
  murmur of aortic valve, 665, 666.
    of mitral valve, 664, 665.
    of pulmonary valve, 668.
    of tricuspid valve, 667.
  neuroses, 678.
  palpitation, 676.
Casts, renal, blood, 1014.
    epithelial, 1014.
    fatty, 1014.
    granular, 1014.
    hyaline, 1013.
    mucous, 1013.
    waxy, 1014.
Catarrh, autumnal, 704.
  bronchial, acute, 712.
    chronic, 717.
  dry, 718.
  gastric, acute, 827.
    chronic, 831.
  nasal, acute, 701.
    chronic, 702.
Cephalodynia, 74.
Cercomonas coli hominis, 322.
  intestinalis, 322.
Cerebellar localization, 487.
Cerebellum, tumors of, 526.
Cerebral anæmia, 492.
  aneurism, 497.
  convulsions, 378.
Cerebral embolism, 494.
  hemorrhage, 498.
    after-history of, 503.
    convulsions in, 501.
    diagnosis, 504.
    paralysis in, 502.
    secondary degeneration in, 500.
    symptomatology, 500.
    temperature in, 501.
    treatment, 506.
    trephining in, 505.
  hyperæmia, 493.
  localization, 471, 481.
  œdema, 493.
  palsy of children, 508.
  rheumatism, 65.
  sinus, thrombosis of, 497.
  syphilis, 528.
  tetanus, 198.
  thrombosis, 493.
Cerebro-spinal meningitis, 152.
    anomalous forms, 155.
    chronic, 155.
    diagnosis, 156.
    malignant form, 155.
    morbid anatomy, 153.
    ordinary type, 154.
    prognosis, 156.
    symptomatology, 153.
    treatment, 156.
  sclerosis, multiple, 522.
Chalicosis of lungs, 756.
Chancre, hard, 306.
Character, 388.
Charcot-crystals, 13.
Charcot-Marie type of muscular atrophy, 616.
Charcot, sensory crossway of, 482.
Charts of temperature, 1067–1071.
Cheesy bronchitis, 252.
  pneumonia, 252.
Cheyne-Stokes breathing, 660.
Chigoe, 348.
Chloral, poisoning by, 351.
Chloroform, poisoning by, 352.
Chloroma, 15.
Chlorosis, 4.
  diagnosis, 6.
  prognosis, 7.
  treatment, 7.
Choked disk, 477.
Cholangitis, 935.
Cholecystenterostomy, 947.
Cholecystitis, 935.
Cholecystotomy, 947.
Choledochitis, 936.
  catarrhal, 936.
Choledochotomy, 947.
Cholelithiasis, 939.
  biliary colic in, 941.
  composition of stone, 940.
  diagnosis, 943.
  impaction of stone, 943.
  jaundice in, 942.
  morbid anatomy, 940.
  prognosis, 944.
  surgical treatment, 946.
  symptoms, 941.
  treatment, 945.
Cholera, Asiatic, 219.
    algid period, 222.
    comma bacillus in, 220.
    diagnosis, 223.
    enteroclysis in, 225.
    intravenous injection in, 225.
    morbid anatomy, 221.
    prognosis, 223.
    prophylaxis, 223.
    stools in, 221.
    symptomatology, 221.
    treatment, 223.
  infantum, 226, 861.
    treatment, 863.
  morbus, 225.
  nostras, 225.
  sicca, 223.
Cholerine, 222.
Chorea, 432. (*Vide* St. Vitus's dance.)
  automatic, 439.
  convulsive, 438.
  electric, 438.
  general, 380.
  Germanorum, 439.
  habit, 438.
  hereditary, 440.
  Huntingdon's, 440.
  hysterical, 438.
  local, 380.
  minor, 432.
  of childhood, 432.
  of pregnancy, 437.
  organic, 438.
  post-hemiplegic, 483.
  pre-hemiplegic, 483.
  reflex, 437.
  rhythmical, 380.
  senile, 438.
Choreic movements, 380.
  tic, 438.
Chylothorax, 774.
Chyluria, 1000.
Cimex lectularius, 348.
Circular insanity, 399.
Cirrhosis of kidney, 1033.
  of liver, 923.
  of lung, 264, 756.
Citric acid, poisoning by, 360.
Clonus, 379.
Cocaine, poisoning by, 355, 356.
Cocainism, 376.
Coccidium oviforme, 321.
Cold in the head, 701.
Colic from lead, 333.
  mucous, 870.
Colica pictonum, 362.
Colitis, pseudo-membranous, 870.
Colles's law in syphilis, 304.
Coloptosis, 855.
Coma, 460.
  in cerebral syphilis, 534.
Combined sclerosis, 588.
Comma bacillus in cholera, 220.
Compression myelitis, 558.
Confusional insanity, 395.
Congenital myotonia, 49.
Congestive malarial fever, 208.
Conium, poisoning by, 358.
Consciousness, double, 467.

- Constipation, 903.
  - diagnosis, 905.
  - symptoms, 904.
  - treatment, 905.
- Constitutional diseases, 41.
- Consumption, pulmonary, 251.
- Contractions, paradoxical, 379.
- Convulsions, 378, 430.
  - cerebral, 378.
  - epileptiform, 378.
  - hysterical, 378.
  - in epilepsy, 419.
  - spinal, 378.
  - tetanic, 378.
- Convulsive tic, 431.
- Coördination, disturbances of, 380.
  - station test for, 380.
- Coprolalia, 440.
- Corpora quadrigemina, tumors of, 526.
- Corrigan's pulse, 666.
- Corrosive sublimate, poisoning by, 300.
- Coryza, 703.
- Costiveness, 903.
- Cough, treatment of, 716.
- Cow-pox, 116.
- Cramp, writer's, 455.
- Craniotabes, 524.
  - in rickets, 52.
- Cranium, tumors within, 524.
- Cretinism, 37.
- Croup, false, 707.
  - membranous, 707.
  - pseudo-membranous, 707.
- Crus, tumors of, 526.
- Cryptogenic septicæmia, 190.
- Cupric sulphate, poisoning by, 361.
- Cycloplegia, 595.
- Cyclothymia, 399.
- Cysticercus cellulosæ, 323, 327.
  - disease, 354.
- Cystinuria, 1009.
- Cystitis, 1058.
  - catheterization in, 1061.
  - diagnosis, 1060.
  - from spinal paralysis, 1062.
  - irrigation in, 1062.
  - local causes of, 1059.
  - morbid anatomy, 1059.
  - prognosis, 1061.
  - symptoms, 1059.
  - treatment, 1061.
  - urine in, 1059.
- Cysts of pancreas, 957.
  - renal, 1042.

D.

- Dactylitis syphilitica, 310.
- Debauch, 369.
- Decubitus acutus, 553.
- Delirifacients, poisoning by, 355.
- Delirium grave, 513.
  - in typhoid fever, 125.
  - tremens, 369.
    - complications, 370.
    - congestion of the lungs in, 372.
- Delirium tremens, diagnosis, 370.
    - hallucinations in, 369.
    - prognosis, 371.
    - treatment, 371.
- Delusions, 384.
  - expansive, 385.
  - hypochondriacal, 385.
  - insane, 385.
  - of persecution, 385.
  - systematized, 385.
- Dementia, 383.
  - paralytica, 516.
  - primary, curable, 395.
  - terminal, 397.
- Demodex folliculorum, 346.
- Dengue, 161.
- Diabetes insipidus, 92.
  - mellitus, 85.
    - coma in, 89.
    - diagnosis, 89.
    - diet in, 90.
    - pancreas in, 86.
    - prognosis, 90.
    - symptoms, 87.
    - treatment, 90.
    - urine in, 88.
- Diaceticaciduria, 1012.
- Diarrhœa of infants, acute, 860.
    - feeding in, 865.
    - treatment, 863.
  - chronic, 868.
    - treatment, 870.
- Diazo-reaction in typhoid fever, 134.
- Dilatation of stomach, 823.
    - diagnosis, 825.
    - lavage in, 826.
    - physical examination, 825.
    - prognosis, 825.
    - symptoms, 824.
    - treatment, 826.
- Diphtheria, 163.
  - antitoxin treatment, 177.
  - bacillus of, 164, 170
  - complications, 169.
  - croupous symptoms in, 169.
  - diagnosis, 170.
  - false membrane in, 166, 170, 174.
  - fibrinous inflammation in, 166.
  - local treatment, 173.
  - Loeffler's solution in, 175.
  - morbid anatomy, 165.
  - mucous membranes, 165.
  - paralysis following, 169.
  - prognosis, 171.
  - pseudo-membranous sore throat in, 165.
  - putrid sore throat in, 166.
  - symptomatology, 168.
  - treatment, 172.
    - antitoxin, 177.
- Diphthérite, 163.
- Diphtheroid, 171.
- Diplegia, facial, 612.
- Diplococcus pneumoniæ, 742.
- Diplopia, 481.
  - crossed, 481.
- Diplopia, homonymous, 481.
  - monocular, 481.
  - simple, 481.
- Disease, Addison's, 41.
  - African sleeping, 463.
  - Barlow's, 55.
  - Basedow's, 33.
  - Bell's, 513.
  - Bright's, 1022.
  - caisson, 449.
  - cysticercus, 327.
  - echinococcus, 329.
  - English sweating, 118.
  - foot-and-mouth, 237.
  - Graves's, 33.
  - Gilles de la Tourette's, 439.
  - Hodgkin's, 16.
  - hydatid, 329.
  - Madura foot, 232.
  - Ménière's, 418.
  - Morvan's, 574.
  - Raynaud's, 619.
  - Schoenlein's, 25.
  - Voltolini's, 418.
  - Weil's, 151.
  - Werlhof's, 26.
- Diseases, constitutional, 44.
  - due to animal parasites, 348.
  - infectious, 95.
  - locomotor, 44.
  - malarial, 204.
  - of adrenal glands, 44.
  - of bile-ducts, 931.
  - of brain, 509.
  - of bronchi, 712.
  - of gall-bladder, 931.
  - of heart, 637.
  - of intestines, 855.
  - of larynx, 707.
  - of liver, 909.
  - of lungs, 730.
  - of mediastinum, 787.
  - of medulla oblongata, 536.
  - of membranes of brain, 488.
  - of nerve-trunks, 594.
  - of nose, 700.
  - of pericardium, 628.
  - of pleura, 770.
  - of spleen, 28.
  - of thymus gland, 40.
  - of thyroid gland, 31.
  - of trachea, 712.
  - trophic, 619.
  - vaso-motor, 619.
- Disseminated sclerosis, 521.
  - tuberculosis, 244.
- Distoma conjunctum, 335.
  - crassum, 335.
  - hæmatobium, 335.
  - hepaticum, 336.
  - heterophyes, 335.
  - lanceolatum, 335.
  - ophthalmobium, 335.
  - pulmonale, 336.
  - sinense, 335.
- Distomiasis, 336.
- Dochmiasis, 339.
- Double athetosis, 483.
- Doubt, delirium of, 387.
- Dracontiasis, 341.
- Dropsy, abdominal, 967.

Dropsy, cardiac, 662.
  of gall-bladder, 936.
  renal, 1018.
    treatment, 1032.
Duchenne's form of atrophic myopathy, 48.
Ductless glands, tuberculosis of, 281.
Dysæsthesia, 552.
Dysentery, 212.
    abscess of liver in, 214.
    amœba in, 212.
    catarrhal inflammation in, 213.
    diagnosis, 216.
    diphtheritic inflammation in, 214.
    follicular inflammation in, 213.
    morbid anatomy, 213.
    prognosis, 216.
    stools in, 215.
    symptoms, 215.
    treatment, 217.
  amœbic, 214.
  chronic, 214, 216.
  tropical, 212.
Dyspepsia, acute, 827.
  chronic, 831.
  nervous, 852.
  treatment, 853.
Dysthesia, psychical, 391.

E.

Echinococci of heart, 332.
  of kidney, 333.
  of liver, 332.
  of lungs, 333.
  of peritoneum, 334.
  of pleura, 332.
Echinococcus disease, 329.
    cysts in, 330.
    diagnosis, 335.
    localization of, 332.
    prognosis, 335.
    symptoms, 331.
    treatment, 335.
Echolalia, 440.
Ehrlich's test for typhoid fever, 134.
Elbow-jerk, 379.
Electrical sensibility, 381.
Elephantiasis, 341.
  Græcorum, 301.
Embolism, cerebral, 494.
    diagnosis, 496.
    prognosis, 497.
    treatment, 497.
  in endocarditis, 653.
  of spleen, 30.
Emotion, 388.
Emphysema, 738.
    alveolar, 738.
    collateral, 739.
    complementary, 739.
    essential, 738.
    Gerhardt's treatment of, 740.
    interstitial, 738.
    senile, 739.
    symptoms, 739.
Emphysema, treatment, 740.
  vesicular, 738.
  vicarious, 739.
Empyema, 777.
  necessitatis, 778.
  pulsating, 781.
Encephalitis, acute hemorrhagic, 509.
  hypertrophic, 510.
  peripheral, 513.
  sclerotic, 510.
  suppurative, 510.
Encephalopathia saturnina, 363.
Endarteriitis chronica nodosa sive deformans, 686.
Endarteritis, syphilitic, 311.
Endocarditis, acute, 651.
    bacteria in, 652.
    cause of, 655.
    diagnosis, 656.
    embolism in, 653.
    malignant, 654.
    morbid anatomy, 652.
    murmur in, 654.
    prognosis, 656.
    pyæmic, 655.
    septic, 654.
    symptoms, 653.
    treatment, 656.
    types of malignant, 654.
    valvular aneurism in, 653.
  chronic, 657.
    brown induration of lungs in, 661.
    calcification in, 658.
    Cheyne-Stokes breathing in, 660.
    dropsy in, 662.
    dyspnœa in, 660.
    embolism in, 662.
    fibrous, 658.
    insufficiency in, 659.
    kidneys in, 662.
    nutmeg liver in, 661.
    parietal form of, 657.
    prognosis, 663.
    stenosis in, 659.
    symptoms, 659.
    treatment, 668, 676.
    ulcerative, 658.
    urine in, 662.
    valvular forms of, 657.
    venous pulsation in, 661.
English sweating disease, 118.
Enteritis, 857.
  acute catarrhal, 859.
    diagnosis, 861.
    feeding in, 862.
    of infants, 860.
      feeding in, 865.
      treatment, 863.
    stools in, 859.
    symptoms, 859.
    treatment, 861, 863.
  chronic, 866.
    catarrhal, 866.
      symptoms, 866.
      treatment, 867.
  diphtheritic, 875.
  follicular, 859, 873.
  gangrenous, 875.
  phlegmonous, 875.
Enteritis, pseudo-membranous, 870.
    enemata in, 872.
    treatment, 871.
  tubercular, 267.
  ulcerative, 872.
    symptoms, 873.
    treatment, 874.
  varieties of, 858.
Enteroliths, 894.
Enteroptosis, 855.
Enuresis, 1057.
  nocturnal, 1057.
Epidemic capillary bronchitis, 104.
  parotitis, 183.
Epilepsia procursiva, 421.
Epilepsy, 418.
  anomalous forms of, 421.
  aura in, 420.
  cardiac, 423.
  convulsions in, 419.
  diagnosis, 423.
  idiopathic, 418.
  Jacksonian, 525.
  morbid anatomy, 419.
  nocturnal, 421.
  procursive, 421.
  prognosis, 424.
  reflex, 423.
  senile, 680.
  spinal, 586.
  surgical treatment, 428.
  symptomatology, 419.
  theory of cause, 419.
  toxæmic, 423.
  treatment, 425.
Epileptic automatism, 422.
  mania, 422.
  status, 422.
Epileptiform convulsions, 378.
Epistaxis, 700.
Erb's form of atrophic myopathy, 48.
Erotomania, 386.
Eructation, nervous, 850.
Erysipelas, 185.
  diagnosis, 188.
  migrans, 187.
  of mucous membranes, 187.
  phlegmonous, 187.
  pneumonia, 188.
  prognosis, 188.
  symptomatology, 186.
  treatment, 188.
Essential anæmia, 9.
Eustrongylus gigas, 339.
Exophthalmic goitre, 33.
    diagnosis, 35.
    electricity in, 36.
    prognosis, 35.
    treatment, 36.
Expansive delusion, 385.

F.

Facial diplegia, 612.
  nerve, neuritis of, 611.
    paralysis of, 596.
  spasm, 431.
Facio-scapulo-humeral type of atrophic myopathy, 48.

Fæces, impacted, 907.
Fallopian tubes, tuberculosis of, 274.
Farcy, 237.
  buds, 237.
  sores, 237.
Fehling's test for sugar in urine, 1011.
Fermentation test for sugar in urine, 1011.
Fever, æstivo-autumnal, 207.
  ardent, continued, 451.
  bilious, 207.
    remittent, 207.
  break-bone, 161.
  camp, 145.
  charts, 1067–1071.
  congestive malarial, 208.
  hay, 704.
  hectic, in phthisis, 261, 299.
  infantile remittent, 131.
  intermittent, 201.
  malarial, 201.
    congestive, 208.
  malignant, 208.
  miliary, 118.
  pernicious, 208.
  pneumo-typhoid, 130.
  prison, 145.
  relapsing, 149.
  remittent, 207.
    bilious, 207.
  rheumatic, 61.
  scarlet, 95.
  ship, 145.
  spotted, 152.
  thermic, 450.
  typhoid, 119.
  typho-malarial, 132.
  typhus, 145.
  walking typhoid, 130.
  yellow, 226.
Fibrinuria, 1006.
Fibroid phthisis, 264.
Fibrous pneumonia, 264.
Filaria bronchialis, 342.
  labialis, 342.
  lentis, 342.
  Loa, 342.
  medinensis, 340.
  sanguinis hominis, 9, 341.
Fish tape-worm, 324.
Fleas, 348.
Flies, house-, 348.
Flukes, 335.
Fœtal rickets, 50 (*note*).
  syphilis, 314.
Foot-and-mouth disease, 237.
Formes frustes, 33.
Formulary, 1063.
Friedreich's ataxia, 589.

G.

Galacturia, 1000.
Gall-bladder, cancer of, 948.
  diseases of, 931.
  dropsy of, 936.
  empyema of, 936.
  inflammation of, 935.
Gall-ducts, cancer of, 948.
  inflammation of, 935.
Gall-ducts, inflammation of,
    diagnosis, 937.
    morbid anatomy, 936.
    prognosis, 938.
    symptoms, 937.
    treatment, 938.
  suppurative inflammation of, 936.
    treatment, 939.
Gall-stones, 939. (*Vide* Cholelithiasis.)
  composition of, 940.
  impaction of, 943.
  incarceration of, 942.
Gangrene, diabetic, 690.
  local, 620.
  pulmonary, 764.
    diagnosis, 765.
    prognosis, 766.
    symptoms, 765.
    treatment, 766.
  senile, 690.
  symmetrical, 619.
Gastralgia, acute, 598, 850.
    treatment, 842.
Gastralgokenosis, 850.
Gastrectasia, 823.
Gastric catarrh, acute, 827.
    chronic, 831.
  neuroses, 849.
Gastritis, 827.
  acute acid, 833.
    catarrhal, 827.
      diagnosis, 829.
      prognosis, 829.
      pyrosis in, 828.
      symptoms, 828.
      water-brash in, 828.
  atrophic, 832.
  chronic catarrhal, 831.
      diagnosis, 833.
      lavage in, 835.
      prognosis, 833.
      symptoms, 832.
      treatment, 835.
  hypertrophic, 831.
  phlegmonous, 829.
  pseudo-membranous, 829.
  toxic, 830.
Gastro-adenitis, parenchymatous, 828.
Gastrodiaphany, 829.
Gastrodynia, 850.
Gastroptosis in chlorosis, 5.
Gastroxyrosis, 852.
Gelsemium, poisoning by, 358.
Geographic tongue, 794.
Gerhardt's treatment of emphysema, 740.
German measles, 107.
Giant urticaria, 622.
Gilles de la Tourette's disease, 439.
Glanders, 237.
  chronic, 238.
  diagnosis, 238.
  prognosis, 239.
  treatment, 239.
Globulinuria, 1001, 1004.
Globus hystericus, 407.
Glossitis, 793.
  parenchymatous, 794.
Glosso-labial paralysis, 537.
Glycosuria, 85, 1009. (*Vide* Diabetes mellitus.)
Gmelin's test for bilirubin, 933.
Goitre, 31.
  exophthalmic, 33.
Gonorrhœal rheumatism, 68.
Gout, 76.
  alcohol in, 77.
  articular, 78.
  baths in, 83.
  chronic, 78.
  diagnosis, 79.
  diet in, 83.
  heredity in, 76.
  kidneys in, 77.
  local treatment of, 82.
  metastatic, 79.
  morbid anatomy, 77.
  prognosis, 80.
  retrocedent, 79.
  retrograde, 79.
  rheumatic, 71.
  symptoms, 78.
  tophi in, 77.
  treatment, 81.
  uric acid in, 76, 78, 80.
  visceral, 78.
Graves's disease, 33.
Grease, 39.
Green sickness, 4.
Grippe, 158.
Guinea-worm, 340.

H.

Hæmatemesis, 839.
Hæmatidrosis, 410.
Hæmatocele, 962.
Hæmatochyluria, 341.
Hæmatoma of dura mater, 488.
Hæmatomyelia, 543.
Hæmatomyelitis, 553.
Hæmatorrhachis, 543.
Hæmatozoa, 321.
  of malaria, 203, 321.
Hæmaturia, 994.
  source of, 995.
Hæmoglobinæmia, 27.
Hæmoglobinuria, 996.
  paroxysmal, 997.
Hæmopericardium, 628.
Hæmoperitoneum, 962.
Hæmophilia, 20.
Hæmoptysis, 732.
  in phthisis, 259.
Hæmosalpinx, 963.
Hæmothorax, 774.
Hallucinations, 384.
Harvest-mite, 346.
Hay fever, 704.
Headache, 456.
  caffeinic, 457.
  gastric, 457.
  gouty, 457.
  lithæmic, 457.
  nervous, 457.
  sympathetic, 457.
  toxæmic, 456.
Hearing, centre for, 476.
Heart, alterations of, in nephritis, 1018.

Heart, aneurism of, 648.
  aplasia of, 638.
  atrophy of, 638.
  dilatation of, 641, 670, 674.
  diseases of, 637.
  echinococci of, 332.
  endocarditis of, 657.
    prognosis, 663.
    symptoms, 659.
    treatment, 668.
  fatty degeneration of, 643.
  fatty infiltration of, 642.
  gummata in, 311.
  hypertrophy of, 639, 669.
  hypoplasia of, 638.
  irritable, 678.
  malformation of, 637.
  murmurs, 664 *et seq.*
  neuralgia of, 682.
  palpitation of, 676, 678.
  rupture of, 649.
  syphilitic disease of, 311.
  thrombosis of, 648.
  tuberculosis of, 283.
  tumors of, 650.
  valvular lesions of, 675.
    treatment, 668, 673, 676.
Heat exhaustion, 450.
  test in albuminuria, 1002.
Hebephrenia, 398.
Heberden's nodules in gout, 77.
Helminthiasis, 322.
Hemeralopia, 933.
Hemianopsia, heteronymous, 479.
  homonymous, 479.
    lateral, 479.
  horizontal, 479.
Hemiatrophy, facial, 625.
Hemichorea, 380, 483.
Hemiopia periodica, 459.
Hemiplegia, 377.
Hemitremors, 483.
Hemorrhagic diathesis, 19.
  encephalitis, 509.
  rickets, 55.
  small-pox, 110, 113.
Henoch's purpura, 26.
Hepatitis, acute parenchymatous, 916.
  chronic interstitial, 923.
  fibrous, 923.
    ascites in, 925.
    diagnosis, 926.
    hemorrhage in, 924.
    morbid anatomy, 923.
    prognosis, 926.
    symptoms, 924.
    treatment, 926.
    varieties, 923.
  suppurative, 918.
    diagnosis, 921.
    enlargement in, 921.
    morbid anatomy, 919.
    prognosis, 922.
    symptoms, 920.
    treatment, 922.
Hereditary chorea, 440.
Heteronymous hemianopsia, 479.
Hiccough, 416.
Hiccup, 416.
Hirudo ceylonica, 336.
  vorax, 336.
Hodgkin's disease, 16.
Homonymous hemianopsia, 479.
  lateral hemianopsia, 479.
Hydatid disease, 329.
Hydatids of heart, 359.
  of kidney, 359.
  of liver, 359.
  of lungs, 359.
  of peritoneum, 359.
Hydrocephalus, 512.
  acute, 246.
  externus, 512 (*note*).
  internus, 512 (*note*).
  spurious, 493.
Hydrochinonuria, 999.
Hydrochloric acid in gastric juice, tests for, 820.
    poisoning by, 360.
    tests for, in cancer of stomach, 847.
Hydromyelia, 572.
Hydronephrosis, 1048.
  diagnosis, 1050.
  intermittent, 1050.
  morbid anatomy, 1049.
  operation in, 1051.
  prognosis, 1051.
  symptoms, 1049.
  treatment, 1051.
Hydropericardium, 629.
Hydroperitoneum, 967.
  cachectic, 967.
  diagnosis, 970.
  fluid of, 967, 971.
  mechanical, 967.
  morbid anatomy, 967.
  physical examination, 969.
  prognosis, 971.
  symptoms, 969.
  treatment, 972.
  vaginal examination, 970.
Hydrophobia, 233.
  spurious, 407.
Hydropneumothorax, 771.
Hydrothionuria, 1013.
Hyperæsthesia, 382.
  psychical, 391.
Hyperbulia, 382.
Hypergeusia, 477.
Hyperorexia, 849.
Hypertrophic cirrhosis of liver, 927.
Hypochondriacal delusions, 385.
Hypomania, 394.
Hysteria, 405.
  anuria in, 410.
  beast mimicry, 407.
  bloody sweat, 410.
  blue œdema in, 410.
  convulsions in, 407.
  diagnosis, 411.
  globus hystericus, 407.
  headache in, 458.
  mental symptoms, 406.
  opisthotonos in, 407.
  phantom tumor, 412.
  prognosis, 412.
  sight in, 409.
  treatment, 412.
Hysterical anæsthesia, 409.
  ataxia, 415.
  breast, 412.
  chorea, 438.
  convulsions, 327, 378.
  paralysis, 408.
  somnolence, 408.
  trance, 408.
  vertigo, 417.

I.

Icterus, 931.
  febrile, 151.
Iliac abscess, 876.
  phlegmon, 876.
Illuminating gas, poisoning by, 351.
Illusion, 384.
Imperative act, 386.
  conception, 385.
Incoherence, 383.
Indicanuria, 998.
Infantile form of atrophic myopathy, 48.
  osteomalacia, 57.
  remittent fever, 131.
  scurvy, 55.
  spastic paralysis, 508.
Infectious diseases, 95.
Influenza, 158.
  complications, 160.
  diagnosis, 160.
  duration, 159.
  prognosis, 160.
  treatment, 160.
Infusoria, 322.
Insane delusions, 385.
  general paralysis of, 510.
Insanity, 389.
  alcoholic, 373.
  circular, 399.
  confusional, 395.
  hallucinatory, 395.
  menstrual, 399.
  periodical, 399.
  puerperal, 515.
  post-febrile, 395.
  stuporous, 395.
  surgical, 395.
Insanities, complicating, 389 (*note*).
  constitutional, 390.
  functional, 391.
  neuropathic, 391, 397.
  organic 389 (*note*).
  pure, 391.
  toxæmic, 390.
Insects, parasitic, 346.
Insomnia, 461.
Intellection, disturbances of, 382.
Intermittent fever, 201. (*Vide* Malarial Diseases.)
Intestinal tuberculosis, 265.
    diagnosis, 268.
    secondary, 267.
    symptoms, 267.
    treatment, 299.
    ulcers in, 266.
Intestine, abnormal contents of, 894.

Intestine, cancer of, 900.
    diagnosis, 902.
    symptoms, 901.
    prognosis, 903.
    treatment, 903.
  enteroliths in, 894.
  hemorrhage into, 855.
    in typhoid fever, 128.
  intussusception, 892.
    diagnosis, 896.
    laparotomy in, 900.
    prognosis, 898.
    treatment, 899.
  knots in, 893.
  obstruction of, 891.
    diagnosis, 896.
    prognosis, 898.
    symptoms, 895.
    treatment, 899.
  obstruction of, chronic, 896.
  perforation of, in typhoid fever, 122, 128, 144.
  strangulation of, 892.
  stricture of, 894.
  tuberculosis of, 265.
  tumors of, 894.
  twisting of, 893.
  ulcers of, amyloid, 873.
    catarrhal, 873.
    follicular, 873.
    stercoral, 873.
Intra-cranial tumors, 524.
Intra-peritoneal hemorrhage, 962.
      diagnosis, 965.
      ectopic pregnancy, 963.
      morbid anatomy, 963.
      physical examination, 964.
      prognosis, 966.
      symptoms, 963.
      treatment, 966.
Iridoplegia, 595.
  reflex, 580.
Itch insect, 346.
Ixodes americanus, 346.
  ricinus, 346.

J.

Jacksonian epilepsy, 525.
Jaundice, 931.
    bilirubin in, 933.
    diagnosis, 934.
    morbid anatomy, 932.
    obstruction to flow of bile in, 932.
    prognosis, 935.
    skin in, 934.
    stools in, 934.
    symptoms, 932.
    treatment, 935.
    urine in, 933.
    vision in, 933.
  acute infectious, 151.
  catarrhal, 937.
  hæmatogenous, 931.
Jiggers, 348.
Joints, intermittent dropsy of, 623.
  tuberculosis of, 285.
Jumpers, 439.
Juvenile form of atrophic myopathy, 48.

K.

Keloid, 625.
Kidney, abscess of, 1040.
  amyloid degeneration of, 1038.
  anomalies of, 989.
  atrophy, granular, of, 1033.
  Bright's disease of, 1022.
  cirrhosis of, 1033.
  congestion of, 1019.
    passive, 1020.
  contracted, 1033.
  cyanotic induration of, 662.
  cystic dropsy of, 1042.
  cysts of, 1042.
  diseases of, 989.
  echinococci in, 332.
  embolism of, 1021.
  fatty, 1028.
  floating, 989.
    diagnosis, 992.
    prognosis, 992.
    symptoms, 990.
    treatment, 993.
    urine in, 991.
  fused, 989.
  gouty, 1033.
  granular atrophy of, 1033.
  induration of, 662.
  inflammation of, acute, 1022.
  large white, 1028.
  movable, 989.
  multilocular cystic, 1042.
  scrofulous, 270.
  small granular, 1033.
  surgical, 1040.
  thrombosis of, 1021.
  tuberculosis of, 270.
  tumors of, 1043.
  wandering, 989.
Klebs-Loeffler bacillus, 164, 170.
Kleptomania, 386.
Knee-jerk, 378.
  in cerebral tumors, 527.
Kyphosis, 601.
  paralytic, 601.

L.

Lactosuria, 1010.
Lamblia intestinalis, 322.
Landouzy's form of atrophic myopathy, 48.
Landry's paralysis, 548.
Laryngeal phthisis, 263.
Laryngismus stridulus, 429.
Laryngitis, acute, 707.
    diagnosis, 708.
    prognosis, 708.
    treatment, 708.
  chronic, 709.
  syphilitic, 710.
Larynx, diseases of, 706.
  œdema of, 710.
  phthisis of, 263.
  tumors of, 711.
Latah, 439.
Lateral curvature of spine, 601.
Lavage in chronic gastritis, 835.
  in dilatation of stomach, 826.
Lead colic, 333.
  paralysis, 334.
  poisoning by, 362.
    albuminuria in, 365.
    amblyopia in, 364.
    diagnosis, 366.
    treatment, 366.
    wrist-drop in, 363.
Leeches, 336.
Lepra, 301.
Leprosy, 301.
  anæsthetic, 302.
  bacillus of, 301.
  diagnosis, 303.
  nodular, 303.
  prognosis, 303.
  treatment, 304.
  tubercular, 303.
Leptomeningitis, 489, 546.
Leptus irritans, 346.
Leukæmia, 12.
  blood in, 14.
  diagnosis, 15.
  prognosis, 16.
  symptoms, 13.
  treatment, 16.
Leukocytosis, 11.
Leukoplakia, buccal, 794.
Lieno-lymphatic leukæmia, 13.
Lipaciduria, 1012.
Lipæmia, 1001.
Lipuria, 1000.
Lithæmia, 80.
Lithuria, 1006.
Liver, abscess of, 918.
  acute yellow atrophy of, 916.
      diagnosis, 918.
      morbid anatomy, 917.
      prognosis, 918.
      symptoms, 917.
      treatment, 918.
  amyloid, 928.
  atrophy of, nutmeg, 912.
    red, 912.
  cancer of, 928.
    diagnosis, 931.
    morbid anatomy, 929.
    prognosis, 931.
    symptoms, 930.
    treatment, 931.
    urine in, 930.
  cirrhosis of, 923.
    atrophic, 924.
    hypertrophic, 927.
  congestion of, 911.
  diseases of, 909.
  echinococci of, 332.
  fatty infiltration of, 910.
  -fluke, 336.
  hypertrophic cirrhosis of, 927.
  malformation of, 909.
  malposition of, 909.
  nutmeg, 661.
  syphilitic disease of, 312.
  tuberculosis of, 268.
  wandering, 907.

Lobelia, poisoning by, 357.
Localization of cerebral disease, 471, 481.
  of spinal disease, 540.
Locomotor ataxia, 575.
    Argyll-Robertson pupil in, 580.
    ataxic gait in, 579.
    crises in, 577.
    diagnosis, 581.
    electricity in, 584.
    morbid anatomy, 576.
    prognosis, 582.
    reflexes in, 579.
    stages of, 581.
    suspension in, 585.
    symptomatology, 577.
    treatment, 582.
    trophic changes in, 580.
  diseases, 44.
Loeffler's solution, 175.
Lordosis, 601.
Louse, 373.
Lumbago, 74.
Lump-jaw in cattle, 231.
Lung-fluke, 336.
Lungs, acute tuberculosis of, 247.
      diagnosis, 248.
      examination in, 248.
      onset of, 247.
      prognosis, 249.
      treatment, 292.
  anthracosis of, 756.
  brown induration of, 661, 731.
  cancer of, 767.
  chalicosis of, 756.
  cirrhosis of, 264, 756.
  congestion of, 730.
  diseases of, 730.
  echinococci of, 332.
  embolism of, 733.
  gangrene of, 764.
  gummata in, 311.
  hypostatic congestion of, 731.
  œdema of, 734.
    collateral, 735.
    hypostatic, 735.
  siderosis of, 756.
  splenization of, 731.
  thrombosis of, 733.
  tumors of, 767.
Lupus, 250.
  treatment, 300.
Lymph-glands, tuberculosis of, 281.
Lymph-scrotum, 341.
Lymphatic anæmia, 16.
  leukæmia, 13.
Lymphoma, malignant, 16.
Lympho-sarcoma, malignant, 16.

M.

Macrocephalus, 512.
Macroglossia, 795.
Macropsia, 594.
Maculosus Werlhofii, 26.
Madura foot disease, 232.
Makrophages in malaria, 205.
Mal de montagne, 417.
Malarial diseases, 201.
    blood in, 210.
    cachexia, 209.
    diagnosis, 209.
    germ of, 203.
    makrophages in, 205.
    morbid anatomy, 205.
    paroxysms, 205.
    prognosis, 210.
    remittent type, 207.
    symptomatology, 205.
    treatment, 210.
  fever, algid, 208.
    comatose, 208.
    congestive, 208.
    hemorrhagic, 208.
    malignant, 208.
    pernicious, 208.
Malignant lymphoma, 16.
  lympho-sarcoma, 16.
  malarial fever, 208.
  pustule, 235.
  small-pox, 110, 113.
Malum senile, 73.
Mammary gland, tuberculosis of, 274.
Mania, 386, 393.
  chronic, 394.
  epileptic, 422.
  following fevers, 395.
  hallucinatoria, 395.
  periodica, 399.
  puerperal, 395.
Mania-a-potu, 369.
Massage, 404.
Measles, 102. (*Vide* Rubeola.)
  black, 104.
  German, 107.
  in swine, 323.
Mediastinitis, 787.
  indurative, 788.
Mediastino-pericarditis, 788.
Mediastinum, diseases of, 787.
    diagnosis, 790.
    treatment, 790.
  tumors of, 789.
Medulla, diseases of, 536.
  tumors of, 526.
Megalopsia, 594.
Megastoma entericum, 322.
Melancholia, 391.
  agitata, 392.
  attonita, 392.
  hypochondriacal, 392.
  periodica, 399.
  religiosa, 392.
Melanuria, 999.
Melasma, suprarenal, 41.
Melituria, 1009.
Memory, periodic failure of, 467.
Meningitis, 489.
  acute, 489.
  cerebro-spinal, 152.
  chronic, 492.
  occlusive, 490.
  posterior, 490.
  spinal, acute, 546.
  tubercular, 246, 491.
    hydrocephalic cry in, 247.
    symptoms, 246.
Menstrual insanity, 399.
Merycism, 850.
Mesoneuritis, 609.
Micropsia, 594.
Migraine, 458.
  ophthalmic, 459.
Miliary fever, 118.
  tubercle, 242.
Mind-blindness, 487.
Miryachit, 439.
Mitral insufficiency, 664.
  stenosis, 665.
Monophasia, 485.
Monoplegia, 377.
Monostoma lentis, 335.
Moore's test for sugar in urine, 1010.
Morbid impulse, 385.
Morbus coxæ senilis, 73.
Morphœa, 625.
Morvan's disease, 574.
Motor centres of brain, 473.
Movable spleen, 29.
Mucinuria, 1001.
Mucous membranes, tuberculosis of, 250.
Multiple sclerosis, 522.
    cerebro-spinal, 522.
Mumps, 183.
Muscular atrophy, neuritic, 615.
      Charcot-Marie type of, 616.
      peroneal type of, 616.
      progressive, 569.
  rheumatism, 74.
  sense, 381.
Myalgia, 74.
Mycetoma, 232.
Myelæmia in leukæmia, 15.
Myelitis, acute, 551.
    diagnosis, 554.
    prognosis, 554.
    softening in, 552.
    stages of, 552.
    symptomatology, 552.
    treatment, 554.
  central, 551.
  centralis, 553.
  cervicalis, 554.
  chronic, 556.
  compression, 558.
  disseminated, 551.
  explosive, 553.
  foudroyant, 553.
  gray, 551.
  hemorrhagic, 551.
  insular, 551.
Myelogenous leukæmia, 13.
Myeloma, 19.
Myiasis, 348.
Myocarditis, 645.
  prognosis, 647.
  treatment, 647.
Myoma, peritoneal pseudo-, 974.
Myomalacia, 646.
Myopathic face, 48.
Myopathy, atrophic, 48.
  primary, 45.
  pseudo-hypertrophic, 46.
Myositis, 44.
  ossificans progressiva, 44.
  primary, 44.

Myositis, rheumatic, 44.
suppurative, 44.
Myotoma, congenital, 49.
Mysophobia, 386.
Myxœdema, 37.
treatment, 39.

N.

Narcolepsy, 408, 464.
Narcotics, poisoning by, 351.
Nasal catarrh, acute, 701.
chronic, 703.
Nelavan, 463.
Nephrectomy, 993.
Nephritis, acute, 1022.
diagnosis, 1025.
dropsy in, 1023.
morbid anatomy, 1023.
prognosis, 1025.
symptoms, 1023.
treatment, 1026.
urine in, 1024.
acute catarrhal, 1022.
alteration of heart in, 1018.
acute croupous, 1022.
acute desquamative, 1022.
acute diffuse, 1022.
acute parenchymatous, 1022.
chronic diffuse, 1028.
diagnosis, 1030.
morbid anatomy, 1028.
prognosis, 1031.
symptoms, 1029.
treatment, 1031.
urine in, 1029.
chronic fibrous, 1033.
albuminuric retinitis in, 1036.
diagnosis, 1036.
dyspnœa, 1036.
hypertrophy of heart in, 1034, 1035.
morbid anatomy, 1033.
prognosis, 1037.
symptoms, 1034.
treatment, 1037.
urine in, 1035.
chronic interstitial, 1033.
parenchymatous, 1028.
glomerular, 1022.
glomerulo-capsular, 1022.
suppurative, 1040.
Nephrolithiasis, 1053.
diagnosis, 1055.
passage of stone in, 1054.
prognosis, 1055.
symptoms, 1054.
treatment, 1056.
Nephroptosis, 989.
Nephrorrhaphy, 993.
Nerve, syphilis of, 618.
Nervous diseases, functional, 889.
dyspepsia, 852.
Neuralgia, 468.
diagnosis, 470.
of bladder, 1058.
of heart, 682.
treatment, 470.
Neurasthenia, 400.
Neurasthenia, diet in, 403.
massage, 404.
rest-cure, 402.
treatment, 401.
Neuritic muscular atrophy, 615.
Neuritis, 603.
alcoholic, 607.
diabetic, 607.
multiple, 605.
parenchymatous, 605.
post-febrile, 608.
segmentary, 606.
senile, 608.
Neuroma, 617.
Neuroses, cardiac, 678.
occupation, 454.
traumatic, 446.
Night palsy, 464, 623.
Night-sweats in phthisis, 299.
Night-terrors, 465.
Nitric acid, poisoning by, 360.
test in albuminuria, 1002.
Nitrites, poisoning by, 357.
Nitro-benzol, poisoning by, 352.
Nitrohydrochloric acid, poisoning by, 360.
Noma, 792.
Nose, diseases of, 700.
Nosebleed, 700.
Note-blindness, 485.
Nucleoalbuminuria, 1001, 1005.
Nyctalopia, 933.
Nymphomania, 386.

O.

Obesity, 58.
Obstipation, 903.
Occipital lobe, tumors of, 526.
Occupation neuroses, 454.
Œdema, angioneurotic, 622.
neonatorum, 625.
Œsophagismus, 815.
Œsophagitis, 809.
catarrhal, 810.
corrosive, 810.
diphtheritic, 810.
dissecans superficialis, 810.
fibrinous, 810.
follicular, 810.
phlegmonous, 810.
Œsophagus, cancer of, 812.
diagnosis, 814.
symptoms, 813
treatment, 814.
dilatation of, 806.
diseases of, 804.
diverticulum, 807.
pulsion, 817.
traction, 808.
ectasis of, 806.
inflammation of, 809.
obstruction of, 804.
paralysis of, 815.
perforation of, 808.
rupture of, 809.
spasm of, 815.
stenosis of, 804.
stricture of, 804.
tumors of, 812.
Oïdium albicans, 792.
Oliguria, 1015.
Omodynia, 74.
Ophthalmic migraine, 459.
Ophthalmoplegia, 595.
externa, 595.
interna, 595.
progressiva, 595.
Opium, poisoning by, 351.
treatment, 353.
Opiumism, 374.
treatment, 375.
Optic disk in cerebral disease, 477.
thalamus, tumors of, 526
Osteitis deformans, 626.
Osteo-arthropathy, hypertrophic, 723.
Osteomalacia, 56.
infantile, 57.
puerperal, 57.
senile, 57.
Osteomyelitis tuberculosa, 285.
Osteoporosis, 524.
Ovaries, tuberculosis of, 274.
Oxalic acid, poisoning by, 360.
Oxaluria, 1007.
Oxyuris vermicularis, 338.

P.

Pachydermia laryngis, 710.
Pachymeningitis, 488.
cervical, 547.
externa, 488.
interna, 488.
hæmorrhagica, 488.
spinal, acute, 546.
Painless tic, 431.
Palpitation of heart, 676, 678.
Palsy, Bell's, 611.
bulbar, 537.
cerebral, of children, 508.
night, 623.
pressure, 603.
wasting, 570.
Pancreas, cancer of, 960.
cysts of, 957.
contents, 958.
diagnosis, 959.
prognosis, 960.
symptoms, 958.
treatment, 960.
diseases of, 950.
hemorrhage of, 950.
tuberculosis of, 269.
Pancreatic calculi, 955.
Pancreatitis, acute, 951.
diagnosis, 953.
symptoms, 952.
treatment, 954.
chronic, 954.
gangrenous, 951.
hemorrhagic, 951.
suppurative, 952.
Paræsthesia, 382.
Parageusia, 477.
Paragraphia, 485.
Paralexia, 485.
Paralysis, 377.
acute ascending, 548.
agitans, 444.
Bell's, 611.
brachial, 377.

Paralysis, bulbar, 537.
  cerebral, of children, 508.
  complete, 377.
  crossed, 377.
  crural, 377.
  facial, 377.
  following diphtheria, 169.
  following typhoid fever, 133.
  general, 377.
  general, of insane, 516.
  hysterical, 408.
  incomplete, 377.
  infantile spastic, 508.
  labial, 537.
  Landry's, 548.
  local, 377.
  multiple, 377.
  oculo-motor, 594.
  of abducens nerve, 595.
  of anterior crural nerve, 601.
  of anterior thoracic nerve, 599.
  of auditory nerve, 596.
  of circumflex nerve, 599.
  of eighth cranial nerve, 596.
  of external popliteal nerve, 602.
  of facial nerve, 596.
  of fifth cranial nerve, 595.
  of fourth cranial nerve, 595.
  of glosso-pharyngeal nerve, 598.
  of gluteal nerve, 602.
  of ilio-hypogastric nerve, 601.
  of ilio-inguinal nerve, 601.
  of insane, 516.
  of intercostal nerve, 601.
  of internal popliteal nerve, 603.
  of laryngeal muscles, 597.
  of long thoracic nerve, 598.
  of median nerve, 600.
  of musculo-cutaneous nerve, 599.
  of musculo-spiral nerve, 599.
  of obturator nerve, 602.
  of oculo-motor nerve, 594.
  of œsophagus, 815.
  of pneumogastric nerve, 597.
  of sciatic nerve, 602.
  of seventh cranial nerve, 596.
  of sixth cranial nerve, 595.
  of spinal accessory nerve, 598.
  of spinal nerves, 601.
  of subscapular nerve, 599.
  of suprascapular nerve, 599.
  of third cranial nerve, 594.
  of trigeminus nerve, 595.
  of trochlear nerve, 595.
  of ulnar nerve, 600.
  periodic, 429.
Paralytic stroke, 498.
Paramusia, 485.
Paramyoclonus multiplex, 444.
Paramyotonia congenita, 50.
Paranephritic abscess, 1051.
Paranephritis, suppurative, 1051.
Paranoia, 398.
Paraphasia, 485.
Paraplegia, ataxic, 588.
  spastic, 585.
Parasites, 321.
Parasitic insects, 346.
  protozoa, 321.
Paratyphlitis, 876.
Paresis, 377, 516.
Paretic dementia, 516.
Parietal lobes, tumors of, 526.
Parorexia, 850.
Parotitis, 796.
  epidemic, 186.
Pediculus capitis, 346.
  pubis, 346.
  vestimentorum, 346.
Peliosis rheumatica, 25.
Pentastoma denticulatum, 345.
  tænioides, 345.
  constrictum, 346.
Pentastomum, 345.
Peptonuria, 1001, 1005.
Perforating ulcer, 621.
Periarteritis, nodular, 692.
Pericarditis, 630.
    course, 633.
    diagnosis, 634.
    paracentesis in, 635.
    physical examination, 632.
    prognosis, 634.
    symptoms, 632.
    treatment, 634.
  chronic, 635.
  dry, 613.
  obliterative, 636.
  sicca, 631.
  tubercular, 275, 631.
Pericardium, diseases of, 628.
Periencephalitis, acute, 513.
    diagnosis, 514.
    prognosis, 515.
    treatment, 515.
  chronic, 516.
    convulsions in, 518.
    diagnosis, 520.
    handwriting in, 520.
    paralysis in, 519.
    prognosis in, 521.
    stages of, 518.
    symptomatology, 517.
    treatment, 521.
  idiopathic, 513.
  septic, 513.
Periencephalo-meningitis, 516.
Perihepatitis, 914.
  acute suppurative, 914.
    diagnosis, 916.
    prognosis, 916.
    treatment, 916.
  chronic, 916.
Perinephritic abscess, 1015.
Perineuritis, 603.
Periodical insanity, 399.
  paralysis, 429.
Peripheral encephalitis, acute, 513.
Peripleuritis, 783.
Perisplenitis, 29.
Peristaltic unrest, 851.
Peritoneum, cancer of, 986.
  diseases of, 962.
  echinococci of, 334.
  inflammation of, 972.
Peritoneum, tuberculosis of, 276.
    diagnosis, 279.
    laparotomy in, 300.
    morbid anatomy, 277.
    prognosis, 280.
    symptoms, 278.
    treatment, 300.
  tumors of, 986.
Peritonitis, 972.
  acute, 973.
    cathartics in the treatment of, 981, 983.
    diagnosis, 978.
    morbid anatomy, 974.
    prognosis, 979.
    results, 978.
    symptoms, 975.
    treatment, 980.
  chronic, 983.
    granular, 984.
    serous, 984.
      diagnosis, 985.
      treatment, 985.
  idiopathic, 973.
  infectious, 973.
  primary, 973.
  rheumatic, 973.
  secondary, 973.
  septic, 973.
  simple, 973.
  spontaneous, 973.
Perityphlitis, 876.
Pernicious anæmia, 9.
  fever, 208.
Persecution, delusions of, 385.
Personality, double, 467.
Pertussis, 179. (*Vide* Whooping-Cough.)
Phantom tumor, 412.
Pharyngeal tonsil, 801.
Pharyngitis, acute, 798.
  catarrhal, 800.
  phlegmonous, 799.
  sicca, 800.
Pharynx, inflammation of, 797.
Phlegmonous erysipelas, 187.
Phosphatic diabetes, 1008.
Phosphaturia, 1008.
Phosphorus, poisoning by, 361.
Phrenitis mania gravis, 513.
Phthisis, acute pulmonary, 254.
    cough in, 255.
    diagnosis, 256.
    laryngitis in, 255.
    physical signs in, 255.
    prognosis, 256.
    sputum, 255.
      staining of, 256.
    temperature in, 254.
    treatment, 292.
  advanced, 258.
  chronic pulmonary, 256.
    cavity in, 261.
    course, 261.
    diagnosis, 263.
    duration, 261, 262.
    examination, 257, 259.
    fever of, 261.
    hæmic murmur in, 261.
    hæmoptysis in, 259.
    intestinal ulcer in, 262.
    menstruation in, 262.

- Phthisis, chronic pulmonary, pain in, 258.
    - prognosis, 263.
    - sputum, 258.
    - treatment, 292.
    - urine in, 262.
  - fibroid, 264.
  - incipient, 257.
  - laryngeal, 263.
  - pulmonary, 251.
    - cavity in, 252.
    - cheesy bronchitis in, 253.
    - cheesy pneumonia in, 253.
    - morbid anatomy, 252.
    - pleura in, 254.
    - pneumothorax in, 253.
    - symptoms, 254.
    - treatment, 292.
- Pica, 850.
- Pin-worm, 338.
- Pitres, pediculo-frontal band of, 487.
- Plague, 162.
  - British, 118.
- Pleura, diseases of, 770.
  - echinococci of, 332.
  - tuberculosis of, 276.
  - tumors of, 787.
- Pleurisy, 775.
- Pleuritis, 775.
    - bacteria in, 776.
    - diagnosis, 782.
    - dry treatment, 785.
    - morbid anatomy, 777.
    - paracentesis thoracis, 785.
    - perforation in, 778.
    - physical examination, 780.
    - prognosis, 784.
    - symptoms, 779.
    - treatment, 784.
  - chronic, 778.
    - relations to tuberculosis, 776, 784.
  - diaphragmatic, 777.
  - dry, 777.
  - encysted, 777.
  - fibrino-serous, 777.
  - fibrinous, 777.
  - hemorrhagic, 777.
  - ichorous, 777.
  - interlobular, 777.
  - latent, 779.
  - metapneumonic, 776.
  - primary, 775.
  - secondary, 775.
  - sero-fibrinous, 777.
  - serous, 777.
- Pleurodynia, 74.
- Pleuro-pneumonia, 747.
- Plica Polonica, 347.
- Plumbic acetate, poisoning by, 361.
- Pneumonia, abortive, 747.
  - acute, 741.
    - bacillus of, 742.
    - blood in, 746.
    - complications of, 747.
    - diagnosis, 748.
    - engorgement in, 742.
    - gangrene in, 743.
    - hepatization in, 742.
    - morbid anatomy, 742.
- Pneumonia, acute, physical examination, 745.
    - prognosis, 748.
    - resolution in, 743.
    - sputum in, 745.
    - stages of, 742.
    - symptoms, 743.
    - treatment, 749.
    - varieties, 746.
  - apical, 747.
  - central, 747.
  - cerebral, 747.
  - cheesy, 252.
  - chronic fibrous, 755.
    - diagnosis, 758.
    - etiology, 757.
    - morbid anatomy, 757.
    - prognosis, 758.
    - symptoms, 757.
    - treatment, 758.
  - chronic interstitial, 755.
  - croupous, 741.
  - ephemeral, 747.
  - fibrinous, 741.
  - hypostatic, 731.
  - lobar, 741.
  - typhoid, 746.
  - wandering, 747.
- Pneumonokoniosis, 756.
- Pneumo-pericardium, 628.
- Pneumothorax, 770.
  - circumscribed, 770.
  - coin sound in, 772.
  - diagnosis, 772.
  - diffused, 770.
  - morbid anatomy, 770.
  - physical examination, 771.
  - prognosis, 772.
  - symptoms, 771.
  - treatment, 772.
- Podagra, 78.
- Poisoning, acute, 350.
    - aconite, 358.
    - alcohol, 351.
    - antimony, 360.
    - arsenic, 360.
    - atropine, 355.
    - delirifacients, 355.
      - treatment, 355.
    - narcotics, 351.
      - artificial respiration, 354.
      - law of crossed action, 354.
      - treatment, 353.
    - Calabar bean, 357.
    - cantharides, 361.
    - carbolic acid, 352.
      - antidote, 353.
    - cardiants, 358.
    - chloral, 351.
    - chloroform, 352.
    - citric acid, 360.
    - cocaine, 355, 356.
    - conium, 358.
    - convulsants, 356.
    - corrosive sublimate, 360.
    - cupric sulphate, 361.
    - ether, 352.
    - gelsemium, 358.
    - hydrochloric acid, 360.
    - hyoscine, 355.
    - hyoscyamine, 355.
    - illuminating gas, 351.
- Poisoning, acute, irritants, 360.
    - lobelia, 357.
    - nitric acid, 360.
    - nitrites, 357.
    - nitro-benzol, 352.
    - nitrohydrochloric acid, 360.
    - oil of rue, 361.
    - oil of savine, 361.
    - oil of tansy, 353.
    - opium, 351.
      - treatment, 353.
    - oxalic acid, 360.
    - paralyzants, 357.
      - treatment, 358.
    - pelletierine, 358.
    - phosphorus, 361.
    - plumbic acetate, 361.
    - prussic acid, 352.
    - santonin, 352.
    - Spanish fly, 361.
    - strychnine, 356.
      - treatment, 357.
    - sulphuric acid, 360.
    - tartaric acid, 360.
    - veratrum viride, 358.
    - woorari, 358.
  - chronic, 362.
    - alcohol, 368.
    - antimony, 368.
    - arsenic, 367.
    - cocaine, 376.
    - lead, 362.
      - treatment, 366.
    - opium, 374.
- Polioencephalitis, 509.
  - superior, 510.
- Poliomyelitis, acute, 560.
    - deformity in, 564.
    - diagnosis, 564.
    - electricity in, 567.
    - paralysis in, 562.
    - reaction of degeneration in, 563.
    - symptomatology, 562.
    - treatment, 565.
  - ascending, 568.
  - chronic, 569.
  - metallic, 568.
- Polyæsthesia, 579.
- Polyarthritis, rheumatic, 61.
- Polymyositis, acute, 44.
- Polyphagia, 850.
- Pons, tumors of, 526.
- Porencephalia, 508.
- Post-febrile insanity, 395.
- Potassium ferrocyanide test in albuminuria, 1002.
- Præcordial anguish, 392.
- Pregnancy, chorea of, 437.
- Pressure palsy, 603.
  - sense, 381.
- Primary curable dementia, 395.
  - myopathy, 45.
  - myositis, 44.
- Prison fever, 145.
- Progressive muscular atrophy, 569.
  - pernicious anæmia, 9.
- Prostate, tuberculosis of, 272.
- Protozoa, 321.
- Prussic acid, poisoning by, 352.

Pseudo-diphtheria, 171.
Pseudo-leukæmia, 16.
Pseudo-muscular hypertrophy, 45.
Psorosperms, 349.
Psychical anæsthesia, 391.
  dysthesia, 391.
  hyperæsthesia, 391.
Puerperal insanity, 515.
  mania, 395.
  osteomalacia, 57.
Pulex irritans, 348.
Pulmonary consumption, 251.
  gangrene, 764.
  hemorrhage, 732.
  insufficiency, 668.
  phthisis, 251.
    acute, 254.
    chronic, 256.
  stenosis, 668.
  valve, disease of, 668.
Pupil, Argyll-Robertson, 478, 580.
Pupils in cerebral disease, 478.
Purpura, 24.
  fulminans, 27.
  hæmorrhagica, 26.
  Henoch's, 26.
  rheumatic, 25.
  simple, 24.
  symptomatic, 20.
Pustule, malignant, 235.
Pyæmia, 191.
  diagnosis, 193.
  treatment, 194.
Pyelitis, 1045.
  diagnosis, 1047.
  morbid anatomy, 1046.
  prognosis, 1048.
  symptoms, 1047.
  treatment, 1048.
Pyloric incompetency, 851.
Pyonephrosis, 1048.
Pyopneumothorax, 771.
Pyosepticæmia, 191.
Pyromania, 386.
Pyuria, 1015.

Q.

Quinsy, 802.

R.

Rabies, 233.
  diagnosis, 234.
  dumb, 233.
  furious, 233.
  prognosis, 234.
  treatment, 234.
Rachitis, 50.
Ranula, 795.
Raptus melancholicus, 392.
Ray-fungus in actinomycosis, 231.
Raynaud's disease, 619.
    chronic, 621.
Reaction of degeneration, law of, 563.
Reflexes, 378.
  ankle, 378.
  deep, 378.
  patella, 378.
Reflexes, reinforcement of, 378.
  superficial, 378.
Regurgitation, 850.
Relapsing fever, 149.
Remittent fever, 207.
Renal calculus, 1053.
    composition of, 1053.
  cysts, 1042.
  phthisis, 270.
Rest-cure in neurasthenia, 402.
Retropharyngeal abscess, 799.
Rheumatic arthritis, chronic, 70.
  fever, 61.
  gout, 71.
  polyarthritis, 61.
  purpura, 25.
Rheumatism, acute articular, 61.
    complications of, 64.
    cutaneous eruptions in, 65.
    diagnosis, 65.
    duration, 64.
    germ theory of, 62.
    prognosis, 66.
    symptoms, 63.
    treatment, 66.
  cerebral, 65.
  chronic articular, 70.
  gonorrhœal, 68.
  muscular, 74.
  scarlatinal, 98.
  subacute, 64.
Rheumatoid arthritis, 71.
Rhinitis, acute, 701.
  chronic, 703.
Rickets, 50.
  diagnosis, 53.
  feeding in, 54.
  fœtal, 50 (*note*).
  hemorrhagic, 55.
  prognosis, 53.
  treatment, 53.
Romberg's symptom, 579.
Rose-cold, 704.
Rötheln, 107.
Round worms, 337.
Rubeola, 102.
  black form of, 104.
  complications of, 104.
  diagnosis, 105.
  hemorrhagic, 104.
  malignant forms of, 104.
  prognosis, 105.
  treatment, 105.
Rue, oil of, poisoning by, 361.

S.

Salaam convulsions, 439.
Salivary glands, inflammation of, 796.
Sand-flea, 348.
Santonin, poisoning by, 352.
Sapræmia, 189.
Sarcopsylla penetrans, 348.
Sarcoptes hominis, 346.
Saturnine cerebritis, 363.
Savine, oil of, poisoning by, 361.
Scapulo-humeral type of atrophic myopathy, 48.
Scarlatina, 95.
  complications, 98.
  diagnosis, 99.
  diphtheria in, 98.
  eruption in, 96.
  fever in, 101.
  maligna, 98.
  nephritis in, 95, 99.
  prognosis, 100.
  simple, 97.
  tongue in, 96.
  treatment, 100.
Scarlet fever, 95.
Schoenlein's disease, 25.
Sciatica, 609.
Sclerema neonatorum, 625.
Sclerodactylia, 625.
Scleroderma, 623.
Scleroses, combined, 588.
Sclerosis, amyotrophic lateral, 588.
  antero-lateral, 585.
  disseminated, 521.
  multiple cerebro-spinal, 522.
Scoliosis, 601.
  paralytica, 601.
Scorbutus, 22.
Scotomata, 480.
Scrofula, 285.
  bones in, 289.
  catarrh in, 288.
  diagnosis, 290.
  etiology, 287.
  lymph-glands in, 289.
  morbid anatomy, 287.
  prognosis, 290.
  symptomatology, 288.
  types of children with, 289.
Scrofuloderma, 250.
  treatment, 300.
Scurvy, 22.
  infantile, 50.
Secondary anæmia, 3.
Seminal vesicles, tuberculosis of, 272.
Senile osteomalacia, 57.
Sensation, disturbances of, 381.
Sense-shock, 464.
Septic infection, 189.
  intoxication, 189.
  toxæmia, 189.
Septicæmia, 189.
Septico-pyæmia, 191.
Serous membranes, tuberculosis of, 275.
Ship fever, 145.
Siderosis of lungs, 756.
Silvester's method of artificial respiration, 354.
Simple anæmia, 2.
Singultus, 416.
Skin, tuberculosis of, 250.
Sleep, disorders and accidents of, 460, 463.
  morbid, 463.
Small-pox, 109. (*Vide* Variola.)
  black, 113.
  hemorrhagic, 110, 113.
  malignant, 110, 113.
Smell, centre for, 476.
Softening of brain, 518.
Somnambulism, 465.

Soul-blindness, 487.
Spanish fly, poisoning by, 361.
Spasm, 379.
  facial, 431.
  local, 431.
  nodding, 432.
  rotary, 432.
Spastic paralysis, infantile, 508.
  paraplegia, 585.
Spinal abscess, 547.
  anæmia, 409, 545.
  apoplexy, 543.
  convulsions, 378.
  cord, anatomy of, 540.
    hyperæmia of, 545.
    tuberculosis of, 284.
  embolism, 544.
  epilepsy, 586.
  hemorrhage, 543.
  irritation, 409.
  localization, 540.
  meningitis, acute, 546.
    chronic, 547.
  pachymeningitis, 546.
  syphilis, 592.
  thrombosis, 544.
  tumor, 548.
Spirillum Obermeieri in relapsing fever, 149.
Splanchnoptosis, 29, 855.
Spleen, abscess of, 30.
  diseases of, 28.
  embolism of, 30.
  enlargement of, 28.
  movable, 29.
  tuberculosis of, 281.
  wandering, 29.
Splenic anæmia, 16.
Splenoptosis, 29.
Sporozoa, 321.
  of malaria, 203.
Spotted fever, 152.
Sputa, nummulated, 258.
Sputum, staining of, in phthisis, 255.
Station test, 380.
Stomach, atony of, 851.
  cancer of, 843.
    cachexia in, 846.
    diagnosis, 847.
    examination of contents in, 848.
    extension of, 845.
    morbid anatomy, 844.
    prognosis, 848.
    symptoms, 845.
    treatment, 848.
    tumor in, 846.
    urine in, 847.
    vomiting in, 845.
  cramp of, 851.
  dilatation of, 823.
  examination of contents of, 820.
    in cancer, 847.
  hyperacidity of, 852.
  hyperæsthesia of, 850.
  inflation of, 817.
  malposition of, 822.
  neuroses of, 849.
    of motility, 850.
    of secretion, 852.
Stomach, neuroses of sensation, 849.
  peracidity of, 852.
  peristaltic unrest of, 851.
  physical examination of, 818.
  spasm of, 851.
  superacidity of, 852.
  ulcer of, 836.
    chronic, 837.
    diagnosis, 840.
    gastralgia in, 842.
    hæmatemesis in, 839, 842.
    morbid anatomy, 837.
    perforation in, 840, 843.
    prognosis, 841.
    symptoms, 837.
    treatment, 841.
Stomatitis, 791.
  aphthous, 791.
  catarrhal, 791.
  gangrenous, 791.
  parasitic, 792.
  symptoms, 792.
  treatment, 793.
  ulcerative, 792.
Strawberry tongue in scarlatina, 96.
Streptococcus erysipelatis, 185.
  pyogenes, 185.
Striated bodies, tumors of, 526.
Strongylus longivaginatus, 339.
Struma, 31.
Strychnine, poisoning by, 356.
Stupor, 460.
  delusional, 395.
St. Vitus's dance, 432.
  diagnosis, 436.
  prognosis, 436.
  symptomatology, 434.
  treatment, 436.
Subacute rheumatism, 64.
Sublingual glands, inflammation of, 796.
Submaxillary glands, inflammation of, 796.
Subphrenic abscess, 914.
  pyopneumothorax, 914.
Sugar in urine, test for, 1010.
Sulphuric acid, poisoning by, 360.
Sunstroke, 450.
Suppurative encephalitis, 510.
  myositis, 44.
Suprarenal capsules, tuberculosis of, 619.
  melasma, 41.
Surgical insanity, 395.
Symmetrical gangrene, 619.
Symptomatic purpura, 20.
Syncope, local, 619.
Syphilide, macular, 307.
  papular, 307.
  pustular, 308.
  tubercular, 309.
Syphilis, 304.
  acquired, 306.
    alopecia in, 308.
    choroiditis in, 309.
    eruption of, 307.
    exostosis in, 309.
    gumma in, 310.
    hard chancre in, 306.
Syphilis, acquired, Hunterian chancre in, 306.
    iritis in, 309.
    macular syphilide, 307.
    nodes in, 309.
    papular syphilide, 307.
    periostitis in, 309.
    pustular syphilide, 308.
    secondary stage, 307.
    tertiary stage, 309.
    tubercular syphilide, 309.
    visceral, 311.
  bacilli of, 304.
  cerebral, 528.
    age in, 533.
    convulsions in, 529, 531.
    diagnosis, 532.
    gumma in, 528.
    headache in, 530.
    insomnia in, 530.
    palsy in, 529, 531.
    prodromes, 532.
    prognosis, 534.
    treatment, 534.
  Colles's law, 304.
  gumma in, 305.
  hereditary, 312.
    diagnosis, 315.
    infantile, 314.
    intra-uterine infection, 313.
    marriage in, 317.
    morbid anatomy, 313.
    osteochondritis in, 313.
    prognosis, 316.
    prophylaxis, 316.
    symptoms, 314.
    treatment, 317.
      of cachexia, 320.
  morbid anatomy, 305.
  of arteries, 311.
  of heart, 311.
  of liver, 311.
  of lungs, 311.
  of nerves, 618.
  of testis, 312.
  spinal, 592.
Syphiloma, 305.
Syringomyelia, 572.
  diagnosis, 575.
  gliosis in, 573.
  prognosis, 575.
  symptomatology, 573.
  treatment, 575.
  trophic changes, 575.

T.

Tabes, 575.
  mesenterica, 266.
  meseraica, 266.
Tabetic anthropathy, 580.
Taches bleuâtres, 347.
Tachycardia, 679.
Tænia acanthotrias, 324.
  cucumerina, 324.
  echinococcus, 329.
  elliptica, 324.
  flavo-punctata, 324.
  madagascariensis, 324.
  mediocanellata, 324.
  nana, 324.

- Tænia saginata, 324.
  - solium, 323.
- Tansy, oil of, poisoning by, 353.
- Tape-worms, 323.
  - diagnosis, 326.
  - etiology, 324.
  - symptoms, 325.
  - treatment, 326.
- Tartaric acid, poisoning by, 360.
- Taste, centre for, 476.
  - loss of, 477.
- Temporal lobe, tumors of, 526.
- Terminal dementia, 397.
- Testes, scrofula of, 273.
  - syphilis of, 312.
  - tuberculosis of, 273.
- Tetanic convulsions, 378.
- Tetanus, 194.
  - antitoxin in, 200.
  - bacillus of, 194.
  - cerebral, 198.
  - diagnosis, 198.
  - idiopathic, 198.
  - neonatorum, 198.
  - opisthotonos in, 197.
  - prognosis, 198.
  - symptomatology, 196.
  - treatment, 199.
- Tetany, latent, 443.
  - treatment, 443.
- Thermic fever, 450.
    - bathing in, 453.
    - diagnosis, 452.
    - sequelæ, 454.
    - treatment, 452.
  - sensibility, 381.
- Thomsen's disease, 49.
- Thread-worms, 337.
- Thrombosis, cerebral, 494.
  - of cerebral sinus, 497.
  - of heart, 648.
- Thymic asthma, 40.
- Thymus gland, diseases of, 40.
    - tuberculosis of, 283.
    - tumors of, 40.
- Thyroid gland, diseases of, 31.
    - tuberculosis of, 283.
    - tumors of, 40.
- Tic, 431.
  - choreic, 438.
  - convulsif, 439.
  - convulsive, 431.
  - douloureux, 431, 613.
  - painless, 431.
  - salaam, 439.
- Ticks, 346.
- Titubation, 381, 488.
- Tongue, dissected, 794.
  - geographic, 794.
  - ichthyosis of, 794.
  - keratosis of, 794.
  - œdematous enlargement of, 795.
  - psoriasis of, 794.
- Tongue-deafness, 485.
- Tonsils, inflammation of, 797.
  - pharyngeal, 801.
- Tonsillitis, 801.
  - acute, 801.
  - chronic, 803.
- Torticollis, 431.
  - rheumatic, 24.
- Trachea, diseases of, 712.
- Trachitis, 712.
- Trance, hysterical, 408.
- Traumatic back, 447.
  - neurosis, 446.
    - diagnosis, 448.
    - treatment, 449.
- Tremor, 380.
  - intention, 380.
  - persistent, 380.
- Trichina spiralis, 342.
- Trichinosis, 342.
  - diagnosis, 344.
  - symptoms, 344.
  - treatment, 345.
- Trichocephalus dispar, 340.
- Trichomonas intestinalis, 322.
  - vaginalis, 322.
- Tricuspid insufficiency, 667.
  - stenosis, 668.
- Trigeminal nerve, inflammation of, 613.
- Trommer's test for sugar in urine, 1010.
- Trophic disturbances, 382.
- Tropical anæmia, 3.
  - dysentery, 212.
- Tubal tuberculosis, 274.
- Tubercle, 242.
  - anatomist's, 250.
  - miliary, 242.
- Tubercula dolorosa, 618.
- Tuberculin, 241.
- Tuberculosis, 239.
    - antiphthisin in, 241.
    - bacillus of, 239, 241.
    - caseation in, 243.
    - cheesy degeneration in, 243.
    - cheesy metamorphosis in, 243.
    - miliary tubercle in, 242.
    - morbid anatomy, 242.
    - predisposing causes, 241.
    - primary, 240.
    - secondary, 240.
    - symptoms, 244.
    - treatment, 292.
    - varieties, 244.
  - acute or disseminated, 244.
    - morbid anatomy, 245.
    - prophylaxis, 290.
    - symptomatology, 245.
    - temperature in, 245.
    - treatment, 292.
  - chronic general, 248.
    - prophylaxis, 290.
    - treatment, 292.
      - alcohol, 294.
      - climate, 295.
      - counter-irritation, 297.
      - exercise, 294.
      - food, 294.
      - germicides, 293.
      - inhalations, 297.
      - internal medication, 297.
      - of fever, 299.
      - of glands, 293.
      - of hæmoptysis, 298.
      - of night-sweats, 299.
      - pulmonary gymnastics, 297.
- Tuberculosis, chronic, tuberculin treatment, 293.
  - local, 249.
    - of alimentary canal, 265.
    - of bladder, 271.
    - of blood-vessels, 283.
    - of bones, 285.
    - of brain, 284.
    - of ductless glands, 281.
    - of Fallopian tubes, 274.
    - of heart, 283.
    - of intestines, 265.
    - of joints, 285.
    - of kidney, 270.
    - of liver, 268.
    - of lungs, 247.
      - diagnosis, 248.
      - examination in, 248.
      - onset of, 247.
      - prognosis, 249.
      - treatment, 292.
    - of lymph-glands, 281.
    - of mammary gland, 274.
    - of mucous membranes, 250.
    - of ovaries, 274.
    - of pancreas, 269.
    - of pericardium, 275.
    - of peritoneum, 276.
    - of pleura, 276.
    - of prostate, 272.
    - of seminal vesicles, 272.
    - of serous membranes, 275.
    - of sexual organs of females, 273.
    - of skin, 250.
    - of spinal cord, 284.
    - of spleen, 281.
    - of suprarenal capsule, 283.
    - of testicle, 273.
    - of thymus, 283.
    - of thyroid, 283.
    - of uro-genital tract, 269.
    - of uterus, 274.
    - of vascular system, 283.
    - pulmonary, 251. (*Vide* Phthisis, pulmonary.)
- Tumors, intracranial, 524.
    - diagnosis, 527.
    - epileptic attacks in, 525.
    - knee-jerk in, 527.
    - treatment, 528.
  - of biliary tract, 947.
  - of heart, 650.
  - of intestines, 894.
  - of kidneys, 1043.
  - of larynx, 711.
  - of lungs, 767.
  - of mediastinum, 789.
  - of œsophagus, 812.
  - of peritoneum, 986.
  - of pleura, 787.
  - of thymus gland, 40.
  - of thyroid, 40.
  - phantom, 412.
  - spinal, 548.
- Tuphlo-enteritis, 876.
- Typhlitis, 876.
- Typhoid, bilious, 151.
  - face, 125.
  - fever, 119.
    - abortive, 130.
    - apyretic, 130.

Typhoid fever, afebrile, 130.
ataxic, 130.
bacillus of, 119, 135.
bathing in, 141.
bilious, 129.
complications of, 131.
convalescence in, 133.
crisis in, 124.
delirium in, 125.
diagnosis, 133.
diet in, 139.
Ehrlich's test, 134.
eruption in, 123.
foudroyant, 130.
gastric, 129.
hemorrhagic, 129.
intestinal hemorrhage in, 128.
morbid anatomy, 121.
perforation in, 122, 128, 144.
Peyer's patches in, 121, 143.
prognosis, 135.
prophylaxis, 137.
pulse in, 126.
relapse in, 124.
sequelæ of, 131, 133.
solitary follicles in, 121.
special symptoms of, 126.
spleen in, 122, 128.
stools in, 127.
symptomatology, 123.
temperature in, 126.
treatment, 138.
feeding, 138.
of accidents, 142.
of fever, 140.
of peritonitis, 144.
of symptoms, 142.
urine in, 129.
varieties of, 129.
walking, 130.
pneumonia, 746.
Typho-malarial fever, 132.
Typhomania, 513.
Typhus biliosus, 151.
fever, 145.
diagnosis, 148.
eruption in, 147.
face in, 145.
prognosis, 148.
symptomatology, 148.
treatment, 148.

U.

Ulcer, corrosive, 837.
of stomach, 836.
diagnosis, 840.
gastralgia in, 838, 842.
hemorrhage in, 839, 842.
prognosis, 841.
symptoms, 838.
treatment, 841.
peptic, 837.
perforating, 621, 837.
Uræmia, 1015.
diagnosis, 1017.
in congestion of kidneys, 1020.
in chronic diffused nephritis, 1030.
Uræmia in chronic fibrous nephritis, 1038.
prognosis, 1017.
Uraturia, 1006.
Uricacidæmia, 80.
Uricaciduria, 1006.
Uricæmia, 80.
Urine, abnormalities of, 994.
albumin in, 1001.
tests for, 1002.
blood in, 994.
casts in, 1013.
color of, 994.
cylindroids in, 1013.
in acute yellow atrophy of liver, 917.
in amyloid degeneration of kidney, 1039.
in cancer of liver, 930.
in cancer of stomach, 847.
in chlorosis, 5.
in cystitis, 1059.
in diabetes insipidus, 93.
in diabetes mellitus, 88.
in endocarditis, chronic, 662.
in floating kidney, 991.
in hydronephrosis, 1049.
in hysteria, 410.
in lead poisoning, 336.
in nephritis, acute, 1024.
chronic diffuse, 1029.
chronic fibrous, 1033.
in phthisis, 262.
in pneumonia, 746.
in pyelitis, 1047.
in renal tuberculosis, 271.
in rheumatism, 63.
in scarlatina, 99.
in tuberculosis, 246.
in typhoid fever, 129.
in yellow fever, 229.
incontinence of, paralytic, 1057.
spasmodic, 1057.
sugar in, test for, 1010.
Urobilinuria, 998.
Uro-genital tuberculosis, 269.
Urticaria, giant, 622.
Uterus, tuberculosis of, 274.

V.

Vaccination, 116.
operation for, 117.
Vaccinia, 116.
Vagabond's disease, 347.
Variola, 109.
black, 113.
coherent, 110.
confluent, 110.
contagium of, 109.
desiccation in, 112.
diagnosis, 113.
discrete, 110.
eruption of, 111.
hemorrhagic, 110, 113.
invasion of, 111.
malignant, 110, 113.
prognosis, 114.
stages of, 110.
Variola, suppuration in, 112.
symptomatology, 110.
treatment, 114.
vera, 110.
Varioloid, 109.
Vascular system, tuberculosis of, 283.
Vaso-motor disturbances, 382.
Veratrum viride, poisoning by, 358.
Vertigo, 416.
cardiac, 417.
epileptic, 417.
essential, 418.
gastric, 417.
hysterical, 417.
lithæmic, 418.
organic, 417.
peripheral, 417.
special sense, 417.
toxæmic, 418.
Vision, contraction of field of, 480.
Voltolini's disease, 418.
Vomiting, habitual, 850.
nervous, 850.

W.

Walking typhoid fever, 130.
Wandering spleen, 29.
Wasting palsy, 570.
Water-brash, 811, 828.
Water-hammer pulse, 666.
Weil's disease, 151.
Werlhof's disease, 26.
Westphal's symptom, 378, 579.
Whip-worm, 340.
Whooping-cough, 179.
complications, 181.
diagnosis, 181.
treatment, 182.
Wool-sorter's disease, 236.
Woorari, poisoning by, 358.
Worms, parasitic, 322.
fluke, 335.
Guinea-worm, 340.
pin-worm, 338.
round worm, 337.
tape-worm, 323.
thread-worm, 337.
trichinæ, 342.
whip-worm, 340.
Wrist-drop, 334.
Writer's cramp, 455.

X.

Xanthopsia, 933.
Xeroderma pigmentosum, 625.

Y.

Yellow fever, 226.
course, 229.
diagnosis, 229.
morbid anatomy, 227.
prognosis, 230.
prophylaxis, 230.
remission in, 228.
symptomatology, 228.
treatment, 230.
zones of, 227.

ekp O.S.

ckcp